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ON

CLINICAL MEDICINE.

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ON

CLINICAL MEDICINE

BY

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GENTLEMEN: To-day, I propose to speak to you on the subject of Alcoholism.

Without concealing any of its difficulties—because I fully recognize their existence—it may still be useful to state to you my views upon this problem, one of the most difficult in pathology. If it be necessary to impress upon young physicians the classical precepts of their profession, it is not less useful, at rare intervals, to make with them more perilous scientific excursions.

The affections of the nervous system, of which I have given you a summary and necessarily incomplete history, present in a less degree than the affections of other organs, but still they do present, a certain degree of uniformity and definite character in their symptoms. To say that a man is *epileptic* is to characterize the individual, the symptoms from which he suffers, and the future which is in store for him. To say that a woman is *hysterical* is to place her in a more vaguely defined class. But subordinate to these grand divisions of nervous affections, how many minute divisions are there which escape even a vague classification!

When, in place of obtaining the elements of our definitions from an interrogation of symptoms, we go back to the causes which have provoked, awakened, or maintained derangements of the nervous system, we attain to a more intimate knowledge of the nature of the disease: but here, as in everything else, the more we leave the surface, the more do obscurities increase, and outlines become uncertain.

You all know, gentlemen, and unprofessional persons know as well as you do, that alcoholic preparations produce obvious effects upon the organism, and that, in the first instance, they show a predilection for the *cerebro-spinal system*, although at a later stage, the other great systems are variously affected, as I shall endeavor to explain to you. You are also well aware that unfortunately there is a great prevalence of dissipation among the class from which the hospitals chiefly derive their patients. This fact constitutes an etiological element which you will too often encounter complicating diseases, disturbing their evolution, and impeding convalescence.

And yet, till very recently, the custom has been to consider the study of the peculiar effects of alcoholism as belonging only to physicians specially devoted to mental diseases. I accept, but never without profound regret, the artifical limits imposed by the administrative exigencies of practice, or by personal deficiency; but in the matter which I have now brought under your notice, I cannot resign myself to any similar conventional arrangement. Extreme cases, it is true, may only be accessible to observation in the wards of a lunatic asylum, but it is otherwise with cases of moderate severity—with effects more limited in character and not culminating in insanity. Alcoholism belongs to the same category as many other cerebral affections. There is an insensible gradation of cases between the attack of apoplexy consequent upon which the mental powers have fallen to the lowest form of dementia, and the apoplectic seizure after which there only remains an almost imperceptible amount of hemiplegia: but in this gradation of cases, you will be unable to appreciate the value of the symptoms which chance has brought under your notice, unless you keep in view the two extremes of the progressive series.

This statement is still more applicable in respect of alcoholism. In the first instance, the effects of alcohol are within the domain of physiology: taken in small quantities, alcohol has its right and proper place in the aliment of the body: but taken in excess, it either leads to formidable maniacal excitement, or the pitiable spectacle of acquired idiocy. You will admit, gentlemen, that within this vast domain, it will only be possible for me here to point out some landmarks.

Alcoholic compounds used in what I may call physiological quantities are unquestionably beneficial to the human subject in a state of health: there are also cases in which they may be useful in a state of disease. I do not require to recapitulate the circumstances under which their administration is appropriate in health or disease. And yet what useful knowledge there is to be learned in relation to that subject! You have often seen me prescribe wine in tolerably large doses in adynamic fevers, and during convalescence from inflammatory affections which have left great depression behind them.

Fermented liquors constitute more than a complement to alimentation: they likewise fulfil another class of indications. For a long period, however, their employment in medicine was proscribed as a perilous enormity; but latterly, by one of those reactions of which therapeutics offers so many examples, there has been no shrinking from giving them with extreme hardihood. Without running any risk by following these aberrations, we may profit by daring trials from which physicians of most reliable character have not shrunk.

The clinical experience of the present day teaches us that even patients suffering from inflammatory affections tolerate fermented drinks in quantities which formerly one would have been far from suspecting. Though it has not been demonstrated that they do as much good as was hoped by the physicians who recommend them, it is certain that they do not produce the injurious consequences dreaded on theoretical grounds. It is an additional proof in support of that law, upon which it is impossible to insist too often, that during the existence of a pathological state, the action of toxic medicines is profoundly modified. Just as you have observed opium pushed with impunity to doses which may be called imprudent in refractory diseases, so have you seen large doses of alcoholic liquors borne without any bad consequences by patients suffering from various affections of the nervous system.

The data which I am about to give are not laid before you as examples to follow; but as facts calculated to assist in the study of the toxic effects of alcohol.

Pathologically, as well as physiologically, it is important to distinguish between the healthy and the sick individual, so that we may not attribute to the one as an incontestable verity, an effect which is a reality only in respect of the other.

When a man in the plenitude of health gives way to alcoholic excesses, more or less frequently, and to a more or less serious extent, he experiences effects which differ in form and degree. If the same individual during self-induced intoxication falls into sickness, he forthwith, in consequence of the incident disease, stands in a different relation to the toxic agent: neither the susceptibility, the phenomena, nor the consequences are necessarily the same as before the pathological change took place. By bearing in mind this fundamental distinction, you will be saved from more than one source of confusion, and get the key wherewith to explain many mistakes.

In the hospital, it is only on rare occasions that you have an opportunity of observing the effects of excessive alcoholic potations upon persons who are not affected by disease. Here, drunkenness appears as one of the remote and often doubtful antecedents of the affections which it engenders, and is a matter of sharpened memory and not of direct observation. At other times, it explains the tendency which patients have to become delirious in the course of diseases in which delirium is not an essential symptom: and it induces a predisposition to disturbance of the nervous system; but its intervention in this class of cases is more conjectural than in the other. You have seen, however, some cases, which I have been careful to point out to you, of alcoholic delirium uncomplicated with any other affection.

Drunkenness in its first degree presents you with a miniature picture of the symptoms of the more advanced stages of ebriose poisoning: on the one hand, there is gastro-intestinal disturbance; and on the other, there are disorders which are either nervous and secondary, or purely sympathetic. The mouth is pasty, the tongue is dirty, the stomach is surcharged, and there is nausea: the head is heavy, the senses are either in an excited or obtuse condition, and there is giddiness verging upon syncope. Cerebral excitement gives place to invincible depression, and the sleep reminds one of apoplectic stertor.

The fit [*l'accès*]—let me use that word—is of short duration, but it leaves behind it a state of discomfort which is more prolonged, and which, in accordance with the temperament of the individual, dominates one or other order of symptoms.

Up to that point, the individual, to a certain extent, retains his individuality. He is good tempered or irritable in his cups [*il a le vin bon ou* *mauvais*] to use the vulgar phrase, according to the tendencies of his character: he is more or less out of sorts, or unwell, according to the nature of his constitution.

In the second degree of drunkenness, the inebriated man is a sick man [*l'ébrieux est un malade*]. The perversion of disposition is so great that the man is no longer himself: the symptoms become developed in the usual way, and are no longer obedient to diversity of temperament. The delirium then assumes a well-marked character, the nervous disorder takes a definite form, and the totality of the symptoms is summed up in the name—*delirium tremens.*

The delirium has a sufficiently special character to enable the experienced practitioner to recognize it, without requiring him to know the previous history of the case. The physicians who have made mental alienation a special study have described *delirium tremens* in a very masterly manner, and to their works I refer you for a complete description of the disease. It will be sufficient for the present, that I point out to you some of its most decisive signs.

In *delirium tremens*, the delirium is characterized by restlessness and mental perplexity, even in cases in which there is extreme violence. The excitement results from fear; for terror itself has its times of audacity. The patient, pursued by hallucinations (particularly of the sense of sight), threatened by assassins, attacked by robbers, is a prey to a thousand miseries. He wishes to start on a journey to some place—it matters not to what place: his desire is to get away from himself: he folds his clothes, and escapes by every possible way not interdicted to his vagabond impulses. In the midst of his disorderly excitement, he still retains the power of collecting his thoughts by the powerful effort of a dominant will; but this recovery of reason is brief, and ere long the patient returns to his vagaries.

Are you not struck, gentlemen, with the great similarity between *delirium tremens* and the delirium which supervenes in the course of many serious diseases? On the one hand, there are hallucinations which are almost exclusively confined to the sense of sight: then there exists the possibility of momentarily suspending the delirious conceptions: and lastly, there is the strange propensity to pack up for a journey. I refrain from pursuing this parallel any further, though it is instructive in more than one respect. I ought, and it is my wish, to confine myself to the briefest indications.

While the intellectual faculties are in this perverted state, that portion of the nervous system which does not preside over the mind is equally stricken. The most constant and the most conspicuous of all the nervous perturbations in this disease is trembling.

From a semeiological point of view, trembling is one of the phenomena most accessible to investigation, and yet the most difficult to describe categorically. This is an obscure object: trembling is a symptom of uncertain meaning, and its import is all the more doubtful, that a tendency has been shown to assign to it a value which it does not possess.

It is a very prejudicial error to rely on a mere pathological phenomenon, as if it were a diagnostic sign. I am quite aware that there is senile trembling, mercurial trembling, and many other kinds of trembling. But to classify after this fashion the different kinds of trembling, as if they were so many distinct unities, is to approximate to ontology more than to reality. In point of fact, trembling is one of the signs of old age, and of mercurial poisoning, as well as of alcoholism. To the latter, it does not exclusively belong, as a pathognomonic phenomenon.

Subject to this express reservation, alcoholic tremor has some peculiarities. In its most marked form, it is a sort of general fremitus. Lay your hand upon the shoulder, and you will feel the patient vibrate so to speak; but this description is not always applicable. It is sometimes characterized by muscular *subsultus*, the trembling being sometimes so convulsive as to make the step hesitating, and the prehension of objects almost impossible; you may perceive it either to cease or to continue under the influence of excessive maniacal excitement, which latter occurrence is the reverse of what takes place in the trembling of paralysis. You will observe that sleep does not suspend as certainly as it interrupts the choreic movements. Be that as it may, gentlemen, bear in mind that trembling is not necessarily the companion of alcoholic delirium, when that delirium comes on in persons otherwise in good health; and that it is far from being constantly met with when drunken delirium [*délire ébrieux*] makes its first appearance under the influence of a disease.

I do not now speak to you of the gastric symptoms relegated to the second phase of the disorder, or possibly entirely annulled by the magnitude of the nervous symptoms peculiar to *delirium tremens;* but we shall forthwith meet with them again and in chronic alcoholism, in which they are aggravated by repetition.

The crisis of *delirium tremens* is violent, but, relatively, it is short; and it is seldom fatal. After many therapeutic attempts—too often fortunate not to awaken some doubt—many physicians have come to restrict themselves to the expectant system, save in exceptional cases.

When the fit [*l'accès*] is terminated, all is not ended. It is an old and a true proverb, that "he who has drank will drink" [*qui a bu boira*]. The drunkard who has come through an attack of *delirium tremens* is as seldom cured of his passion for alcohol as is the gambler for his passion for play, and is in general excited to new debauches. Dipsomania is more frequently the sequel than the antecedent of a first attack of the delirium resulting from intoxication. The occasions on which the delirium shows itself recur; and the intoxication, which at first was acute, ultimately becomes chronic.

This, however, is not always the manner in which chronic drunkenness begins. While it is sometimes, as I have stated, the result of a repetition of acute fits, at other times, it originates in successive gradual poisoning, producing no shock, and arising from the habitual ingestion of alcohol in slowly increasing doses. Chronic alcoholism has been recently the subject of scientific researches; and you are no doubt acquainted with the remarkable description of it which has been given by Magnus Huss.* You know in how excellent a spirit of observation and method he has classified phenomena, the etiology of which he has so well elucidated. He has placed in one category *nervous disorders limited to different encephalic affections*, and in the other category, *disorders of the digestive system* resembling other cachexiæ. You are aware that within this vicious circle, so often met with in medical cases, perturbation of the nervous system contributes to impair nutrition.

In his truthful, lively, graphic picture, the scientific professor of Stockholm exhibits chronic alcoholism—which derives its characteristics from its very chronicity—assuming numerous forms, all of which, if you will pardon the use of an energetic popular locution, are really the small change [*la monnaie*] of the acute stage of *delirium tremens*. The mental phenomena are the same—they develop themselves more slowly, but their change of type is only apparent. Slacken the tumultuous pace of the fancies which jostle and caper in the maniac's brain, and although you do not thereby effect any essential modification, you will completely alter the aspect of his delirium. For the disgust, the repugnance to food which characterizes febrile anorexia, substitute passive indifference, absence of appetite, the gastric state of chronic alcoholism: in place of disturbance of vision, or the changing hallucinations of delirium tremens, there are confused perceptions, *muscæ volitantes*, cloudiness, fogginess, and transient flashes of false light.

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* Huss (Magnus): Chronische Alkoholskrankheit. Aus dem Schwedischen übersetzt von Gerh. van dem Busch. Stockholm, 1852.

But Dr. Huss made his observations under conditions of so special a character, that you require to exercise some reserve in drawing general conclusions from them. He studied alcoholism in a population addicted to the general abuse of alcohol, ill-fed, subsisting almost exclusively on vegetable diet; and which, under peculiar conditions, suffered the consequences of inveterate tippling habits. This population had no compensatory intervals of relative sobriety between the fits of debauchery, nor was its daily corporeal waste restored by generous fare. The type of drunkard sketched in so masterly a manner by Huss is that of the drunkard of northern countries: it is not the type of the drunkard met with in France, except among the very dregs of the people. Here, as a rule, intoxication is not gradual and successive, but is generally intermittent—acute during the drunken fit, and suspended during some succeeding days. The description loses its uniformity of application; the symptoms are more adventurous, for even poisoning has its diversities and its adventures.

But there is a question of still greater delicacy to which I wish to direct your earnest attention. Not only is chronic alcoholism far from showing itself with the aggregate of its characteristic signs, but as a consequence even of the intervals which I have been pointing out to you, it may exist, and yet never reveal itself by any phenomenon: it is this latent alcoholic saturation which is specially interesting to the physician, because it breaks forth at decisive moments. If an acute disease attack the individual who is thus saturated, the alcoholism explodes, and throws into the scale the formidable odds of its unforeseen delirium, or of an ataxia disproportionate to the actual disease.

When this is the programme—and in our country it is a very common programme—have we not at least indications to guide us in forming a retrospective diagnosis, a matter of so much difficulty even to physicians who understand its importance?

Since the time of Dupuytren, it has been asked: To what extent is the high delirium of patients upon whom amputations have been performed, a manifestation of latent alcoholism? This question has been answered in different ways. Physicians have had no doubts in respect of their department. A man, during the course of a moderately severe attack of pneumonia, in which the fever does not run high, is struck unexpectedly, and almost quite suddenly, with delirium: by what sign are we to recognize the true cause of this perturbation? I wish that I could furnish you with decisive signs; but I have not them to give, and science does not possess them. In your diagnosis, rely neither on the trembling, nor on the nature of the delirious fancies. Both are met with in patients whose antecedents are beyond suspicion. You will either be forced to proceed to a sort of inquisition into the habits of your patient, an inquisition always beset with sources of uncertainty and error; or you will have pretty nearly to trust to uncertainties and erroneous data, which it has been wished—but it has not been known how—to banish from medicine.

When you have summed up the information which you have collected, when you have deliberately weighed the import of the symptoms, and formed your opinion—what are you to do? The classical rule directs the p cian to take into account the habits which the patient has acquired, and give him, within judicious limits, the excitant for which he has the artificial craving. If this treatment is successful, the case serves as a confirmation of the rule.

Unfortunately, experience is more complex than it seems to be. At the beginning of this lecture, I dwelt upon the action of alcoholic stimulants in the course of acute diseases; and you have seen the length to which that system of incendiary therapeutics may be carried with impunity. I guarded you against adopting conclusions which are often imperfectly justified. In such cases, take your indications less from the habits of the patient when in health, than from the phenomena of his disease; do not have recourse to alcoholic stimulants, unless they seem to be required by the symptoms actually present.

Gentlemen, in the preceding picture I have only presented the effects of alcohol upon the nervous system and its injurious influence upon innervation; but that is only an incomplete history of its symptoms. Thanks to modern researches, we can now trace what may be called the physiological history of alcoholism, and follow the alcohol in its passage through the organism. I am also going to show you how alcohol acts upon each of the organs—how it produces lesions of them the sooner it reaches them, the longer it is in contact with them, and the more fragile and impressionable they are.

It is because the encephalon is fragile and impressionable that it is the first organ to feel the effects of alcohol, as well as the organ which feels them in the highest degree; and it is because these effects at once manifest themselves by an assemblage of striking symptoms that the history of alcoholism has for so long a period been cut short. There has been a greater disposition to describe the symptoms immediately resulting from the absorption of alcohol, the early manifestation of which left no doubt as to the relation between the symptoms and the alcohol; while the more remote effects produced by the contact of alcohol with the tissues has either been left in the shade, or quite misunderstood.

The symptoms which I am now about to describe to you in a succinct manner are precisely due to this contact, and they take their graduated ranks [*ils se hiérarchisent*] so to speak, according to physiological laws. Scarcely are alcoholic substances ingested than they exercise an action upon the stomach, and then, in less degree upon the intestines: absorbed to a great extent by the veins of the stomach, they pass through the portal system into the liver, into the right side of the heart, and thence into the pulmonary artery. These substances pass from the lesser into the greater circulation, and there act successively upon the walls of the arteries, and upon the tissues of different organs. It has been shown by the recent experiments of Maurice Perrin, Ludger Lallemand, and Duroy,* that alcohol is not decomposed in traversing the organism, and that it does not resolve itself into secondary products such as carbonic acid and aldehyde, as used to be believed; but that during the whole period of its sojourn in the tissues, it is alcohol, and as alcohol acts on them. Such being the case, it is evident that it will act the more energetically, the greater the quantity of it in a given organ; in the first place, according to whether that organ receives it before or after the alcohol has passed through the lungs—for a great part of the aclohol taken into the system is thrown of by pulmonary exhalation;

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* Ludger Lallemand, Maurice Perrin, and Duroy: Du Rôle de Alcool et des Anesthétiques dans l'Organisme.

and in the second place, according to the degree in which the organ is vascular.

The liver is the first organ in the track of absorption, and the whole quantity of alcohol absorbed traverses it. Moreover, it is not only a very vascular organ, but one in which the blood sojourns a long time for the requirements of the biliary and glycosic secretions. You can, therefore, at once perceive that the liver ought, more than any other organ, to be affected by the alcohol ingested; and such is, as I shall show you, really the case both in acute and chronic alcoholism. But the alcohol alters the condition of the walls of the pulmonary artery, and, consecutively, the tissue of the lungs, just as it alters the state of the parietes of the vena porta.

A very considerable portion, however, of the absorbed alcohol disappears during respiration by exhalation from the surface of the pulmonary vesicles and bronchial tubes. It is, therefore, only a smaller portion, the remaining portion, which affects the other organs, including the nervous centres, which feel its influence all the more energetically that the nervous elements, tubes, and cellules, are exceedingly impressionable, as I have already given you to understand. Next come the kidneys, organs which are very vascular, and which like the liver, preside over an important secretion—a depurative and eliminative secretion. Hence, the kidneys are in prolonged contact with the alcohol: and consequently, we see them frequently and seriously compromised in chronic alcoholism, although less frequently, and sometimes less seriously, than the liver, for the reasons which I have stated, viz., that the liver is traversed by the whole of the alcohol which has been absorbed, while the kidneys only receive that portion which has escaped from being thrown off by the lungs.

As a sequel to this general and purely physiological view of the effects of alcohol upon the organism, allow me, gentlemen, to enter into some details.

I have led you to anticipate the statement, that the stomach is morbidly affected in inveterate drinkers. There exists a real *gastritis*. It is this condition which explains the anorexia which goes on increasing till at last it becomes absolute—it is this which explains the dyspepsia of drunkards: it is this also which causes the mucous vomiting in the morning to which drunkards are subject—*vomitus matutinus potatorum*—a true gastrorrhœa related to chronic gastritis. This affection is characterized anatomically by a reddish color and some ecchymotic spots on the mucous membrane, particularly in the neighborhood of the cardiac orifice and smaller curvature: these are the characters of the first stage of the affection: afterwards, the mucous membrane is thick, and puckered, of a grayish or slate color, and stained by the pigmentary matter of hematinous deposit: still later, it is hard and friable, and after a time, it undergoes true softening. The glands of the stomach are hypertrophied. In great beer drinkers who imbibe enormous quantities of that fluid, the cavity of the organ may be dilated, and in such cases there is generally thinness of the mucous membrane: usually, the cavity is diminished in capacity from puckering of all its tunics.

If the inflammation, acute or subacute, reach the submucous cellular tissue, we may have phlegmonous gastritis with submucous abscess. This is a rare but an undoubted consequence of alcoholism.

In some drinkers, chronic inflammation of the mucous membrane terminates in ulceration. *Ulcerous* gastritis is much rarer than simple chronic gastritis. The ulcerations are either one or several: they are situated principally at the points where we have seen that inflammation is most liable to occur. According to Dr. Lancereaux, the almost constant presence of the coloring matter of the blood at the bottom or round the edges of the ulcers, as well as the disposition which they have to be elongated in the course of the vessels, would seem to indicate that at least some of the ulcers are due to alteration or consecutive obliteration of these vessels. There would be necrosis of the mucous membrane from vascular obliteration.

In these cases, it is very evident that the drunkards are diseased persons, and suffer not only from anorexia, dyspepsia, and mucous vomiting, but have also acute pain in the stomach, a radically bad digestion, vomiting of food (cometimes incontrollable), and often gastrorrhagia.

The *liver* is the organ which comes next to the stomach in point of frequency of lesion. It is subject to two distinct kinds of alteration from the contact of alcoholic substances: it may either undergo fatty degeneration, or it may be the seat of chronic inflammation: in the former case, it is *steatosis;* and in the latter, *cirrhosis.*

Steatosis consists in the deposit of fat in the hepatic cells. This alteration of tissue is almost invariably met with in alcoholic drinkers. When partial, it is compatible with health—at least with apparent health. It appears to be then only the first stage of a more profound change, which is met with particularly in those who sink under the acute symptoms of alcoholism, such, for example, as *delirium tremens.* The liver is augmented in volume and pale in color, where the lobules are infiltrated with fat.

When the steatosis is general, the liver presents a dull yellow hue, or is fawn-colored: it has a granular or embossed appearance produced by the infiltrated and consequently enlarged lobules projecting beyond the surrounding tissue. The liver is more voluminous in this than in the preceding form of steatosis, and has sometimes its left more hypertrophied than its right lobe. The accumulation of fat, by distending the hepatic cells and acini, compresses the capillary vessels, and so, of necessity, induces anæmia of the parenchyma. Hence originates a new cause of paleness of the organ.

In these cases, there is no pain in the liver. By palpation and percussion, its increased volume can be ascertained. The stomach performs its work of digestion the more imperfectly that both it and the liver are diseased. Generally, the stomach is distended with gas, and tender when pressure is made on it. The stools are few in number, and have a pale argillaceous appearance. Diarrhœa occurs sometimes: hemorrhages also take place, but not so frequently. Finally, Addison has pointed out a pale, waxy, cutaneous discoloration, in which the skin is supple, soft, and sometimes oily to the touch.

Cirrhosis, "gin-drinker's liver" [*foie des buveurs de gin*], is characterized by an exuberant formation of interstitial cellular tissue. I shall tell you, in a special lecture on cirrhosis, that it is a disease not always identical with itself, and that the cause which produces it imparts to it a special form. In alcoholism, the liver is everywhere equally saturated with alcohol, and everywhere equally affected with disease. At first, there is increased volume and vascularity of the organ: at a later period, the interstitial exudation becomes transformed into adventitious cellular tissue, which afterwards becomes fibrous, and then contracts: the organ everywhere decreases in bulk: the lobules and acini, indurated and diminished in size, slightly project above the depressions, more or less decided, which are everywhere constituted by the interlobular and interacinous partitions. The result is a general uneven state which is altogether characteristic. Here, I shall only mention—as I mean to return to the topic on a subsequent occasion—the profound alteration of nutrition which leads to progressive and altogether special emaciation, to possible hemorrhages, and particularly to that kind of ascites which accompanies cirrhosis.

Along with this profound disorganization of the liver, it is convenient to place transient, acute alterations of that organ, and particularly the functional disorders which follow alcoholic excesses. I refer to *jaundice*, which is apt to supervene some days after a debauch, and is preceded by more or less decided gastric disorder. It is probable that there is at first simple hyperæmia of the liver, caused by irritation from engorgement with alcoholic substances; but excesses are constantly repeated, and you can easily understand that the hyperæmia will also be reproduced, and will forthwith lead to an inflammatory condition, generally of a bastard character, the ultimate consequence of which will be—according to the nature of the case—steatosis or cirrhosis.

The *salivary glands* have been found by Dr. Lancereaux, to be "soft, yellowish, and having their epithelium manifestly invaded by granulo-adipose degeneration."

The *pancreas* has six times been seen by the same observer to be affected—sometimes altered in the same manner as the salivary glands, sometimes shrivelled and atrophied like the liver of cirrhosis. You see, therefore, that this organ undergoes changes similar to those of the liver, and also, that the mode in which they are produced is similar.

The *serous membranes of the abdomen*—the peritoneum, mesentery, and omentum—are the seat of an excess of fatty deposit, or of adhesive inflammation. At other times, as in a case observed by Dr. Blachez, the peritoneum was studded with ecchymotic spots produced by a mechanism which I shall immediately explain to you. Excessive fatty deposit in the omentum and mesentery, when coincident with the visceral regions which I have pointed out, is sufficient to establish the existence of alcoholism. As to the adhesive neoplasm, it is accompanied by a serous exudation, which is very scanty when the liver is fatty, or, in other words, when cirrhosis does not exist [in cirrhosis, there is ascites]. The neoplastic exudation sometimes produces adhesions of the abdominal viscera. Besides this adhesive inflammation, Dr. Lancereaux has pointed out that there is a granular peritonitis peculiar to drunkards, which is characterized by small granular masses very similar to the tuberculous granulations of acute phthisis. "Along with these lesions," says Dr. Lancereaux, "there was fatty alteration of the liver, and in one case, an ulcer of the stomach. This occurred in robust persons in whose antecedents there was nothing tuberculous."

Were the *vena porta* more frequently examined in cases of alcoholism, it is exceedingly probable that it would be found to be very much altered, as it receives in the first instance the alcoholic substances proceeding from the stomach. Dr. Lancereaux has pointed out the occurrence of inflammation of the vena porta accompanied by pseudo-membranous exudation.

The *pulmonary artery* which receives the venous blood, and consequently the blood surcharged with alcohol coming by the vena cava from the liver, may, in drunkards, present a similar alteration. This, also, has been pointed out by Dr. Lancereaux. "There exists," remarks this able observer, "a form of arteritis which is characterized anatomically by membranous formations on the interior of the vessel. This form of arteritis which I have always met with in the pulmonary artery may determine to a great extent, and in a manner altogether mechanical, coagulation of the blood, leading to obstruction of the vessel and death. The frequency with which it is met with in drunkards does not seem to be fortuitous; and there is every reason to believe that it owes its origin to the abuse of alcoholic drinks."* One of the earliest symptoms is dyspnœa: it increases by slow degrees, and is a symptom the more remarkable that it is not indicated on auscultation by any physical sign, a circumstance which depends upon the cause of the dyspnœa not being seated in the respiratory passages, but in the pulmonary artery itself. There is either slight cyanosis, or there is loss of color in the integuments. If the coagulation of blood be considerable, or if it be within a large trunk of the vessel, the thrombus inevitably causes rapid death from apnœa.

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* LANCEREAUX: Gazette Médicale, Paris, 1862.

You can well understand that it is impossible for the lungs, through the medium of the pulmonary artery, to remain constantly in contact with the alcoholic substance, without their delicate tissue being injured. This is in reality what does occur. In drunkards, every variety of pulmonary lesion is met with, from congestion to inflammation and tubercle.

Pulmonary congestion is the most frequent of these lesions, for it is necessarily the first stage of all the others. It is generally located in the posterior margins and at the base of the lungs, as in cases where there exists adynamia. The pulmonary tissue is flaccid, soft, little aerated, but still capable of being inflated: it has a brownish color, which is with difficulty removed by washing. One stage more—and there is true *hemorrhagic infiltration:* the diseased vessels have burst. Finally, the pleura may be studded with ecchymotic spots. There is seldom an opportunity of observing these lesions, except when the patient sinks under the subacute symptoms, such as *delirium tremens.*

The symptoms are those of dyspnœa with a sensation of constriction in the chest, cough accompanied by mucous expectoration streaked with blood, disseminated crepitant and subcrepitant râles without bellows-murmur: in two words, the signs are those of congestion. The congestion is all the more natural, that a portion of the absorbed alcohol traverses the pulmonary tissue that it may be eliminated during expiration, and that in doing so, it must necessarily irritate that tissue.

Pneumonia, likewise, is the consequence of the lung being thus impregnated with alcohol. Not unfrequently, we find in the centre of the congested points, as I have just said, indurated lobules of a brownish, dirty yellow, or greenish color, infiltrated with blood, pus, or fat. This is bastard pneumonia. There is also met with genuine pneumonia, usually adynamic or ataxo-adynamic, and frequently terminating in suppuration. It is sufficient to point out the frequency of pneumonia in drunkards: to give the signs by which to detect its presence is unnecessary. It is characterized by nervous complications, by the serious nature of the general symptoms, and by the possibility of the attack terminating in suppuration of the parenchyma. Another possible termination of the pneumonia of drunkards is its passing into the chronic state. Resolution does not take place, and the parenchyma becomes indurated. This has been pointed out by Magnus Huss.

Alcoholism debilitates: every affection which debilitates may lead to tuberculization: independently of alcoholism, tuberculization is common: for all these reasons, it can be understood that under the influence of a predisposition or weakness on the one hand, and of constant irritation of the lung on the other, tubercle becomes developed. Bell of New York has refuted the strange fancy that the excessive use of alcoholic drinks protects from tuberculization. He has shown that the reverse of this statement is the truth. My own experience leads me to the same conclusion. The pulmonary tuberculization dependent on alcoholism may be either chronic or galloping; and that is all I have to say to you on the subject.

Hitherto, I have followed the alcohol in its passage from the stomach to the lungs: I have not traced it beyond the venous system or lesser circulation: I now propose to follow it through the greater circulation. Again, I say, that here it is less in quantity, part having been eliminated by the lungs; also, that it is more diluted, and consequently less irritating. However, it sometimes does not the less on that account determine direct lesions of the heart and arteries, particularly of the aorta.

Dr. Lancereaux says that the appearance of the heart is quite special. At first, it is scarcely larger than in the normal state; and is chiefly remarkable for the fatty deposit at its base and on the parietes. The fat does not merely line the heart: it likewise penetrates between the muscular fibres, and induces partial atrophy by the compression which it exerts upon them. Milky-looking spots are observed on the surface. At a later period, the fleshy tissue of the heart is yellowish, soft, and very friable. At a still later date, the muscular fibres are altered, their striated appearance is less manifest, and they have either become granular or fatty. The myolemma is thick, and the cellular tissue, rendered exuberant by irritative congestion, ends by compressing and as it were partially suffocating the muscular tissue.

The endocardium may be affected by alcohol; but this class of lesions has not been well studied. Dr. Lancereaux found the aortic valves below the tubercles of Arantius thickened, white, grayish, and, to a slight extent, contracted or insufficient.

The lesions of the arterial system are a little better known than those of which I have been speaking. Magnus Huss pointed out the existence of atheromatous patches in the thoracic aorta and cerebral arteries of drinkers. In fact, throughout the entire arterial system, traces are to be found of the ravages committed by the passage and contact of alcohol. Dr. Lancereaux has met with—chiefly in the thoracic aorta—patches, more or less thick, irregular in shape, and formed of cellular tissue. It is this which, in its ulterior metamorphoses, is transformed into an atheromatous patch in the large arteries, which is the starting-point of fatty degeneration of the small arteries, and in particular of the small cerebral arteries, where chiefly the lesion has been studied.

On 14th December, 1867, Dr. Blachez presented to the Medical Society of the Hospitals morbid parts in which were well seen, not only the lesions of which I now speak, but also the symptoms to which they might lead. Here, in a few words, is the case to which I refer:

A man, 46 years of age, was admitted as a patient to the wards of Dr. Blachez. This patient was in a state of almost furious delirium which came in fits, during which he cried, vociferated, and endeavored to strike every one near him. During intervals of comparative composure between the attacks, he looked around him with a stupid air. When touched, he cried out that he was being hurt. The entire cutaneous surface was in a state of hyperæsthesia. There was neither paralysis nor coma. The pulse was 120: the skin was covered with sweat.

On the following morning, during the visit, the patient was drowsy, but could be easily roused, though it was impossible to obtain from him any sign of intelligence: he had a fixed look, and talked incoherent nonsense. The hyperæsthesia continued, but without paralysis or contraction. Coma supervened during the morning, without a fit coming on, and without any fresh excitement. Before noon, the patient died.

At the autopsy, there was found "double meningeal hemorrhage," to use the title under which Dr. Blachez has published his interesting case. In my opinion, however, the case was one of hemorrhagic meningitis, which, nosologically is a very different affair. In point of fact, at the convexity of each hemisphere, there was "a trembling gelatinous mass which elevated the dura mater." Upon cutting open the dura mater, a sanguineous effusion was found beneath it. The symptoms had at first been those of meningitis, and the patient had no coma till the last hours of life.

Besides other pathological changes, Dr. Blachez points out the atheromatous patches in the vessels at the base of the brain. Upon the thoracic pleura, there were numerous livid patches, which were evidently ecchymotic. The lungs were exceedingly congested. The internal surface of the pericardium was, like the pleura, covered with ecchymotic patches. Upon the heart, there were patches of a milky color. The internal surface of the aorta, particularly at the arch, was as it were marbled with yellowish-white stains, and in the same situation, the artery was thickened. On the peritoneum, there were ecchymotic patches. The liver was manifestly pale and hypertrophied. On examining it with the microscope, it was found to be less fatty than might otherwise have been supposed. All the hepatic cells could be easily recognized, but many of them contained more than the normal quantity of fat, and some fine fatty granulations were scattered around them. The volume of the kidneys was enormous; this increase in their size resulted from hypertrophy of the cortical substance, which had a grayish color, a greasy aspect, and was studded with small ecchymoses. The cortical tubes were infiltrated with a protean, finely granular substance.

Dr. Blachez asks whether the numerous hemorrhages observed in this patient were not dependent upon an alteration in the blood having been induced by alcohol.* But, concurring with Dr. Peter, I believe that the reality of this alteration in the blood has not yet been demonstrated; and, consequently, I prefer to explain all the phenomena by the positive lesions of the vessels. I entirely concur in Dr. Peter's view, that it is a magnificent example of alcoholism in its acute and rapidly disorganizing form. It is probable that the individual was in the habit of daily imbibing large quantities of alcoholic substances, and that he took more brandy than wine.

"Therefore," remarks Dr. Peter, "it is certain that this man had general inflammation of the arterial system. As a necessary consequence of this general inflammation, the blood must have carried everywhere an irritating substance; and to enable this irritating substance to act so rapidly, and with so much intensity, it must have existed very abundantly in the blood. Now we know, that the irritating substance which habitually produces similar disorders is diluted alcohol.

"Under such circumstances, there is evidently inflammation of the arterial system; and it was this inflammation which caused all the disasters, sometimes exciting inflammation and sometimes inducing hemorrhage. The irritant poison circulating from artery to arterial branch, and from arterial branch to capillary, produced everywhere similar primary disorders, and consecutive lesions, varying according to the diameter or resisting power of the vessel. In the aorta, the white patches are the result of the proliferation of nuclei, the first stage of an irritative process.

"In the kidneys, the ecchymotic spots, disseminated in the form of red spherical points, are apparently hemorrhages in the situation of the Malpighian tufts, that is to say, the minute tortuous arteries are ruptured where they curve, in consequence of the pathological change induced in them by alcoholic irritation.

"That is not all: in addition to the primary vascular lesion, there is a consecutive visceral lesion.

"In the patient of Dr. Blachez, the liver had undergone a morbid change: it was not only increased in volume, but was likewise pale in color, considerably infiltrated with fat, while, at the same time, it was the seat of a proliferation of nuclei of adventitious tissue.

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* BLACHEZ: The Union Médicale for April and May, 1867.

"There was also a morbid state of the kidney, which, like the liver, was pale, and like it was the seat of a proliferation of nuclei of epithelium in the tubular substance, irrespective, I repeat, of the hemorrhages in the situation of the tufts.

"It is probable that similar lesions, if looked for, would have been found in other glands, such as the pancreas, salivary glands, and testicles.

"It is to be regretted," continues Dr. Peter, "that no inquiry was made into the state of the portal vein and pulmonary artery, the vessels which first come in contact with the absorbed alcohol. Had they been examined, there would probably have been found inflammation of their internal membrane, which would have imparted a great degree of certainty to the etiology and pathogeny which I propose.

"To sum up: these numerous lesions—and they might have been found to be still more numerous—seem to me to have been due to one and the same cause, irritation produced by the contact of an alterative substance in the blood, that substance being alcohol.

"If from a purely micographic point of view, the white patches on the aorta are due to a proliferation of nuclei, this proliferation (the primary consequence of the irritative process of Virchow) looked at from a nosological point of view, is only the first stage of inflammation. These nuclei would afterwards have been transformed into fat by retrogression, and one would have had *atheroma*, which would subsequently have become a calcareous plate, by a deposit of salts taking the place of fatty molecules.

"Also, had the patient lived long enough, similar changes would have been observed in the liver and kidneys: there would have been found the fatty liver and Brightian kidneys of drunkards [*reins brightiques des ivrognes*].

"The pathological drama was suddenly stopped at the first act, by death, the result of pachymeningitic hemorrhage.

"This abrupt termination was a fortunate occurrence, if looked at from a scientific point of view, as it enabled us to detect the first phase of a series of lesions which generally we do not observe except in the more advanced stages—that is, in fatty degeneration."*

I have quoted *in extenso* these remarks of Dr. Peter, because they connect by a common etiological chain all the lesions observed in alcoholism, and because they express a pathogenic theory which I accept.

It appears, then, that the patient of Dr. Blachez presented a combination of the lesions of acute and chronic alcoholism. The alcoholic impregnation of the brain caused meningitis, and the morbid change in the vessels led to hemorrhagic meningitis. When the alcoholic impregnation has been great, *delirium tremens* is the result: it causes by its action on the nervous system, in respect of the motor powers, tremors—in respect of sensation, a great variety of affections—and in respect of the mind, dulness, alienation, or dementia.

Were I to discuss at greater length the lesions due to alcoholism, I should unduly extend these lectures: my chief aim has been to show you that alcohol exercises a similar action upon every part of the organism.

There is one organ, however, of which I wish to speak before I conclude: that organ is the *kidney*. It may be affected either with *granular* or *fatty degeneration*. In the former case, the morbid change is chiefly in the cellular tissue of the organ, and is analogous in its nature to cirrhosis of the liver: in the second instance, it is principally the epithelium which is infiltrated with fatty matter. In the former, the kidney gradually diminishes in size, and acquires an unequal surface from contraction of the cellular tissue: the volume of the cortical substance becomes less and less, in consequence of the atrophy occasioned by the thickening of the cellulo-fibrous tissue, particularly in the situation of the tufts. Finally, the epithelium is granular, and is frequently altered. In fatty degeneration, the kidney remains bulky, and retains the normal smoothness of surface; but it gradually loses its color, and becomes yellowish, either wholly or in isolated patches. The tubuli become more voluminous and infiltrated with granulations (fatty for the most part), developed in the epithelial cells, which are turgid and deformed. In both cases, there is Bright's disease, accompanied by symptoms with which you are acquainted. It is impossible to avoid looking upon these lesions of the liver and kidney as similar in their nature, and as originating in the same cause, that cause being the action of alcohol.

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* PETER (Michel): Bulletin de la Société Médicale des Hôpitaux for December, 1866.

Again, it is this very same action, which, by dilating the vessels of the *skin*, produces lividity of the face, particularly of the nose, where the circulation being naturally more slŭggish, the blood circulates still less actively in consequence of the vascular dilatation. Similar changes take place in the glands of the skin as in the other glands: the sudiparous and sebaceous glands generally undergo the granulo-fatty alteration: in point of fact, we generally find the epithelium of the sudiparous glands in a granular state, and the sebaceous glands filled with an enormous quantity of fatty matter. Inflammation is another process by which the sebaceous glands are altered: it is this which constitutes the *acne rosacea* (sometimes copper-colored)—the indelible stigma of drunkenness.

Let me add, in conclusion, that all these lesions are not met with at the same time in the same individual; and that the most common are the lesions of the stomach and liver, after which come those of the nervous centres, and finally, those of the kidneys.[*]

Gentlemen, I have endeavored in this rapid sketch to place before you the manner in which the tissues of the human body are acted upon by alcohol taken to excess. To enable me to do so, I have had to follow it stage by stage in its passage through the organism, and to show you that everywhere it is equally a destroyer; and also, that it everywhere produces lesions essentially similar, presenting only differences in appearance arising from the difference of tissue of the affected organs. It is the aggregate of these lesions, and of the symptoms which correspond to them, which constitute true alcoholism in its acute or chronic form. Whether alcoholism be acute or chronic, it is always the same organs which are injured; and the symptoms observed, though they vary in degree, are always the same in kind. Thus, to name together *delirium tremens*, gastric symptoms, jaundice, and perhaps also disturbance of the urinary secretion, is to give a succint description of acute alcoholism: alcoholic tremors, general dulness of the nervous system, chronic or ulcerous gastritis, cirrhosis, and Bright's disease, are the phenomena of inveterate alcoholism. In three words, the difference presented by the latter may be described as consisting in prolongation of the cause, greater intensity in the effects, and more profound morbid alteration of organs.

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[*] See the following works: RACLE (A.)—"De l'Alcoölisme;" Thèse d'agrégation, 1860. FOURNIER (Alfred)—Article "Alcoölisme" in the Dictionnaire de Médecine et de Chirurgie Pratiques, t. i, Paris, 1864; and particularly the excellent Article, "Alcoölisme," by Dr. LANCEREAUX, in the Dictionnaire Encyclopédique des Sciences Médicales, 1865.

LECTURE LIII.

ON NEURALGIA.

§ 1. *Neuralgia is generally a Symptom either of a Local Lesion, or, more commonly, of a General Affection.—Cutaneous Hyperæsthesia over the Peripheral Expansion of the Nerves, followed sometimes by Anæsthesia.—The Painful Spots indicated by Valleix are not Accurate.—The Spinous Processes of the Vertebræ are always Tender on Pressure, but the fact was not mentioned by Valleix.—The Cause of a Neuralgia influences its Seat.—Periodicity and Intermittence are Frequent Characters of Neuralgia, whatever be its Origin.*

GENTLEMEN: I am averse to treating in this place questions relating to pathology, as they should be discussed elsewhere, but when several cases of the same disease occur at the same time in our clinical wards, or when a remarkable case, full of interest, comes under our notice, it is my duty to take the opportunity of pointing out to you how far clinical cases differ from, or resemble, those which are generally regarded as types; for this practical study, based on observation, leaves in your mind recollections which cannot be effaced, and prepares you, in a remarkable manner, for a study of pathology, which can never be entered upon and completed unless controlled by clinical observation.

Strangely enough, we have at present in our wards four somewhat remarkable cases of neuralgia. At No. 7, in St. Bernard Ward, there is a woman who is suffering from hepatic and intercostal neuralgia, following hepatic colic; at No. 12 is another woman, laboring under rheumatic neuralgia; and at No. 13 a third, suffering from neuralgia of nearly all the branches of the lumbar plexus, following on a subaponeurotic abscess of the iliac fossa; and lastly, at No. 23, there is a chlorotic girl, afflicted with neuralgic pain in various regions, as so frequently occurs in chlorosis.

For the last two or three months you have also had occasion to see other cases of the same kind, to which I called your attention at the time, so that I can now point out to you in a few words the chief forms of neuralgia, and the various modes of treatment to which I have recourse. Let me first remind you that the majority of pathologists have divided neuralgias into two great classes; namely, those which are not due to an organic lesion, of which they are a sympathetic expression, and secondly, those which are dependent on a more or less grave lesion, involving some nerve branches, or compressing or irritating them.

I do not wish to find fault too much with this division, which may aid memory, and may help one to understand neuralgias better; but I wish to observe that, after all, all neuralgias are only symptomatic. Surely there are notable differences, which I shall presently point out, whether the neuralgia occurs, as it so frequently does, in chlorosis, or in chronic lead-poisoning, or in anæmia from various causes, and in rheumatism, or in cases of carious teeth, of necrosed bone, or of a tumor in the pelvis, or of phlegmonous inflammation of that cavity; but whether the neuralgia be due to chlorosis or to a carious tooth, it is still a symptom, in the first case, of chlorotic cachexia, in the second, of the caries of a tooth. As we shall see presently, there is a great difference between these two forms of neuralgia, as regards their obstinacy and their degree of curability, but not as regards *pain*. All neuralgias, regarded as painful affections, resemble one another, with the exception, however, of that neuralgia which I have called *epileptiform*. It is certainly true that the cause of the neuralgia most frequently possesses a manifest influence on the recurrence, the duration, and the period of invasion of the paroxysms of pain as well as on the seat of the pain, but the pain itself exhibits very nearly identical characters. If you recall to mind how I looked for and found painful spots, you must be convinced that the form of the pain did not vary, whether chlorosis, syphilis, rheumatism, marsh miasmata, or an acute inflammatory or a chronic affection, had caused the neuralgia. When the branches of the fifth cranial nerve were affected, the most painful points were at the exit of the ophthalmic of the superior and of the inferior maxillary branches; next to these came the frontal point, which was rarely absent, and then the parietal point, which was less frequently met with; lastly, the occipital nerve was nearly always simultaneously affected, although its origin is independent of that of the trigeminal. It is a strange and inexplicable fact, but which has been constantly present in all the cases which we have carefully observed and noted, whether the fifth was affected by itself, or the occipital nerve as well; pressure made on the spinous processes of the first two cervical vertebræ *always* caused pain, and, in a certain proportion of cases, immediately brought on shooting pain in the diseased nerves.

When the nerves of the brachial plexus were affected, pressure made over the spinous processes of the last cervical vertebræ gave pain; and the same thing occurred when the spinal column was examined in cases of intercostal, lumbar, or sciatic neuralgia.

It may, therefore, be stated in general terms that, in neuralgia, the spinous processes of the vertebræ are tender on pressure at a spot nearly corresponding to the point of exit of the nerve from the intervertebral foramen, and that the pain pretty frequently extends a little further up along the vertebral column. Thus you have seen, how, in individuals suffering from sciatica, pressure on the sacrum gave pain; while, in females afflicted with neuralgia of the lumbar plexus, there was tenderness on pressure of the last dorsal vertebræ.

It might seemingly be inferred from this fact, that the starting-point of the neuralgia is perhaps in the spinal cord, and that the peripheral is only an irradiation of the spinal pain. Yet I confess that it may be equally admitted that the lesion of the periphery of the nerve-trunk, or of some portion of it, transmits to the cord the painful impression which is made so acute by pressure on the spinous processes. Let me add, that the latter view is the more probable of the two, since, in most cases, marked peripheral lesions are the starting-point of neuralgias, as in cases of decayed teeth, of necrosed bones, of tumors of various kinds developed either in the vicinity or in the substance of nerve-trunks, or of inflammations including nerves within their area. On the other hand, it cannot be denied that frequently, particularly in rheumatic affections of the spinal cord, the disease begins in the nervous centre and radiates from it to the periphery.

Whatever be the explanation, however, neuralgia reveals itself by acute pain, when pressure is made on the spinous processes which correspond to the origin, or the point of exit, of the diseased nerves. I have told you, that this applied to the fifth nerve, and that, in no case which I had examined, had I failed to find great tenderness on pressure of the first two cervical vertebræ, as well as of the trunk and branches of the nerve. It is true that in such cases, although the phenomenon is constantly present, it is not so easily or so satisfactorily explained. In neuralgia of the lumbar plexus I can pretty easily understand, from the anatomical condition of the parts, how pain may be excited by making pressure on the spinous processes of the last dorsal, and the upper lumbar vértebræ, but I do not see what relations exist between the first two cervical vertebræ and the trigeminal nerve.

As this phenomenon is almost invariably present, it becomes a valuable element for diagnosis. When, after some external violence, an individual complains of a stitch in the side, there is no tenderness of his spinous processes, and this is also the case at the onset of an attack of pleurisy or of pleuro-pneumonia. Neuralgia is not yet developed; there is merely local pain, which sometimes, however, passes into neuralgia at a later period. But if pain in the side sets in in an anæmic, or chlorotic, or dyspeptic patient, of course, apart from all local lesion, there always is tenderness on pressure of the spinous processes of the vertebræ.

I will give you another illustration. In toothache, arising from the presence of a false tooth with a pivot, the spinous processes are not tender on pressure, however acute the pain may be; but if this pain, which is at first limited to the locality of the tooth, in the lower jaw, for instance, extends to the inferior maxillary division of the fifth, then to the superior maxillary branch, and lastly to the ophthalmic, the spinous processes then become tender on pressure, and the case is one of neuralgia.

The same thing obtains in hepatic colic. Fearful pain sets up suddenly in the pit of the stomach, and in the region of the gall-bladder, and of the ductus communis choledochus. So far there is merely local pain, without neuralgia, and there is no tenderness on pressure of the dorsal spinous processes; but after two or three days spent in acute pain, a sharp pain is frequently complained of in the seventh, eighth, and ninth intercostal spaces, in the shoulder, in the neck, and in the arm on the same side; from that time, neuralgia exists, and the vertebræ become very tender on pressure.

You see, gentlemen, that the apparently subtle distinction which I established just now is founded, and that local pains should not be mistaken for neuralgias, as we possess a precious sign, which enables us to distinguish them. It seems also, in the cases which I have just cited, and in which a local pain gives rise to neuralgia, that the spinal cord is influenced, and then, through reflex action, excites neuralgia, in which it appears to be always involved.

The general condition of the individual, cachectic states in particular, plays an important part in the development of neuralgias. If we find, that persons suffering from chlorosis or from a rheumatic diathesis have a strange liability to neuralgia, which, as it were, develops itself spontaneously in them, it is conceivable that, in such individuals, an acute pain or a very painful tumor will give rise to irritation of the spinal cord, from the centre of which neuralgias will develop with extreme violence. This is, indeed, what occurs in such cases. Whereas, for example, in a woman of robust constitution, chronic inflammation of the uterus or its appendages may exist for a long period without exciting neuralgia, the least irritation of those same parts will, in a chlorotic female, bring on neuralgia of the thighs, of the groin, &c. We had an illustration of this in the case of a young girl who occupies bed No. 27, and whose history I will relate briefly to you. "This young girl is seventeen years old; she has menstruated regularly until this last time, when on her taking a cold bath on the last day of her menstrual period, the menses were immediately suppressed, and she shortly afterwards felt an acute pain in the region of the left ovary. Within a few days she had palpitation of the heart, got out of breath easily, and complained of disordered digestion and of vague pains; she had become chlorotic. She was then admitted under my care, on account of an acute pain in the chest, which she complained of. The pain was so intense that it impeded respiration, but I easily made out that it was merely due to intercostal neuralgia on the left side. As this was apparently dependent on a chlorotic condition, which generally gives rise to neuralgic pain in various regions at the same time, I looked out for some other neuralgia and discovered a lumbo-abdominal and a crural neuralgia, both on the left side also. The patient did not complain of them, however, as her attention was wholly engaged with her intercostal pain which partially interfered with her breathing."

The many examples which I have related to you, and your personal examinations of the spinal column of individuals suffering from neuralgia, have, therefore, shown you how tender on pressure the spinous processes are over the spots corresponding to the presumed lesion of the spinal cord.

There is another peculiarity to which I have drawn your attention at the bedside, and on which authors, who have written on neuralgias, have not been explicit enough, namely, *cutaneous hyperæsthesia* at the points of exit of the nerve-trunks. This phenomenon is best studied in cases of intercostal, lumbar, and crural neuralgia. Its characters are such that they cannot be mistaken, and may almost be regarded as invariable. When the skin is scratched with the tip of a nail, or is gently rubbed with a hard body, as the blunt end of a pencil, the patient complains of a pricking pain, of a sensation of burning, which he compares to that felt on rubbing a portion of skin which has been burnt to the first degree. The acuteness of the sensation varies according to individual conditions difficult to appreciate, for it is somewhat dull in some, and singularly exalted in others. In those regions where the nerve-trunk, from being deepseated, becomes superficial, as in the case of the external popliteal, and the internal saphenous nerves, the track of the painful nerve may be followed with the tip of the finger as far as its cutaneous expansion. In the case of the intercostal nerves, which at their exit break up immediately into extremely numerous branches, the cutaneous pain spreads over a somewhat considerable area, instead of being circumscribed, as in the above instances. This circumstance gives rise to errors in diagnosis every day, and it must be admitted that the patients themselves greatly contribute to mislead us. You remember the young woman who lay at No. 10, in St. Bernard Ward. She had several very mobile neuralgias, and you very often heard her complain of an acute pain in the stomach, which I tried to calm by the internal administration of bismuth, belladonna, opium, &c. One day, on looking out very carefully for the tender spots of an acute intercostal neuralgia of which she complained, I found exquisite tenderness of the spinous processes of the sixth and seventh dorsal vertebræ, and extreme exaltation of cutaneous sensibility over the parts to which was distributed the anterior extremity of the two affected intercostal nerves; and as these nerves send terminal branches to the whole of the epigastric region, the skin over it was extraordinarily painful. I easily understood, then, that the case was not one of *gastralgia*, but of *epigastralgia*, and it gave me the key to the interpretation of many cases which had formerly seemed very difficult to me. When once my attention was drawn to this point in pathology, I saw a good many analogous instances, both in private and in hospital practice.

It is common, indeed, to meet with women who cannot bear a string round their waist, or the least pressure over the pit of the stomach, and, on examining them, it is found that in nearly every case there is intercostal neuralgia with epigastralgia. It is very common also to meet with women who complain of cardiac pains which are merely due to intercostal neuralgia, and as this neuralgia is peculiarly frequent in chlorotic and nervous women, that are more than others liable to palpitations of the heart, it follows that both the patient and her medical attendant believe that there exists a connection between the palpitations and the pain in the side, while the connection is merely apparent. You saw very recently a curious instance of this in a young girl who occupied bed No. 9, in St. Bernard Ward, during the month of November, 1863. She complained one morning of violent palpitations of the heart, and at the same time of acute pain over the cardiac region. It was easy to see that her heart beat more quickly and more strongly than usual, and a soft blowing murmur was audible at the base, coinciding with a continuous blowing murmur in the vessels of the neck; the patient was chlorotic. You remember that, on examining the cardiac region, as I generally do, to look out for intercostal neuralgia, I found considerable hyperæsthesia of the skin, together with tenderness on pressure of the spinous processes of the fourth and fifth dorsal vertebræ. She besides complained of neuralgic pains in various other regions, in the face, in other parts of the trunk, and along the lower extremities. These cases, to which I might add a good many more, prove to you sufficiently the importance of this hyperæsthesia as a symptom, and how it may give rise to many errors when it is wrongly interpreted.

But, gentlemen, there is another phenomenon, quite the reverse of the above, which sometimes, although more rarely, exists in neuralgia, namely, *anæsthesia.* It often follows on idiopathic non-spinal neuralgias, that is, on neuralgias apparently of rheumatic origin, or due to a slight lesion of the cord. At the outset, and often for a somewhat lengthened period, there is only an exaltation of sensibility; but when the affection has lasted a long time, the exaltation is replaced by a diminution, and lastly by a complete loss, of sensibility. In such cases, I admit, there is something more than a neuralgia, and the anæsthesia may be regarded as the consequence of a change in the structure of the cord or of the nerve-trunk, as occurs in cases of neuritis. It is still pretty frequent to find cutaneous anæsthesia succeed hyperæsthesia, especially in herpes zoster. This complaint, as you are aware, is often attended with persistent neuralgic pains, and I have known these to last sometimes more than ten years, until the patient's death. When the pains have lasted for a long time, however, it sometimes happens that the hyperæsthesia is followed by a curious kind of insensibility, of which the patient complains bitterly. Anæsthesia comes on pretty frequently also in the course of sciatic neuralgia, particularly over the area of distribution of the branches of the external popliteal nerve, but only when the affection has lasted a very long time.

I have always felt surprised, gentlemen, at the facility with which the profession accepted certain views respecting the diagnosis of neuralgia propounded by Valleix,* namely, the *superfical tender spots.* You have witnessed the scrupulous care with which I look for these spots in every one of my cases. In cranial neuralgias the spots are those indicated by Valleix, and they were already known before him. The fifth pair divides into three principal branches, and it is at their point of exit that pain is most particularly felt, namely, over the supraorbital notch, where the ophthalmic branch becomes superficial, over the infraorbital foramen, which gives passage to the superior maxillary branch, and over the mental foramen, through which emerges the inferior maxillary division of the nerve. This is easily ascertained by making pressure over those spots with the blunt extremity of a pencil, and even with a finger. But there are other tender spots as well. When the supraorbital neuralgia is rather intense, the nasal branch is extremely tender, and pain is excited by gently compressing the skin over the point of exit of this small nerve. Although the ophthalmic branch, on leaving the supraorbital notch, breaks up into numerous ramifications over the forehead, and although on dissection no branch can be seen, like the nasal, becoming suddenly superficial from being deep-seated, yet pressure generally causes a somewhat acute pain over the frontal eminence. Pressure also over the parietal eminence is slightly painful, although that branch of the fifth which goes upwards in front of the ear, and ramifies in the scalp as far as the parietal eminence, is not subcutaneous like the nasal branch of the ophthalmic. There is another remarkably tender point in the track of this small branch, namely, over the zygomatic process in front of the ear.

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* Traité des névralgies, Paris, 1841.

After all, the fact observed by me is in accordance with others already made out; for when nerves are affected with neuralgia, there are two points where pain is most felt. 1st, where the branch, after being deepseated, becomes superficial; 2d, where the branches and small twigs of the nerve terminate in the skin. There is nothing surprising, therefore, in the fact that the small temporal branch and the ophthalmic division of the fifth are painful only in the two extreme points which I have just indicated.

As to the occipital branch, it is generally painful where it comes out of the substance of the muscles, and it is tender for a pretty short distance.

Valleix was guided by his anatomical knowledge more than by facts, when he pointed out the spots where pain was particularly felt. Seeing, for instance, that in the face pain was chiefly felt over the points of exit of the nerves, namely, where they issue from the bones, and become subcutaneous, he thought that the same thing occurred in the case of other nerves, but this is not borne out by observation. He then indicated three tender spots in intercostal neuralgia, which is such a common affection; the first, situated over the angle, the second, about the middle of the rib; and the third, about its sternal end. Now, his assertion is perfectly unfounded, and a few days' researches are sufficient to prove it.

But where, after all, are these tender spots found? They are three in number, of which two are more important than the third, namely, one, which has not been mentioned by Valleix, but is of considerable value in diagnosis, and which I have termed the *spinous point;* and another, which is nearly as important, which I have called the *spot of peripheral expansion.* The *spinous point,* as its name indicates, is situated over the spinous processes of the vertebræ, and since my attention has been drawn to it, I have never known it to be absent. It is easily found out by making pressure in succession on the spinous processes of the vertebræ, beginning with the first two, immediately beneath the occipital bone, down to the loins. When the tender spot is reached, the patient makes an abrupt movement, and tries to avoid being touched, and sometimes even cries out. Pressure on the vertebræ above and below the point gives no pain.

The *spinous point* is met with not only in neuralgia of the face and of the trunk, but also in sciatic neuralgia, as was the case in two women suffering from this affection, who were under my care in November, 1863. Before I examined them, I must say that I expected to find tenderness on pressure of the spinous processes corresponding to the lumbar swelling analogous to what is met with in intercostal neuralgia; but to my great surprise, no pain was produced by pressure, however strong, made on the spinous processes. Pressure on the sacrum, however, gave rise to the same kind of pain as that felt on pressing the dorsal vertebræ in intercostal neuralgia. This circumstance is probably owing to the neuralgia only beginning on a level with the sacral plexus.

In one of these cases, the neuralgia was of an erratic character, and you could observe the great difference which exists, in this respect, between sciatic neuralgia and neuralgia of the crural nerve and of other branches of the lumbar plexus. For the sacrum ceased to be tender on pressure as soon as the neuralgia disappeared, and deep pressure made over the spinous processes of all the lumbar vertebræ gave no pain, while pain was complained of when the eleventh dorsal vertebra was pressed, which nearly corresponds to the origin of the nerves of the lumbar plexus.

You may perhaps recollect a woman who lay in bed No. 14, in the nurses' ward, who suffered from various neuralgias of a rheumatic origin, and whose spinous processes were tender in several places. When pressure was made on her occipital protuberance, which is the analogue of the spinous processes of the vertebral column, and which may be regarded as the spinous process of the great cranial vertebra, considerable pain was excited in the branches of the fifth, especially in the ophthalmic branch of Willis. In some cases, and by a similar process, a pretty sharp pain is excited in the sciatic nerve by making pressure on the spinous processes of the sacrum. When the spinous point has been detected, the *spot of peripheral expansion* has to be determined; and in so doing, one must be very careful to keep free from error. Suppose the case, for example, to be one of neuralgia of the sixth intercostal space. The spinal point will correspond to the fifth and sixth spinous processes, and the peripheral tenderness will be complained of over an antero-lateral portion of the chest corresponding to the sixth intercostal space. Now, whereas the upper ribs form nearly a right angle with the vertebral column, the middle and lower ribs form an acute angle, so that these ribs go from below upwards as far as their corresponding cartilages, which are, on the contrary, placed horizontally or even obliquely from below upwards. If therefore on going from the tender spinous process, one does not keep to the rib and the intercostal space, the relation cannot be made out which exists between the *spinous tender point* and the *spot of peripheral expansion*. For an imaginary line, drawn round the chest perpendicularly to the vertebral column, does not follow the direction of the ribs in order to reach the median line. Now, as the fifth and sixth spinous processes are exactly opposite the middle of the sternum, this is where the tender spot of peripheral expansion is looked for; whereas it is situated three or four inches lower down, that is to say, about the end of the rib, near where the rib joins the sternal cartilage. I have dwelt on this point, gentlemen, because I have, on several occasions, when going round the wards, seen students unable to find out the antero-lateral tender spot in an intercostal neuralgia, after they had succeeded in detecting the *spinous point*. I have told you already, that there is cutaneous hyperæsthesia over the area of peripheral expansion, and how to recognize it. This phenomenon is nearly constant.

When the neuralgia is due to a cachexia, the nature of the latter has also a somewhat marked influence on the seat of the neuralgia. Thus, chlorosis gives rise to neuralgia in several regions at the same time, although the trigeminal nerve and the nerves of the solar plexus are those which are most commonly affected. Gastric and intestinal neuralgia is rarely absent in women whose constitution is enfeebled by uterine hemorrhages or by leucorrhœa. In the cachexia due to marsh miasmata, the ophthalmic nerve is the one most generally affected. In rheumatic individuals, the occipital and the sciatic nerves are most frequently attacked. These statements must be taken generally, of course, and are liable to very numerous exceptions.

In cancerous and syphilitic cachexias, neuralgia has no favorite seat, but sets up within the area of irritation or of pain developed round some local lesion; thus we meet with sciatic neuralgia in cancer of the pelvis, and with lumbo-abdominal and crural neuralgias in cancer of the kidneys and of the uterus; and with temporal and occipital neuralgias, and brachial neuralgias, when a syphilitic tumor develops itself on the parietal bones or over the humerus.

It is rather strange, gentlemen, that diseases of the nervous system should very often give rise to intermittent phenomena. Epilepsy, catalepsy, certain kinds of chorea, and many other convulsive affections, pretty frequently assume not only an intermittent but also a periodic type. The same thing occurs with many neuralgias, visceral or external. This *intermittent* and periodic character shows itself even in organic affections of the greatest gravity. Thus I saw a lady, in the year 1845, in consultation with Messrs. Récamier and Maisonneuve, who was affected with cancer of the inner wall of the uterus. She had every day paroxysms of awful pain, recurring at exactly the same time; the pain was seated in the hypogastrium and radiated to the kidneys, to the buttocks, and to the thighs, along the track of the principal nerve-trunks. It lasted from three to four or five hours, and then ceased, to reappear on the following day exactly at the same hour.

In the year 1850, I saw another lady afflicted with the same complaint, with my excellent friend Dr. Lasègue: the cancer involved the inner wall of the womb, and it seemed as if this form of the disease, which is somewhat rare, had the sad privilege of giving rise to intermittent and periodic pain. I have never known another case in which such excruciating pain was felt. When the pain was at its maximum, the unfortunate patient rolled on the floor, uttering fearful groans. It recurred every day, but, strangely enough, it returned from half an hour to three-quarters of an hour or an hour later each time, so that, in the space of a month or more, the hour of attack had gone round the clock. The neuralgic paroxysm did not last more than four or five hours, and then quiet was restored; all pain ceased, and there merely remained a very slight sense of fatigue and weight in the hypogastric region.

Again, in 1862, I saw in Paris, with Messrs. Nélaton and Bouillaud, a Greek lady, who had exceedingly profuse menorrhagia and awful neuralgic pain in all the branches of the lumbar plexus. She had fibrous tumors of the walls of the uterus, protruding into the cavity of the viscus. I attended her for nearly a year, and during that period the pain recurred every day with hopeless violence and obstinacy, between twelve and two o'clock, and ceased about five or six in the day. Sometimes, though rarely, it occurred during the night, but it then lasted a short time only. The patient was nearly well in the intervals, with the exception that she felt extremely weak in consequence of the loss of blood, and of the nervous shock caused by such intense pain. I need not add that, in these three cases, bark in all its forms was largely administered, but that it never succeeded in modifying, in the least, the pain or its periodic recurrence.

You may have seen at No. 32, a young woman whose case I have often quoted, for it is peculiarly interesting, and affords a sad illustration of the influence which a local and persistent pain may exert on the whole system. She lost her left eye after a grave attack of ophthalmia, but as she preserved a very mobile stump, she has been able to wear an artificial eye which is moved by the stump, so that it is very difficult to find out her deformity. The artificial eye, however, being a foreign body, has irritated what remained of the globe of the eye, and given rise to a supra and an infra orbital neuralgia, just as might have been caused by a foreign body introduced into a carious tooth. For a very long period the affection was limited to the two upper divisions of the fifth nerve, but the continuous pain and loss of sleep have brought on a strange nervous susceptibility, and, subsequently, multiple neuralgias, at first in the other side of the face, and next in the cervical, the intercostal, and the lumbar nerves, &c. You will often meet with the same phenomena, in women particularly, as a consequence of local lesions which have given rise to neuralgia. Thus, it is not uncommon to find a false tooth with a pivot, which at first merely caused a very sharp local pain, and later, neuralgia of that portion of the trifacial nerve which supplies the diseased jaw, ultimately bring on neuralgia of all the branches of that nerve, and at last, nearly general neuralgic pain.

The evolution of the neuralgia commonly takes place after the following manner: when the nerve has several branches, the pain sets up in all the branches in succession, as if the lesion had extended from one branch to the trunk, and thence to the other branches. In the case of a plexus, the connection between its various branches produces the same effects as those observed in a nerve with a single trunk. This occurred in a woman of 38 years old, who was at No. 13 in the nurses' ward. She had been delivered four months previously, and complained of a lancinating pain in the whole of the anterior aspect of the thigh. The hypogastrium was tender on pressure along the track of the crural nerve, and in the right iliac fossa, but there was no engorgement of the fossa. The pains recurred chiefly about seven in the evening, and lasted two hours; they came on after a frontal headache, which disappeared completely. At the end of six days, a tumor with a round contour was detected in the iliac fossa; five days later there was very marked fluctuation, and the pain in the thigh had increased considerably, and extended into the knee, impeding the movements of the limb. Pinching of the skin on the anterior and inner aspect of the thigh gave great pain. I requested Mr. Jobert to open the swelling, and by means of an incision parallel to Poupart's ligament, this skilful surgeon opened the abscess, which was situated deeply under the fascia iliaca, without wounding the peritoneum, and gave issue to a tumblerful of laudable pus, which had no fetid smell. The pain in the thigh ceased immediately after the operation, and from that time, the crural and the lumbo-abdominal neuralgia has never appeared again.

In this case, then, the pain was so connected with the existence of the inflammatory lesion that it began and ended with it; and on the other hand, the neuralgic pain began in the branches of the lumbar plexus involved in the inflamed tissues, and extended next to all the branches of the nerve-trunk, and, lastly, to all the branches of the plexus.

The connection between all the branches of a plexus exists likewise between all the nerves that issue from the spinal cord, which may be regarded, to a certain extent, as a real plexus. What I have said respecting the tenderness, on pressure, of the spinous processes, which is so constant in neuralgias of the head, trunk, and limbs, accounts in some measure for the influence which the neuralgia may subsequently exercise on the development of other neuralgias in regions that are very distant from the primary one, although this influence cannot be understood at first sight.

§ 2. *Neuralgias of Rheumatic Origin.—Their Multiple Manifestations.—They frequently Alternate with the Articular Pains.*

At No. 31, in St. Bernard's Ward, is now a woman who is one of our hospital nurses. This is the fifth or sixth time that she comes under my care. The prominent point in her case is the rheumatic diathesis under which she labors, and which is characterized by the diversity of its manifestations. She sometimes has rheumatism of the spinal cord, with nearly complete paraplegia; sometimes her brain is affected, and she lies in a kind of stupor, without expression in her eyes, while her ideas are confused, her head feels heavy, and she is unable to move. This time, the patient suffered from pain in her joints; both her hands were affected, especially the metacarpo-phalangeal articulations, which were swollen, red, and painful, and then other joints were attacked in succession. A few days afterwards, the articulations got well, and then neuralgic pain set up in various branches of the brachial plexuses, either in several simultaneously, or in succession. A fortnight had scarcely elapsed when the brain became affected again, and the previous symptoms returned, while, after a few days, symptoms of congestion of the cord showed themselves, attended with great feebleness of the lower limbs, a true incomplete paraplegia. These phenomena disappeared and were replaced by neuralgia of the lower limbs; and at last, about October 20, six weeks after her admission, fresh pains came on in the joints again, proving the rheumatic origin of all the other symptoms. Both knees were swollen and painful, and fluctuated markedly, while at the same time, the external saphenous nerve on the left side was affected with neuralgia.

A week after this the patient felt very little pain in her knees, and had no neuralgia of the left saphenous nerve, but other nerves were attacked: the supraorbital, the parietal, the occipital, and the frontal, on the left side, and, as is usually the case, there was tenderness on pressure of the spinous processes of the first two cervical vertebræ. At the end of another week, she had neuralgia of the sixth intercostal space on the left side, and exquisite sensibility of the skin over the epigastrium on the same side.

Thus in the space of two months, this patient has presented symptoms of rheumatism, of a flying, but of a very painful nature, affecting sometimes the cerebrospinal axis or its envelopes, sometimes the articulations, and sometimes, again, various nerve-trunks. The implication of the joints is proof sufficient of the rheumatic nature of all the phenomena. There is a peculiarity also to which I must draw your attention in this case, namely, that, however various the neuralgias were, they always showed themselves on the left side exclusively.

This case then exhibits the influence of rheumatism on the production of neuralgia; and it frequently happens that individuals suffering from pain, evidently seated in the joints, and attended with swelling, are seized with neuralgias when the joints are free from pain, while they again complain of articular pain when the neuralgia disappears. In some cases, as in that of the young girl who is now at No. 9, and in whom, by dressing with morphia blisters made by ammonia, neuralgia of the trifacial nerve was removed, there may be at the same time pain along the nerve-tracks, and pain evidently confined to joints.

I must explain myself further, in order not to leave on your mind erroneous impressions, and to remove as much as possible all confusion arising from the too elastic appellation of rheumatism.

Acute articular rheumatism is a name generally given to a pyrexia characterized by very sharp febrile action, by considerable and generally transitory effusion into the joints, and by a tendency to implicate extra-articular synovial membranes, the serous membranes of the chest and of the brain, and the lining membrane of the heart.

I have not here to justify and defend the name given to this disease, but it differs essentially from another complaint which also attacks the joints, and is known under the name of *gout*. It is as different again from another affection, which is termed *rheumatism*, and which affects joints, muscles, and limbs in their continuity, without nearly ever giving rise to effusions into joints, and in which serous membranes and the heart are never implicated. This last form is certainly the most frequent of all, and is the one which particularly attacks nerve-trunks, and causes those neuralgias, which, from want of a better term, I have called rheumatic. In gout, properly so called, neuralgia occurs occasionally also, but much less frequently than in the non-febrile form of rheumatism. Rheumatic neuralgia is much more mobile than neuralgias dependent on a cachectic condition, as chlorosis, for example. It has a remarkable tendency to attack the brain and spinal cord, but in general temporarily only. In some cases, however, it fixes itself on the nervous centres, and causes paralytic symptoms, probably due to some superficial lesions of the nerve-trunks or their roots.

§ 3. *Syphilitic Neuralgias should not be confounded with the Pains due to Exostoses.—There is no Tenderness on Pressure of the Spinous Processes in cases of Pain due to Exostoses, or in Pleuritic Stitch.*

The pains which in syphilis are felt in the bones, simulate neuralgia; and it sometimes happens, as I shall tell you presently, that when the bones are diseased in syphilis, real neuralgia is set up from compression of nerve-trunks. Thus, a bony tumor in the pelvis, or an exostosis developed on the track of some branches of the fifth nerve, may give rise to neuralgic pain of extreme violence. When the pain is exactly limited to the seat of the exostosis it no more deserves the name of neuralgia than does the pain caused by a whitlow or an abscess. Yet, I am aware that when the growth is situated exactly over the parietal eminence, the pain does not differ much from that caused by neuralgia of the ophthalmic nerve; and you may remember a case bearing on this point, namely, that of a young woman who lay in bed No. 7, in St. Bernard Ward. She complained of an exceedingly acute pulsatile pain, resembling neuralgia in every respect, in the right frontal eminence. On examining her, I found an exostosis there, and swelling of the periosteum of both mastoid processes, which were equally painful.

On close examination, however, there will be found certain characters which enable one to recognize neuralgia, properly so called, from intra-osseous pain occurring in syphilis. Thus, ask yourselves how often you have seen true neuralgia of the parietal eminence exactly limited to the point of exit of the small branch which comes off from the outer frontal branch of the ophthalmic. In neuralgia it is easily ascertained that the principal branch is always painful over the supraorbital notch, whereas it is quite the reverse in osseous pain. Excessive pain is felt on pressing the most prominent part of the bony growth, but by carefully exploring the neighboring parts, it is found that the pain diminishes in proportion to the distance from the central spot; in a word, the same thing happens as in the case of a boil, the pain of which diminishes in proportion to the distance from the seat of the boil.

The difference is still more striking if the *tender spinous points* be looked for. You know that, up to this time, there has not been a single case of facial neuralgia under my care, in which the posterior surface of the first two cervical vertebræ was not very tender on pressure; while you saw that this essential sign was absent in the young woman who complained of pain in the frontal bone, simulating neuralgia.

I told you just now that syphilitic bony growths may, by compressing a nerve-trunk, give rise to true neuralgia, just as any other tumor. In such cases the pain will not be exclusively seated in the growth, but along the track of the affected nerve. I cannot say whether in such cases there will be tenderness on pressure of the spinous processes, as in other neuralgias, for since my attention has been directed to this point, I have not had the opportunity of seeing a single case of neuralgia due to the presence of a tumor. The absence of tenderness on pressure of the spinous processes is useful again for distinguishing pleuritic from neuralgic pain. You remember a young woman who, in November, 1863, was placed at No. 1, in St. Bernard Ward, and was affected with double acute pleurisy. I gave a clinical lecture on her case. On the third day of her getting ill she had, on both sides, amphoric, cavernous respiration, and gurgling, all which disappeared completely at the end of eight days, as she became convalescent. She had pleuritic pain on the right side. I took the opportunity, at the time, of showing you first that the pleuritic stitch generally occupied in the walls of the chest another seat than intercostal neuralgias, for it rarely happens, indeed, that in front it extends beyond a line drawn perpendicularly from the nipple to the abdomen; while intercostal pain, on the contrary, is felt in front of this line, and is diffused over the base of the sternum as well as over the epigastric region. Again, slight pinching and scratching of the skin gave no pain where the pleuritic pain was most sharply felt, whereas the reverse obtains in neuralgia. If, on the other hand, deep pressure was made over the painful intercostal space, it was easily ascertained that the pain was deeply seated, and increased in intensity in proportion to the degree of pressure. Lastly, as in cases of frontal exostosis, the pain corresponded to the inflammation.

All the spinous processes could be squeezed hard without giving the least pain; so that this is an important distinction between intercostal neuralgia and pleurisy; and I cannot therefore admit the views of those pathologists who believe that pleurisy gives rise to a neuralgia, and that this constitutes the stitch in the side which characterizes inflammation of the pleura.

Again, in the case of the tenderness on pressure, which is in many cases met with at the outset of tubercular phthisis, when pressure is made on the first intercostal space in the vicinity of the sternum, a pretty sharp pain is always excited on the side where tubercles are deposited; but this pain, which is evidently connected with the chronic pleurisy developed round the upper lobe of the lung, is neither accompanied with tenderness on pressure of the spinous processes, nor of the area of peripheral expansion of the nerves, as you may easily verify in all cases of phthisis. Hence, this differential sign may be of some value in diagnosis.

It is in general pretty easy to recognize neuralgias of syphilitic origin. In most cases they are due to an outward lesion, pretty readily detected, such as an exostosis, periosteal swelling, a node, inflammation of the mucous membrane, ulceration, or necrosis.

§ 4. *Treatment of Neuralgias.—The First Indication is, to give Relief.—Powerful Effects of Narcotics.—Later the Specific Cause is to be Combated.—Marvellous Effects of Revulsion in all its Forms.*

It is a well-understood thing, gentlemen, that you are not to expect, in our clinical conferences, a complete description of neuralgias, and in some sort a treatise on the subject. As several cases of neuralgia came at the same time under observation, I embraced the opportunity of giving you some general notions on these affections, which are often considered hopeless, both by physician and patient. I have been particularly anxious that these cases should enable you to judge of the effects of the principal modes of treatment which are generally had recourse to in neuralgias.

A general indication is that of relieving the pain, whatever may be the cause under the influence of which the neuralgia has developed itself. There are some cases, undoubtedly, in which the cause may be removed immediately, and the pain disappears with the removal of the cause. Thus, neuralgic pain due to a neuroma ceases at the very moment when the portion of nerve in which the tumor has been developed is cut away; and in the same way, neuralgic pain in the fifth nerve is sometimes instantaneously removed by the extraction of a diseased tooth; and in such cases it is not only the toothache which ceases, but also the reflex neuralgia which had affected the other branches of the trifacial nerve.

But it rarely happens that we can thus get at the cause, and remove it in a moment. When the neuralgia is dependent on a diathesis, a prolonged treatment is needed, and in many cases the cause cannot be removed. In very many instances we fail to cure the rheumatic, the gouty, and the herpetic diathesis, and we are, *à fortiori*, powerless against tumors of a malignant nature, cancers, and fibrous tumors. In such cases we must, above all, calm the pain, if possible, and either later or at once try to act against the cause which has produced or determined the neuralgia. Even when we can act upon the cachexia to which the neuralgia is due, as in chlorosis, anæmia, and syphilis, the influence of general treatment being essentially slow, our first duty is to moderate the violence of the pain, while trying to modify the general condition of the patient.

It is all the more important to follow this rule, that the neuralgia itself often prevents the general treatment from being successful; for a chlorotic female, for instance, will not bear the steel and the bitters which are so needed in her case, if she be continually a prey to horrible pain, which deeply shakes her nervous system, and disturbs all her functions. Of the means in our power for relieving the pain, drugs which have a *stupefying* influence rank first, such as chloroform, ether, opium, solanaceous preparations. Cyanide of potassium comes next, and only under circumstances which I shall presently indicate.

Next in order, again, come turpentine, irritating applications, acupuncture, electropuncture, faradization, and warm baths of very prolonged duration. Lastly, specific remedies, when there is a specific cause,—iron, bark, mercury, &c.

This summary indication of remedies which are so numerous and so different, would be of little help to you, if I did not studiously enter into somewhat minute details respecting those various methods of treatment, without which they are apt to lose their power and their opportune application.

When the neuralgia is superficial, and is, for instance, seated in the temples, the forehead, or the scalp, *belladonna* or *atropia, applied locally*, is sufficient in a pretty large number of cases. Solutions of atropia have the great advantage of being very powerful, and of not messing the clothes or the skin. I generally use the following formula:

℞. Atropiæ sulphatis, gr. v.
Aquæ destillatæ, ℥iij. Solve.

Compresses steeped in this solution are applied over the painful parts, and covered over with a piece of oil-silk so as to prevent evaporation, and the whole is kept in place by a bandage or a handkerchief. The application is renewed several times in the twenty-four hours, and continued for at least an hour each time.

The efficacy of this solution varies in different individuals, so that the dose of atropia should be diminished or increased according to the effect produced. When there is considerable impairment of sight and dryness of the throat, the quantity should either be diminished, or the application be less frequently renewed. On the other hand, when the absorption of the drug is marked by slight phenomena, while the pain is not relieved, the dose should be increased, and the compresses applied almost continuously. When the neuralgia occupies a very limited area, a much more concentrated solution of atropia should be used, namely, three grains of atropia dissolved in about half an ounce of water, with the addition of a small quantity of alcohol; this should be used in drops rubbed on the part with the finger. This is done two or three times in an hour, and a very powerful effect is often obtained in this way. When the neuralgia affects the scalp, and involves the occipital nerve, for instance, the roots of the hair and the scalp should be well wetted, and absorption then takes place with very great facility. A solution of atropia, applied locally, is pretty successful also in neuralgia of the superficial cervical plexus, but is not so useful in intercostal and brachial neuralgia, and loses all efficacy in neuralgia of the lumbar plexus, of the sciatic nerve, and of the abdominal viscera. Yet, you must not think that it is always useless in such cases.

The extract of *Datura stramonium* and of *Belladonna* may be substituted for the atropia. Formerly ointments made with axunge or cerate were used; but the fatty matters diminished the rate of absorption so much that the treatment was often useless. Glycerin and starch are now used instead, and when the extract is mixed with them, in the proportion of one-third or of one-fourth, a paste of the consistency of an ointment is obtained, which offers the great advantage of spreading easily on the skin and of being soluble in water, so that if a piece of wet lint covered over with oil-silk be laid over it, the skin is placed in the most favorable conditions for absorbing the remedy. As good results are obtained in this way as with atropia, and although the skin of the face and of the throat, and the hair may be a little soiled, the mixture offers immense advantages in regions to which soothing fomentations or poultices may be applied, such as the walls of the chest, the abdomen, and even the limbs. In a word, since this combination has been more frequently used in practice, there has been a greater proportion of cases in which alleviation, if not the cure of pain, has been obtained, whether the pain were merely neuralgia, or were due to an organic lesion, or even a local inflammation.

Opium, in all its forms, is far from being as useful as the preparations of solanaceous plants. The *salts of morphia* offer this immense advantage, however, that they can be applied to the raw surface of the skin, an important method of treatment of which I must speak with some details.

It is not an indifferent matter how the true skin is exposed. A blistered surface made by means of cantharides is far from giving the same result as one obtained by means of ammonia, and in the latter case the results are not always the same everywhere. When cantharides are used, a morbid process goes on in the skin, which probably lasts a pretty long time after the blister is removed, and impedes absorption to a certain extent. It is not easy for me to tell you the reason why, but it is a clinical fact; and the same dose of sulphate of morphia sprinkled over the surface of the skin made raw by means of cantharides, produces considerably less effect than when ammonia has been used. I entered into very minute details in my treatise on Therapeutics,* on the *mode of using ammonia* for raising a blister, and on the manner in which the blister should be dressed. The other day, in a young woman at No. 31, who was suffering both from chronic peritonitis and neuralgia of the fifth pair, I raised a blister myself with ammonia, first, in order to show you how it should be done, and secondly, that you might see the rapidity with which salts of morphia are absorbed, and which cannot be credited unless it has been actually seen. I had recourse to the simplest plan. I filled three-fourths of a thimble with very dry cotton-wool well pressed down, and placed in the remaining fourth another piece of cotton-wool steeped in caustic ammonia. I then held the thimble over one temple for about five minutes, and on removing it you could see that the skin which had been in contact with the ammonia was rather paler than the rest, and that all round it the integuments were congested. By passing the finger over the circular mark left by the thimble, the epidermis was seen to move and get wrinkled, thus showing that it was detached. On then rubbing the surface with a piece of linen rather roughly the epidermis was entirely removed, and the cutis vera exposed. I next made a paste of semi-liquid consistency with a drop of water and one-fifth of a grain of morphia, and laid it over the denuded portion of skin; lastly, I covered the spot with a round piece of oil-silk kept in place by a larger piece of sticking-plaster. I shall presently tell you why I had recourse to this kind of dressing.

At the moment when I laid the paste over the raw surface, I asked you to look at your watch, and wait for the first signs of narcotism. I made the patient sit down in order that these signs should be evident. Scarcely had a minute and a half elapsed when she complained of flushings of the head; in another minute she felt giddy, and lastly, three minutes after the dressing had been applied, she felt such malaise that she could not sit any longer: she then laid down, feeling sleepy, and by that time already, as you heard her declare, her pain had diminished markedly. On the following day, as you may remember, the phenomena indicating absorption of the medicine showed themselves with still greater rapidity; but they were long before appearing on the third day, and they could be scarcely detected in the course of it; but when, on the next day, the small sore was dressed, this apparent anomaly was explained, for the sore had almost healed up, and the greater part of the morphia still remained on the surface of the skin.

I called your attention, on the second day, to a rather important point, which would otherwise have been unnoticed. The skin looked raw when I removed the dressing, and yet I told you that there was a thin fibrinous membrane on it, which I then removed by gently rubbing the surface. Bear this simple fact in mind, gentlemen, for if you used the morphia again without removing the false membrane, it would be more slowly and less completely absorbed. You saw at once the reason why I dressed the raw surface as I did on the first day. Had I merely used a strip of sticking-plaster or a piece of rag rubbed over with some fatty substance, part of the morphia would have been wasted in the dressing; and the fibrinous secretion of the sore, instead of forming a false membrane, would have soaked the dressing, so that when the morphia came to be used again, the skin would have been seen to be irritated, and much less capable of absorption than when the thin false membrane which has formed underneath the oil-silk is removed.

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* Traité de Thérapeutique (Trousseau et Pidoux), Art. Ammoniaque et Opium.

There is another circumstance which I wish you to remember. When a raw surface made by ammonia is dressed with morphia, the patient feels stupefied two or three minutes afterwards, and the effects which are proper to opium go on increasing for several hours, in a degree which varies considerably according to the age and sex of the patient, and according also to certain circumstances which cannot be made out. Now, if on the first day the narcotic effect have been moderate, one is tempted to increase the dose on the following day, on the supposition that the system is already used to the opium and will feel its influence much less the second time. But the fact is, and I made you notice it, that the first effect of the remedy is still more rapid than on the previous day, so that it is not uncommon to meet with women who feel perfectly giddy a minute or a minute and a half after the use of the salt; and again, the effect produced is unquestionably greater on the second day, even when the same dose has been used. This is due to a circumstance which may be easily recognized, provided some care be taken. When the ammonia has just been applied, there is a violent irritation of the true skin, which for nearly an hour is attended with a very abundant secretion of serosity. This trickles from under the dressing, and if you taste it (as I have often done), you will find that it is extremely bitter, from the presence of the morphia, which is dissolved in it. Hence it follows, that a variable quantity of morphia is carried away and is consequently not absorbed; whereas, in the same evening or on the following day, when the false membrane is removed, the true skin no longer secretes any serosity, the whole of the salt remains in contact with the surface of the sore, and the narcotic effect necessarily increases. Hence the rule that, in order to produce the same effects, a smaller dose of morphia should be used the second time. Anyhow, gentlemen, a small quantity of morphia should alone be used in dressing a raw surface made by ammonia, for absorption is nowhere so active as over the skin deprived of its cuticle, and grave accidents may result from the use of high doses, from the first. Never begin, in the case of a woman, with more than one-fifth of a grain; and in that of a man, with more than two-fifths; take care to increase this quantity only when you have ascertained how your patient bears the remedy.

The first effect of the morphia is marvellous; a few minutes are sometimes sufficient to calm an awful pain; and it rarely occurs that great relief is not afforded in a violent neuralgia. But there is a world-wide difference between this and a real cure, and the pain generally returns when the narcotic effect of the drug has passed off. It is necessary then to keep the system under the influence of the remedy for a more or less prolonged period; and a fresh application is to be made in the evening, and twice again on the following day. In that way the pain may, in a great number of cases, be entirely suppressed for some time.

I have already told you that, on the third day, the raw surface made by ammonia no longer absorbs, so that a fresh sore has to be made, in the same manner, in the vicinity of the former, or in another part, according to the intensity of the pain. Care should always be taken that the skin is irritated sufficiently to produce detachment of the cuticle, but never so as to give rise to a very prominent bulla, because otherwise superficial ulceration of the skin would be produced, and absorption rendered more difficult, and on the other hand, a persistent scar would be left behind, a circumstance to be taken into consideration when dealing with the face and other parts exposed to view.

The narcotics should thus be persevered in for eight, ten, or fifteen days, sufficiently long to destroy a vicious habit of the system. When the external application of ointments containing narcotics, of solutions of atropia, or of morphia on blistered surfaces has failed, still this mode of treatment has not said its last word. There still remains the *hypodermic method*, which, in a great many cases, proves more useful than the others. You know how this is employed: invented by Rynd, it was popularized in England by Wood chiefly, and by M. Béhier in France. With the small syringe which Pravaz invented for the purpose of injecting a coagulating fluid into aneurismal tumors, a very concentrated solution of basic sulphate of atropine or of sulphate of morphia is injected subcutaneously, as near as possible to the nerve-trunk which is the seat of pain. Atropine is generally preferred, and the solution is made with one grain of the sulphate to 100 minims of distilled water: when morphia is used, one grain is dissolved in twenty minims of distilled water. Each drop of the atropine solution is equivalent to $\frac{1}{100}$th of a grain of the salt; and each drop of the morphia solution to $\frac{1}{20}$th of a grain of morphia. Now, as each turn of the handle of the syringe propels one drop, the quantity which is injected may be measured with extreme facility. From four to five, and even ten and fifteen drops of the solution are thus injected: the doses should be small at first, so as to test the susceptibility of the patient, and should be increased by degrees.

This method is chiefly used in deepseated neuralgias, and although some of my colleagues have praised it almost to exaggeration perhaps, it is of great value, and deserves a place by the one which I have just described in detail to you. The small puncture of the skin frightens pusillanimous persons only, but it offers certain advantages of its own, and I have several times abstained from injecting after having punctured, and this simple acupuncture has sufficed to produce great improvement. I shall presently tell you what we may expect from acupuncture and electropuncture alone, in the treatment of neuralgias.

You are aware, gentlemen, with what difficulty atropine is borne. Some persons cannot take internally a granule containing $\frac{1}{50}$th of a grain of atropine without being poisoned, as it were, or without feeling, at the very least, very uncomfortable sensations in the throat and eyes. I confess that, knowing how much the gastric juice occasionally modifies certain vegetable substances, I expected to obtain, by injecting a narcotic solution into the cellular tissue, much more powerful effects than by having recourse to the ordinary mode of administration. I was, however, mistaken, and I found, to my great surprise, that ten, and sometimes fifteen, twenty drops of the solution of atropine, which were equivalent to $\frac{1}{10}$th and even $\frac{1}{5}$th of a grain of the salt (a dose which would certainly give rise to serious symptoms of poisoning if taken internally), were borne with the same facility as $\frac{1}{50}$th or $\frac{1}{25}$th of a grain when taken in the usual way. The same observation applies to morphia, and I must add that the treatment should be persevered in for some time, in order to obtain good results from it.

More than thirty years ago, I thought of a mode of treatment which has always given me more complete results than the endermic and the hypodermic methods, in cases of deepseated neuralgias, and of sciatica in particular. You probably recollect two men in St. Agnes Ward, at No. 8 and No. 14, who were both affected with a very painful and obstinate sciatica. You remember how useless subcutaneous injections of atropine proved to be,. although large doses were used and persevered in; a few hours' relief was merely obtained, and at the expense of a feeling of considerable discomfort. Turpentine, also, given in very large doses, produced but trifling improvement; and I then had recourse, with complete success, to a method which I formerly called *hypodermic*, a name which is more appropriate to the subcutaneous method.

In this method, the patient is made to lie on the abdomen, and a portion of the skin of his buttock is pinched up, perpendicularly to the axis of the trunk and over the point of exit of the sciatic nerve. A straight bistoury is then run through the pinched integuments at the base of the fold, and is next made to cut from within outwards. The incision gives scarcely any pain, and when made in that way, is clean and without tail. The subcutaneous cellular tissue is exposed at the bottom of the wound, and, after having filled this with some dry lint, kept in place by strips of sticking-plaster, the patient is left till the next day. Sometimes, however, although rarely, this simple operation suffices not only to give relief, but actually to cure. On the ensuing days small boluses, prepared according to the following formula, are placed in the wound.

℞. Extracti belladonnæ vel stramonii, ...	gr. xl āā
Extracti opii, ...	
Pulveris guaiaci, ...	gr. lxxx.
Mucilaginis gummi Adragantæ, ...	q. s.

Divide into twenty boluses, and dry in an oven.

Each bolus contains two grains each of extract of opium and of extract of stramonium or of belladonna. The guaiacum and the gum adraganth only serve to harden the mass, without preventing them from softening and yielding some of their principles.

Two, and even three boluses, should be placed in the wound, and kept there by a piece of sticking-plaster, under which, if possible, a small plate of very flexible lead, or several layers of tin, should be laid. A very dry pea should be placed at the same time in the wound, and, on this swelling out considerably during the day, the wound is maintained in a perfectly open condition. The absorption of the drugs goes on pretty actively, and the narcotic effects may be easily graduated by increasing or diminishing the number of boluses: if necessary, other boluses are prepared, containing a smaller quantity of extract.

One dressing is sufficient in the twenty-four hours, although a more rapid cure is obtained by dressing the wound morning and evening, when less boluses should be used each time. In order to obtain the full benefit of this mode of treatment, the system should be kept constantly under the influence of the drugs. So long as the pain continues, the treatment should be persevered in as above, but when the pain has ceased, a single bolus is laid inside the wound on each occasion with a dry pea; and when all pain has ceased completely for eight or ten days, peas alone are used, as in the case of issues.

I must declare that this mode of treatment has given me better results than any other in sciatic neuralgia, for it combines the action of narcotics and issues. We have seen already the effects of local applications of narcotic substances in the treatment of neuralgias, and I will presently tell you what may be obtained from superficial and deep revulsion. You may understand, therefore, that the combined influence of deep cauterization and of narcotics is followed by better results than the isolated use of either of these measures. This method offers another advantage again, namely, that after all pain has disappeared, there remains a superficial sore, a true issue, which, by being kept open for a few days, or a few weeks, renders the cure certain, and makes it easy, if pain should return in the least, to have again recourse to the boluses, without having to make a fresh incision.

Dr. Lafargue (of Saint Emilion) has recommended a mode of treatment which is of real utility in superficial neuralgias of a slight character, and which consists in introducing under the skin, by a process of vaccination, small quantities of morphia or of atropine, by means of a lancet dipped into a solution of either of those substances. A twofold action is combined in that process, namely, the effects of irritation of the skin, which is excited by the puncture and the somewhat painful papulæ which are thereby produced (although the benefit derived from this is small, as it is not obtainable by the application of one or more blisters); and secondly, the influence of narcotics, the inoculation probably acting in the same manner as the application of narcotic substances to the exposed cutis.

The application of a solution of *cyanide of potassium* on the skin protected by its epidermis, gives pretty good results when the affected nerves run superficially, as in the face and on the scalp. The strength of the solution should be of one part of the salt to eighty of water (one drachm to ten ounces). A compress, doubled up several times, is steeped in the solution and applied over the part; a piece of oil-silk is then laid over, and the whole kept in place by a handkerchief. Each application should last from half an hour to two hours, and should be renewed three or four times every twenty-four hours. This method offers great advantages, but it has disadvantages also, of which I must warn you. Pure cyanide of potassium is a caustic, and causes a certain amount of irritation, even when dissolved in eighty parts of water, producing bright redness of the skin, and then a vesicular or papular eruption, which sometimes gives rise to such discomfort that the treatment has to be given up. A greater disadvantage of this solution is that, like strong alkalies, it makes the hair break easily and turns them of a rusty color, which disappears only when the hair has grown again. I tell you this, gentlemen, in order that you should avoid, if possible, using a solution of cyanide of potassium in parts covered with hair.

Chloroform may be used in nearly the same circumstances as cyanide of potassium, but it should never be applied in a pure state, at least on the face and on parts which are habitually uncovered, because it gives rise to considerable irritation and even vesicates sometimes. It may then act on neuralgias in two ways: by revulsion, in the same manner as flying blisters, sinapisms, and the application of tincture of iodine; and by its sedative power. In order to obtain sedative effects, the liniment should contain from one-third to one-half of chloroform. This simple plan, which may be easily carried out, only succeeds, however, in neuralgias of no great intensity, and especially when they are superficial. I shall presently tell you of what use chloroform inhalations are in the most severe forms of the complaint.

As yet, I have only spoken of the *external application* of narcotics; but their *internal administration* is also of unquestionable utility. Solanaceous preparations, and opium, in its various forms, either separately or in combination, have always been, and will always be, powerful remedies against neuralgias. By a patient and intelligent administration of these drugs, results may be obtained which can scarcely be expected beforehand. The dose is of cardinal importance, and it is impossible to lay down precise rules what it should be, for it must vary, according to the duration and intensity of the disease, and according to the manner in which the remedy is borne by the individual. In general, however, no fear need be entertained as to keeping the patient fully under the influence of the drug, as shown by the manifestation of the physiological effects proper to each of them. Chloroform and ether, used as anæsthetics, render marked services also in the treatment of neuralgias. You know the good effects of *chloroform* inhalations in hepatic and nephritic colic, even when a calculus has got inside the excretory duct. You remember the case of that woman who suffered from biliary calculi, and whose pain was immediately relieved, even when the paroxysm was at its height; she suffered also from a very acute intercostal neuralgia, which was immediately relieved as soon as she got under the influence of chloroform. A youth, from Poissy, affected with gravel, was often seized with awful nephritic colic, whenever a large pyriform calculus tried to pass through the ureter. The pain, however acute it might be, was instantly relieved by chloroform inhalations, and the patient could be then placed with his head downwards and squeezed over the region of the kidney, so as to push the calculus back into the pelvis, when the attack ceased. In this case, the inhalation had to be pushed on to the stage of unconsciousness; but in order merely to relieve nephritic or hepatic colic, it is sufficient to induce a state of semi-unconsciousness, as when chloroform is used during labor.

Although the unconscionsness produced by chloroform or ether be transitory only, the narcotic effect induced continues for a pretty long period, and it frequently occurs that neuralgic pain yields or becomes very bearable at least, during half an hour or an hour even. It happens pretty frequently also that the anæsthetic stops the paroxysm completely, especially when the neuralgia assumes a paroxysmal character.

When chloroform or ether inhalations are used against neuralgic pain, no apparatus is needed, nor even a handkerchief. The patient makes a sort of cone with his hand, by bending his little finger completely into his palm, and his other fingers less completely: the cone is open between the thumb and index finger, and as the fingers are slightly separated from one another, air passes through easily. From ten to twenty minims of chloroform are dropped into the palm of the hand, which is then held before the nostrils, while the patient draws a deep breath. A single inspiration, when well made, is sufficient to cause giddiness, which is in some cases violent enough to make the patient fall, if he happens to be standing at the time. Many among you who have tried this simple plan have been often compelled to sit down, in consequence of the deep impression produced by the chloroform inhaled in this way. The inhalation may be repeated once or twice, successively, and as frequently afterwards as the return of the pain requires it. I need scarcely add, that the quantity of chloroform or of ether thus inhaled is too small ever to give rise to any accidents.

The internal administration of quinine is often of very great service in the treatment of neuralgias. When the affection is due to the influence of marsh miasmata, bark cures it, as it does all the other manifestations of the same specific cause; but, even when the neuralgia is not due to this cause, quinine exerts a powerful influence on the disease. It acts perhaps in the same manner as in rheumatism and in gout, and this is all the more probable because the neuralgia is very often the expression of a rheumatic or gouty diathesis. In such cases, quinine should be given in larger doses than in intermittent fever, and these larger doses should be continued for a longer period.

Iodide of potassium also cures certain neuralgias which have evidently nothing to do with syphilis.

Spirits of turpentine have been long vaunted in the treatment of neuralgias, and Récamier and Martinet have dwelt on the advantages which may be obtained from its internal administration. But the disagreeable taste of turpentine prevented its becoming a popular remedy. The patient had an insurmountable aversion to it after a few days, and gave it up. It had another and a somewhat serious disadvantage, namely, that it irritated the mucous membrane of the fauces and œsophagus on its passage, and to such a degree as occasionally to excite acute pain and vomiting. The irritating action of the drug was also felt in the stomach, and the manner of administering it added to the drawbacks. Some of these were avoided by administering it per rectum, and this method was particularly advised in sciatica; but the extent of absorbing surface was insufficient on the one hand, and on the other the rectum soon became intolerant of the drug, so that this useful plan had to be given up.

All these disadvantages are now removed, by the administration of turpentine in capsules, which thus reaches the stomach without being tasted, and without irritating the pharynx and œsophagus. On the other hand, the medicine should be taken at mealtime, a precaution to which I attach great importance, and by the aid of which turpentine and many other drugs are easily borne. It is of the highest practical importance that irritant remedies which are administered by the mouth do not get in contact with the unprotected membrane of the stomach. Preparations of iron, quinine, turpentine, iodine, and mercury, and many other therapeutic agents possessed of irritant properties, cannot, in most cases, be taken without inconvenience, for the sole reason that they are administered between meals; when taken with the food, they do not irritate the mucous membrane, while their specific power is not modified in the slightest degree.

You see me, therefore, gentlemen, prescribe turpentine in all my cases invariably, and you hear me give strict injunctions to the sister that the patient should take the capsules at his meals, and you witness yourselves the facility with which large doses of the drug are borne, while it very rarely happens that unpleasant symptoms compel me to suspend its administration.

Lehuby's capsules, which contain from eight to ten drops of turpentine, are those generally used in the Paris hospitals. I give from four to five or six of these capsules, twice a day (that is, from 100 to 120 drops a day), and this is generally enough, although this dose may be doubled, or trebled, without inconvenience. The medicine is continued for six or eight days in succession; I then intermit it for four or five days, after which I resume it, and so on for several weeks.

I must, in justice, however, declare that turpentine fails in half the cases of neuralgia, although it is a good remedy, of which practitioners should avail themselves. Quite recently you saw me on several occasions prescribe it in the case of a woman, lying in bed No. 29A, in St. Bernard Ward, who was affected with violent neuralgia of the trifacial and of the nerves of the stomach. The facial neuralgia disappeared first, and the gastric pain a few days later. Some of you must surely have thought it strange that I gave turpentine in large doses when the stomach seemed to be so affected. But a moment's reflection enables one to understand easily that neuralgia of the plexus which supplies the stomach, does not indicate inflammation of the mucous membrane, and that there is no reason why turpentine should not be given in such a case, as in hepatic or intercostal neuralgia. It is a fact, however (and I shall not attempt to explain it), that gastric neuralgia is in general more easily and more surely cured by turpentine than other neuralgias.

Irritant applications are of great service in the treatment of neuralgias, although much less so than is believed by most medical men. From Cotugno, who recommended, with such confidence, the application of *blisters* in sciatica, over three selected spots, the buttock, the head of the fibula, and the malleolus externus, down to Valleix, who regarded blisters almost like a panacea, practitioners have used them constantly in the treatment of neuralgias, although I think that they have owed their favor to the extreme facility with which they can be used, for I cannot believe in the exaggerâted praises which have been accorded to them for a century.

When the neuralgia is recent, and is apparently connected with rheumatism, it is often removed by irritating applications to the skin, such as painting it with *tincture of iodine*, or covering it with a *mustard poultice*, or rubbing it with *croton oil*, or with an *ammoniacal ointment*. But when the disease is chronic, and may be reasonably ascribed to a diathesis, as the gouty, the herpetic, the chlorotic, the aguish, or the syphilitic, the relief procured by the irritant applications is only temporary, and lasts for a few days, or even a few hours only. Yet by having recourse to powerful revulsion, proportionately to the old date and the violence of the neuralgia, results are sometimes obtained which less energetic treatment does not procure; and it is thus that *moxas* and *flying cauteries* are of unquestionable utility.

I often mentioned in my lectures, when I was professor of therapeutics in the Faculty of Medicine of Paris, that the public executioner at Lyons formerly enjoyed the reputation of curing sciatica. He wrapped the whole lower extremity in a large pitch plaster, and the eczematous eruption, which soon showed itself after this from the hip to the toes, acted with a power which a less heroic treatment could not exert.

You have seen me, within the last few years, try in the hospital an instrument invented by a man who does not belong to our profession, and which he has termed the *awakener* (*le réveilleur*). It consists of a stem, bearing at its extremity numerous small steel points which cannot penetrate deeper than a millimetre (about half a line). The stem has a bell spring attached to it, which, on being touched, drives the points into the skin with extreme violence and vivacity, thus producing multiple and very superficial acupuncture. The skin is next anointed with essential oil of mustard, dissolved in some olive oil, which soon gives rise to great local irritation, more painful than is generally caused by mustard alone.

This method has been chiefly used in the treatment of rheumatism, but it is really of some service in cases of neuralgia. It is analogous to *acupuncture*, which you have seen me use successfully in the wards on several occasions. For that purpose I use steel needles, which I soften, by heating them to redness in the flame of a candle. The head of each needle is protected by a small piece of sealing wax, and one or more of them are pushed into the painful regions, without taking care to avoid the nerve-trunks. They should be left in for ten minutes, and even for an hour, and the operation should be repeated two or three times a day, for several days, and for a few days after all pain has ceased.

Electropuncture is a more painful process, but it is of still greater utility. In 1863, I saw, in consultation with Dr. Demarquay, an old gentleman of 65 years old, who, after an attack of zona on one side of the forehead, had for more than a year been tormented by pain which almost drove him mad. Quinine, in large doses, local irritant applications, and narcotics, given internally and applied externally, had failed. The patient obtained admission into the *Maison municipale de Santé*, and came under Dr. Demarquay's care. This skilful surgeon had recourse to electropuncture, and in a few sittings the pain disappeared, but only for a time, however, for it showed itself again with its former intensity at the end of a month. Dr. Duchenne (de Boulogne) conceived the happy idea of trying very powerful *faradization of the skin* in obstinate neuralgia; and this plan, which is extremely painful, sometimes produces marvellous effects. It is not uncommon to see the horrible neuralgic pain of *angina pectoris* yield to its influence. Epileptiform neuralgia, which is such an exquisitely painful and a cruelly incurable affection, is sometimes quickly modified, but not *cured*, by this method. But, although the violence and persistence of neuralgic pains induced patients to submit to cutaneous faradization, few of those who have been but recently affected can be persuaded to adopt a method of treatment which causes such insufferable pain.

I have often shown you the extraordinary effects following the *application of heat* to joints that are the seat of chronic painful engorgement, by means of sandbags heated to as high a temperature as the patient can bear, without being burnt. This plan succeeds very well also in superficial neuralgias, as those of the scalp, the face, and the neck; and even when the pain affects limbs in their continuity. You have seen me, on several occasions, wrap in these sandbags the heads of patients, for twenty minutes at a time, and repeating the process twice a day.

This plan is certainly not so efficacious as those which I have already described to you, although it proved very useful in some cases in which every other treatment had failed completely.

There is another method, to which I have had recourse several times in my life, but in which I have found few imitators, however much I have said on the subject. I allude to *division of the temporal and of the occipital arteries*, with the view of curing obstinate neuralgias of the head. Division of the painful nerve had already been recommended in epileptiform neuralgia, and, in the majority of instances, it was impossible, except by actual dissection, to divide the nerve without also cutting the artery which usually accompanies it. I have given you already, in my lecture on epileptiform neuralgia, my opinion of this operation in such cases; but after having on several occasions divided the artery, in obstinate cases of the ordinary form of neuralgia, I obtained such immediate results that I in vain ask myself to this day how the method acted. I tried it for the first time in 1833, in the case of a lady, about 30 years old, who had been for more than 10 years afflicted with temporo-facial and cranial neuralgia, of excessive violence. I had tried no end of remedies; and, as a last resource, I determined on dividing the temporal artery above the zygomatic arch. I wrapped the blade of a bistoury in a piece of sticking-plaster, leaving only a third of an inch of the point uncovered, and, holding the instrument like a pen, I pushed it through the tissues, quite close to the ear, perpendicularly, and on reaching the bone, I went on cutting until there was a gush of arterial blood, always keeping the knife parallel to the upper edge of the zygomatic arch. The neuralgia ceased as soon as the section was made. As my object was not to draw blood, I immediate applied a compressing bandage, which was removed twenty-four hours afterwards. The neuralgia was cured for a rather prolonged period, and, although it returned subsequently, I still regard the case as a very successful one. Division of the occipital artery is not less efficacious in the treatment of neuralgia seated in the posterior region of the head; and it is often found necessary to divide both arteries, an operation as easily performed as it is free from inconvenience. I said just now that I could not account for the suddenness of the improvement following division of the arteries. I am well aware that these vessels, especially the occipital, are accompanied by nervous twigs of some importance; but although I understand how the pain ceases in parts supplied by nerves included in the section, I cannot understand the complete cessation of pain in the greater number of nerve twigs, which a moment before caused such acute pain, and which have no apparent connection with the cut branches.

The following case is a most curious instance in point:

I was asked by Dr. Mathieu, to see a man about 30 years old, in the Rue Neuve Saint Méry, who was suffering from acute encephalitis. The poor man complained of excessive pain in the head, and uttered fearful cries; narcotics, employed internally and externally, had failed to relieve, and bleeding had proved as ineffectual. I advised division of the temporal artery, and operated at once; the relief was instantaneous, although scarcely a spoonful of blood had been lost. The patient was admitted into my wards in the hospital, and after his death, an abscess was found in his brain. I have related this case, gentlemen, merely to show you the utility of dividing the artery, even in cranial neuralgias that are symptomatic of the gravest lesions.

It may be asked whether the rapid improvement which follows division of the artery and of the nervous twigs which accompany it is the result of a mental impression, in some respects analogous to the impression produced in toothache by the sight of a dentist's instrument; but I shall not attempt to answer the question. Yet, when we see facial neuralgia and sciatic neuralgia itself cured by division or cauterization of the lobule of the ear (and there are now pretty numerous cases of the kind on record), how can we account for the beneficial influence due to division of the temporal and occipital arteries in neuralgias of the head?

As yet, gentlemen, in the long list of remedies which I have enumerated to you, I have only had the element *pain* in view, and I have not taken into account the cause to which the neuralgia was due, except when this cause was immediately cognizable, as in toothache dependent on a carious tooth, in neuroma, in wounds, or in injury to a nerve-trunk, &c. But when the cause acts on the whole system, as syphilis and chlorosis, for example, we can and we doubtless should calm the pain as quickly as possible, but the chief aim of treatment should be to remove the cause. Otherwise the neuralgia is only relieved for a time, and returns after a brief interval; for when it disappears easily under the influence of the various measures which I have described to you, it is brought on by causes which manifest themselves by transient phenomena only, as rheumatism, for example.

Neuralgia of syphilitic origin is of rare occurrence, except when there is a local lesion, such as exostosis, periostitis, nodes, &c., and the general rheumatoid pain which is sometimes present in cases of constitutional syphilis, is probably due to irritation of the spinal cord. It is often brought on again by an acute inflammation, and by ulceration of mucous membranes, as in syphilitic coryza and otitis. All these neuralgic phenomena are rapidly removed by specific treatment, but if necrosis be present, mercury and iodine will, of course, fail, and will only influence the disease which brought on the necrosis. I have told you the signs by which you could recognize neuralgias of syphilitic origin, and you have seen, in the wards, how quickly a specific treatment did away with the pain. You remember the case of a woman, who, in June, 1863, lay at No. 7, in St. Bernard Ward. The neuralgia was worse rather late in the evening, and was better in the morning. I gave her two grains of calomel a day, to be taken in doses of the tenth of a grain in the course of the day, and on the third day of this treatment, as soon as the gums began to swell slightly the pain ceased almost entirely. The solution of Van Swieten (*Liquor*, *corrosivi sublimati*) was then substituted for the calomel, and subsequently, iodide of potassium was administered.

Calomel, in divided doses (*fractâ dosi*) is the preparation which I always use when I want to act quickly on the system. I have powders made up with $\frac{1}{10}$th of a grain of calomel and from two to four grains of sugar; and the patient takes ten such powders every day at nearly equal intervals, for three, four, five, or six days. It rarely happens that the gums do not begin to swell by the third day; when they do, five powders only are given, instead of ten. When the pain is relieved, I prescribe chlorate of potash to cure the mercurial stomatitis, and I next give the solution of Van Swieten for a month or two, finishing afterwards with iodide of potassium. The effects of the treatment manifest themselves immediately; from the first night, the pain is often diminished, and it is of rare occurrence that it is not perfectly bearable after three days of this treatment. Any exostosis which may be present does not, of course, disappear in that time, but it immediately becomes less tender on pressure, and afterwards disappears slowly.

I am aware, gentlemen, that iodide of potassium does real service in such cases, and you have seen me administer it with great success on several occasions. But I must state, that although it is superior to mercury when administered in the ordinary way, it is infinitely less powerful than calomel given according to the above method. As to intermittent neuralgias, which have been specially termed larvated fevers when apparently due to paludal influences, they are cured by preparations of bark in pretty large doses, larger in general than in ordinary intermittent fevers. But you must not think that intermittence and perfect periodicity in cases of neuralgia are proof positive that the disease is of paludal origin, for I have told you of cases in which a grave organic lesion gave rise to perfectly periodic neuralgic pain, and in which bark failed.

When the neuralgia returns in multiple paroxysms every day, even when these paroxysms are periodic, quinine exerts very little influence. It possesses more power if there be but one paroxysm, while it is a sovereign remedy, as it were, when the paroxysms are tertian or quartan, for these forms, when periodic, are more certain proofs of the existence of paludal influence in the case. Yet, gentlemen (and I cannot account for it), even when there is not the least suspicion that the same causes which bring on intermittent fever have been at work, quinine, in large doses, exerts a powerful influence on neuralgia, even when the attacks are not intermittent, and, *à fortiori*, when they are intermittent and periodic. Hence you see me, in most cases, have recourse to quinine first, and try other remedies only when it has failed.

LECTURE LIV.

ON HYDROPHOBIA.

Nervous Phenomena characteristic of Hydrophobia.—Sensory Hyperæsthesia.—Priapism, a Frequent Symptom.—The Manifestation of Lyssi, during the Stage of Incubation, is not at Variance with the Pathology of Virulent Diseases.—They might be the Primary Localization of the Virus.—Can Hydrophobia be prevented by Cauterizing them?—Analogies and Differences between Human and Canine Rabies.—Rabies never Spontaneous in Man.—Treatment as Varied as Powerless.

GENTLEMEN: At one of our last meetings, I called your attention to the case of a man who exhibited all the symptoms of hydrophobia, and who died on the same day that he was admitted into the hospital. From the information we received from his friends, and which was confirmed by Dr. Bienfait (of Rheims), and by Messrs. Leblanc, veterinary surgeons in Paris, no doubt could exist as to the nature of his complaint.

I know of nothing, gentlemen, more painful to hear, or more fatiguing to read, than a lengthy medical case overloaded with details; and yet, details should not be neglected, when they relate to a complaint which you will rarely have an opportunity of observing. A complete case, better than a long dogmatic treatise, leaves on the mind an impression which time cannot blot out, especially when you have been an eye-witness of the chief phenomena of the disease. You will, therefore, forgive my entering into details which may at first sight appear superfluous, but will by and by be perfectly explained.

The case is as follows: During the night of January 23, 1861, my clinical assistant, Dr. Dumontpallier, was summoned in all haste to see a patient who was suffering from what was termed "an *indigestion of water.*" On his way there, Dr. Dumontpallier was told that the patient complained of intense thirst and was firmly bent on drinking, but could not carry water to his lips without being seized with a deep feeling of terror. He could not take any solid food either. This difficulty of swallowing, supervening suddenly after slight *malaise*, in a man aged 37, was a strange phenomenon. By closely questioning the patient's friends, he then ascertained that about the end of September, that is, four months previously, M. B. had been bitten in the hand by a small pet dog, which had on the same occasion bitten a little girl, 8 years old, and a man-servant about 30 years of age, and a young cat, with which he usually played. It was added that the dog had died of inflammation of the bowels in Mr. Leblanc's infirmary, in the beginning of October, 1860. Mr. B.'s friends were honest in giving this information, because Mr. Leblanc had thought proper to conceal the name of the dog's real complaint. As the child, the servant, and the cat that had been bitten were perfectly well at the end of January, 1861, nothing had occurred to create any alarm in Mr. B.'s family. The rapid death of the dog, however, a few days after he had bitten his master, and the complete dysphagia which had supervened in the latter, inclined Dr. Dumontpallier to think that the patient was most probably hydrophobic. When he saw him, he was walking about his room, in the greatest agitation, unable to remain quiet a single moment. He looked fixedly before him; his pupils were dilated, his face extremely pale, and his hair and beard disordered. The expression of his physiognomy was that of great anxiety; he spoke in a curt and jerked manner, and complained of great dryness of the throat and of his being obliged to keep spitting incessantly. Whenever he spat out the saliva, his whole body shivered. The room was lighted up with candles and a lamp; over the mantelpiece was a looking-glass, and on the shelf a water jug. As the sight of these objects gave the patient no pain, there was, therefore, no hyperæsthesia of the eye, but the skin was painfully sensitive. The patient dreaded to touch his face and to rest his hands on his clothes; he refused to allow his pulse to be felt, and in order to shorten the examination, he wished by taking a glassful of water to show that he could not drink, although he had a firm intention of doing so, for he took up the glass and raised it to his lips, but he immediately rejected the water which, by a rapid movement, he had got into his mouth. This voluntary experiment brought on no convulsions; the patient was merely more agitated for a few minutes, and then having calmed down, he tried to relate what he had felt since January 20.

While speaking, he made prodigious efforts for remaining calm. He had been sad for a long time, he said, in consequence of money losses, and had gone to Rheims on January 13, to stay with some friends, in hopes that he might be cheered up. From January 13 to January 20, he had felt no *malaise;* at that last date—a Sunday—he went out early in the morning in an open carriage, the temperature being cold, on an excursion into the country with his friends. He ate in the morning with his usual appetite, but in the afternoon he felt so intensely thirsty that the carriage had to be stopped several times to allow him to drink at some houses on the road. He had then no difficulty in swallowing, but what he drank felt very cold to him. He was shortly afterwards seized with violent shiverings in the carriage, and went to bed immediately on getting back to Rheims. He did not sleep at all during the night; he got up constantly because he felt giddy when lying down, and walked about in his room, feeling very agitated. He did not feel hungry, and he could still drink, although he had strange unpleasant sensations. He was very agitated during the whole of Monday, both during the day and at night. Mr. B. himself gave all these details, for his mind was perfectly clear. Dr. Bienfait (of Rheims) kindly wrote me two letters containing a description of the symptoms which he had observed from January 21, when he was first called in. I will read to you what he said:

"The patient was in a state of considerable agitation; his face was pale and his eyes extraordinarily mobile. His own idea was that he was suffering from an overloaded stomach and that he was going to be sick. His breathing and the action of his heart was somewhat hurried. The tongue had a slight yellowish coating of fur, and was of a somewhat brighter tint along the edges and the raphe. The patient drank in my presence, but with a certain degree of convulsive haste, as everything else he did."

Dr. Bienfait thought that the case was one of indigestion attended with a nervous condition depending on the patient's idiosyncrasy; he prescribed a mixture containing opium. He was, however, uneasy on account of that nervous condition, and therefore went back to see him in the evening. He found the patient in a still more agitated state; he had taken his mixture, but each time after heroic efforts. "I asked him to take a spoonful of it in my presence," Dr. Bienfait goes on to state; "but it was immediately thrown out by a sudden spasm which seemed to spread from the muscles of the pharynx to the orbicularis oris. Yet the unfortunate patient had collected all his strength before attempting to swallow; he had taken three steps backwards, and by an instinctive movement, had thrown his arms about him as if he wished to get all obstacles out of the way." A bath was ordered, to the patient's great joy, but was not taken. Dr. Bienfait no longer entertained any doubt as to the nature of the case, and although he knew nothing of the previous history, he believed the patient to be suffering from hydrophobia.

On the following morning, the symptoms had become aggravated, and there was, moreover, general hyperæsthesia. The patient's friends, better informed, it would appear, than his own relatives, then told Dr. Bienfait that, about the month of September, Mr. B. had been obliged to sacrifice a small king-Charles, who was suffering, according to a veterinary surgeon's account, from rabies. Mr. B. was probably aware of the circumstance, but never made any allusion himself to his dog having been mad. Nothing had been done to try and avoid ill consequences from the bite.

In his second letter, Dr. Bienfait stated that previous to allowing Mr. B. to return to Paris "he had, on ausculting his chest, found that the vesicular breathing was perfectly pure, but was interrupted at each inspiration by one or more suppressed sobs, as it were. The heart's impulse was notably irregular, and the pulse at the wrist was equally so; the irregularity of the latter being attended with a sort of vascular spasm, which could not be defined, but was very remarkable.

"During the whole of his stay at Rheims, the patient had no delirium, or anything like it, and never had the least wish to bite. He only spoke of some instinctive dread, and had a marked tendency to be communicative. He had no idea of the real nature of his complaint, and had no recollection of having been bitten. Imagination, therefore, seemed to have nothing to do with the manifestation of the symptoms recorded."

Mr. B. insisted on returning to Paris. During the journey from Rheims to Paris, he was very agitated and extremely thirsty, his thirst being temporarily relieved only by keeping small lumps of ice in his mouth; but in all probability the patient, who was constantly spitting, could not swallow the melted ice, and therefore complained of a sensation of constriction and great heat in the throat. He had, during the journey, frequent erections and seminal emissions; the hyperæsthesia of his genital organs gave him great pain.

Such was his condition when Mr. B. reached Paris in the evening. I have already told you in what state he was seen by my clinical assistant. The latter advised his immediate removal to the Hôtel-Dieu, and on the following morning, January 24, that is, 3½ days after the invasion of the disease, I had the opportunity of ascertaining with my regretted colleague, Legroux, the following facts:

The patient was so agitated that many thought him to be suffering from acute mania. He had a strange aspect and was unable to swallow liquids. Several among you doubtless remember the painful scene when he attempted to drink; he clutched the glass with force, saying, "I wish to drink, and I will," and then carried it with determination to his lips. But as soon as the water had passed his lips, his face assumed a look of excessive pain, and his whole body was within a short time shaken with violent convulsive trembling. He then exclaimed, "I cannot drink, do make me drink."

When calm had returned after this paroxysm, I was enabled to ascertain that there was redness of the soft palate and pharynx, and great dryness of the tongue. The patient's beard was also soiled with a frothy saliva which he kept constantly spitting out.

The lateral and under surfaces of the tongue were carefully examined; the patient was docile, and had no desire to bite; but we could not discover any of those swellings which have been called *lyssi*. We could not, however, draw any conclusion from their absence, because we shall see, by and by, that they have been seen only during the period of incubation by the very persons who described them, and that they disappear before the symptoms of confirmed rabies show themselves.

I merely recommended that the patient should be watched, and nothing else, as experience had taught me that we are completely powerless against this cruel malady.

In the course of the day, the patient's wife and some of his friends came to visit him; he was always very agitated, and the presence of his friends gave him pain. He begged that everything should be tried to cure him; he would be saved, he said, if he could be made to drink.

About half-past four in the afternoon, my clinical assistant, with the aid of several pupils, succeeded in introducing an œsophagus tube through the nares into the stomach, and about seven ounces of broth were gently poured into it. Half of this quantity had already reached the stomach, when the fluid suddenly ceased to run, from the flexible tube being compressed by a violent spasm of the pharynx and œsophagus. The spasm soon spread to the respiratory muscles, the face turned livid, and the opened eyes stared; the tube was quickly removed, and as the patient, who had been sitting on a chair, slipped down on the floor like an inert mass, it was thought that he had died; but nevertheless water was sprinkled on his face, his tongue was drawn out of the mouth, which was kept open by firmly drawing down the lower jaw, while the thoracic walls were alternately squeezed and left to expand. A whistling inspiration then followed; compression of the chest was kept up, and respiration was soon re-established, after which the patient ejected to some distance a certain quantity of saliva or of bronchial froth. During the paroxysm there had been an erection of the penis, with ejaculation. The patient evinced no fear on finding himself lying on the ground; he knew that he had just run a great danger, but he believed that he was saved. Advantage was taken of his being calm to advise him to get into bed, and he was persuaded to allow himself to be tied in it lest he should fall out. He let us do what we liked with him, and warmly expressed his gratitude; he squeezed our hands, and wished to kiss those who had saved his life, he said. In the course of the evening he had several convulsive paroxysms, and died suddenly at half-past ten, after having been violently agitated for a few seconds.

A post-mortem examination was made on the very next morning. The body was exceedingly rigid, the face livid, and the whole posterior aspect of the trunk and limbs presented numerous sugillations. The brain and parenchymatous organs were congested. The mucous membrane of the mouth and pharynx were very markedly injected. The salivary glands were taken out and sent to Alfort, with some saliva that had been collected during the patient's life. Mr. Reynal, who had undertaken to inoculate dogs with this saliva, afterwards informed me that the experiment had given no result.

Note, gentlemen, that the child and the man-servant, who had been bitten, did not exhibit any symptom of rabies when Mr. B. died. The cat, also, which had been bitten at the same time, was still in the house, and there had occurred no change in its habits which authorized a suspicion that it had been inoculated with the virus; yet it was thought prudent to sacrifice it on that same evening.

If we now sum up the principal facts in this case, we find that a man is bitten by a dog in September, 1860. A little girl and another man are bitten on the same occasion, as well as a cat, by the same dog, and at the end of January, 1861, the master of the house, the person first bitten, alone manifests symptoms of rabies. Before that date Mr. B. was sad, but this was ascribed to money losses. He leaves Paris in search of recreation, when suddenly, a few hours after a breakfast eaten with relish, he complains of very intense thirst. He soon is seized with general shivering, and from that moment he loses his appetite, and the capacity of swallowing liquids, while he becomes extremely agitated. He goes on in that way for about thirty-six hours. General hyperæsthesia is then noted, and from that time he cannot wash his hands or face, the least attempt of the kind immediately bringing on great agitation and violent shivering, and the same thing occurs when he tries to comb his hair or beard; he dreads touching his person with his own hands. It is probable that the hyperæsthesia which existed then determined, through a reflex action on the least contact of the skin, convulsions which assumed the form of general shiverings. There were rare intervals of quiet. To the cutaneous hyperæsthesia there is then superadded a very acute and frequently repeated excitation of the genital organs, and frequent erection, attended with seminal emission, increases the patient's agitation. Three days after the invasion of the disease, his aspect creates alarm; he is agitated, extremely garrulous, speaking in a curt, jerking manner; he cannot drink, although he is intensely thirsty, and when he attempts to doze, he is immediately seized with clonic convulsions and spasms. The hyperæsthesia and satyriasis persist throughout the 24th of January, the convulsive paroxysms become more frequent, and death occurs on the fourth day of the complaint, without the patient having manifested any mental disorder, any hallucination of sight or hearing.

We could not succeed in finding the least trace of a bite on Mr. B.'s hands, although his symptoms did not admit of a doubt as to the nature of his malady, and the dog that had bitten him had likewise died of hydrophobia. Two periods were observed in Mr. B.'s case; one of sadness and melancholy, during which he left his house and went away from Paris in search of amusement; the other, which may be termed period of *agitation*, and which set in with very intense thirst and shivering, soon followed by a dread of water and by convulsions, which continued until his last moments. It is important to note that, as well as general hyperæsthesia, there was satyriasis present, a fact rarely mentioned in records of cases of rabies, as in the numerous instances contained in Ch. Andry's* work, and in the article by Trolliet and Villermé in the Dictionary, in sixty vols. Boerhaave, however, mentions priapism as one of the symptoms of rabies in man,† and Van Swieten‡ states in his Commentaries that Galen has mentioned the fact, which has also been noted by other observers. He relates the case of a porter who became hydrophobic, and who, during the last three days of the complaint, had involuntary and constant seminal emissions. This man, Van Swieten adds, lost at the same time his seed and his life: *Semen et animam simul efflavit.*

Dr. Peter has also noted frequent seminal emissions in the case of a soldier, who was admitted, in 1862, into the military hospital of Gros-Caillou, under Dr. Worms. The priapism was almost constant, and the emissions were attended with voluptuous sensations, as showed by the prurient expressions used by the patient. Shortly afterwards, in his delirium, he bitterly reproached his medical attendants for having recourse to witchcraft in order thus "to take away from him the very principle of life." He had been bitten forty days previously by a very small dog, which had entered the barrack-room and did not look ill. The disease had set in with a fearful spasm, which occurred as he was going to wash his face. When he was brought to the hospital he was perfectly rational, but carefully avoided all allusion to the bite which he had received, as well as to the nature of his complaint, which he apparently suspected. It is a very remarkable circumstance, which I am anxious to bring forward prominently, that rabid individuals persistently conceal the probable cause of their complaint. It would seem as if they recoiled from the fearful truth, and dreaded to acknowledge it to themselves or to others. In the present instance, a canteen-woman informed Dr. Peter of the bite. Death occurred within thirty-six hours. Another peculiarity of the case consisted in the most exquisite exaltation of some of the senses; the scent of some lilacs in flower distressed him, although they were at a distance of about thirty metres from him: the least movement of the air, as by the opening of a door, made him complain of a disagreeable sensation as if his face were slapped, and caused him to jump in bed.

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* Recherches sur la rage. Paris, 1781.

† VAN SWIETEN: Commentaria in Boerhaave aphorismos, § 1138, Rabies canina. Paris, 1758, t. iii, p. 550, 1771.

‡ Ibid., p. 556.

Nymphomania has been sometimes noted in women, and in the case of a young woman who died of hydrophobia in the year 1861, this symptom was mentioned by Dr. Bricheteau, who published the case. The fact has been only noted during the period of excitation of rabies; yet Mr. B., even while he was given to sadness and melancholy, had shown sexual aptitudes which had created much surprise, as for a long time he had been very frigid.

You will find many cases of hydrophobia recorded in books which treat of this complaint, but I will nevertheless relate to you some of the cases which came under my own observation.

In 1823, when I was a pupil of Bretonneau's, the son of a joiner, seven years old, was brought to the Tours Hospital. Bretonneau at once recognized hydrophobia on seeing the boy. The poor little fellow could not lie quietly in his bed, and was singularly agitated; all bright objects frightened him, and the unfolding of a napkin before him brought on a paroxysm; in fact, everything alarmed him. He died three or four hours after his admission into the hospital. He had been bitten by a mad dog three months previously.

This was the first time that I had ever seen a case of hydrophobia, and the impression, therefore, which it made on me, was a lasting one.

A few years later, while I was a resident assistant in the Charenton Hospital, Mr. Calmeil and I were going round with Esquirol, when I was summoned to see a patient who had just come to the infirmary, and who, it was said, had a strange aspect. He was about twenty years old: everything alarmed him, and his face expressed terror. I remembered the little boy I had seen in the Tours Hospital, and going back to Esquirol, I informed him that a man suffering from hydrophobia had just been admitted into one of his wards. The man could not swallow, and the sight of bright objects created a strange terror in him. His intellect was perfect, and he told us himself that he had been bitten in the leg by a dog five months previously, but had not felt any uneasiness on that score. Esquirol ordered him to be put into a cell: the poor fellow apologized for the trouble he gave, saying that he had been unwell for the last two days only, and that he had not slept during the previous night. By Esquirol's orders, he was tied to his bed without his offering any resistance, but when he was asked to drink, he was seized with fearful spasms and convulsions as soon as the glass was raised to his lips. His pulse was full, and he was greatly agitated. This was a time when Broussais' doctrines had many followers; so that the patient was ordered to be bled. I held his arm, and as he kept spitting all the time, my face was covered with his saliva. On a napkin being thrown over his head, he took fright, and became seized with convulsions; the blood ceased to flow, and he died. Had the bleeding anything to do with the rapidity with which death occurred, or did the patient die in consequence of a spasm of the respiratory muscles, such as is most commonly observed in individuals who have reached the second stage of the hydrophobic disease?

In 1831, I saw with Bonnet (of Lyons) in one of Mr. Récamier's wards at the Hôtel-Dieu, a man still young, from whose aspect, expression of the face, and extreme agitation we diagnosed hydrophobia. He had been bitten, seven or eight months previously, by a cat, which had disappeared from the house and had not since returned. Messrs. Magendie, Caillard, Petit, and Récamier entertained no doubt as to the nature of the case. The man could only drink with very great difficulty; he was constantly spitting, and was very much agitated. By Magendie's advice, a mixture containing 36 drops of the officinal preparation of prussic acid was ordered, but no sooner had he taken this than he looked as if he had been struck by lightning, his pupils were dilated and immovable, and I thought he was dead. I left Bonnet with him, and ran to the dispensary to ascertain whether some mistake had not been made in preparing the mixture. On then hearing that the strong instead of the dilute acid had been used, I left the hospital to go to the Bureau Central, fully persuaded that the patient had been poisoned. Soon afterwards, Bonnet came and told me that the man still lived and consented to drink; that his pupils were still dilated, but that he was no longer agitated. Now it was a question whether the prussic acid had done him good? I saw him an hour afterwards; he was again very agitated and was unable to drink. I then prescribed six drops of dilute prussic acid; there was no error this time, and the dose of the drug was six times less, yet the patient had no sooner attempted to swallow the mixture than he was as rapidly thrown into the same condition as before. By degrees, however, breathing was restored. After this, we felt little inclined to ascribe to the prussic acid the pnenomena we had witnessed, and we regarded them as the consequences of the efforts of swallowing, which, in such cases, bring on spasms of the respiratory muscles and rapid asphyxia. A third attempt was made to get the patient to swallow two drops of prussic acid in a mixture, but he became agitated again, and the convulsive paroxysms growing more and more frequent, death supervened forty-eight hours after the invasion of the complaint.

These cases are not given with full details, it is true; but it is a well-ascertained fact that all these patients had been bitten, and that after a period of incubation of varying duration, and without any appreciable determining cause, mental or physical, they had felt general discomfort and been very much agitated. Inability to swallow fluids had soon come on; the sight of fluids or of bright objects had been enough to bring on convulsions, at first clonic and then tonic, and lastly, the patients died of asphyxia through spasm of the respiratory muscles. It cannot be affirmed that death always takes place by asphyxia, because the practitioner is not always there to witness the mode in which the fatal termination occurs; but there is such marked asphyxia during the paroxysms, and dissection so often discloses after death the lesions proper to that condition, that one is authorized to believe that most commonly the patient dies of asphyxia during a paroxysm. I shall, by and by, analyze what takes place during a paroxysm in hydrophobia, and I shall inquire at what time the patient is in danger of dying.

The following case, which was communicated to me by Dr. Eugène Fournier, will show you the part played by asphyxia as a cause of death in hydrophobia. On June 18, 1860, a man, aged 27, a joiner by trade, was brought in the evening to Beaujon Hospital, by order of the police commissary of Batignolles, because he was thought to be suffering from rabies. He had been bitten, two months previously, by a small dog, which he was teasing. The dog had disappeared, and never returned. The bite had inflicted a small wound in the ring finger of the right hand, which healed up very rapidly; it was not cauterized, and no trace of it could be found. The man went on with his occupation for two months, without experiencing any peculiar sensation. But on June 15, he felt some discomfort and had nausea. On the following day, whilst working in a room, he nearly fell down from a chair on which he had got up, and on trying to save himself by catching hold of a piece of furniture with his right hand, he felt an acute pain in his right arm. As this pain persisted, he told people that hydrophobia was seizing him in the arm where he had been bitten by a dog suspected to be mad.

He felt ill on June 17, and remained indoors. The next morning, early, somebody went and told his sister that he was delirious. Still he recognized his sister, and stoutly refused to be taken to the hospital, so that a police warrant had to be obtained before he could be removed, and it was only with very great difficulty that he was brought to Beaujon Hospital. He got pretty calm when he was once in bed, and quietly said that he had felt an inclination to be sick and had had an indigestion, but could not conceive why violence was had recourse to to bring him to the hospital, where, he added, he was very glad to be. When he was questioned as to the origin of his complaint, he turned his eyes away, and refused to admit that he had been bitten by a mad dog. His skin was hot, especially about the face, his pulse frequent, but not tense; the pupils were neither dilated nor contracted, the eyes were sunken in and surrounded by a bluish circle. He had a headache, and no appetite, and did not complain of thirst. He was offered drink, but refused. He was left by himself in a room, confined to the bed by a strait-jacket. Shortly afterwards, however, fearful cries were heard, and on hurrying to him, he was found in convulsions: his face was livid, and asphyxia seemed to be imminent. He spat at times; but there was no foam about his mouth. Other paroxysms probably came on, and death occurred at half-past eleven, about three hours after his admission into the hospital. No treatment had been attempted. There was great cadaveric rigidity three hours after death, and there was noted subcutaneous emphysema of the anterior aspect of the throat.

The body was examined about thirty-six hours after death. There was congestion of the meninges and of the cerebral substance. The lungs were crepitant, bluish on the surface, black on section, and gorged with blood. There was a perforation in the upper lobe of the right lung, near which there was subpleural emphysema, while air bubbled up through it when the lung was compressed. The congestion of the lungs, and the presence of emphysema, at first subpleural and afterwards cervical as well, from rupture of the lung-tissue, prove that during the last moments of life there must have been extreme obstruction to breathing from a spasm of the glottis, which by shutting up the natural aperture of exit of the air compressed inside the lungs during the convulsive paroxysm, led to rupture of the lung-substance, and consequently to emphysema.

Most authors entertain no doubt as to asphyxia being the cause of death in hydrophobia, and in a case related with very great care and commented on with rare talent by my colleague in the hospitals, Dr. Bergeron, the details given show that the patient died of asphyxia. In this instance, however, the asphyxia seemed to have been *gradual* not *sudden;* for it is stated in the report of the case* that lividity of the face began to show itself about three hours before death, and became more intense after a time, and that speech was impeded by an accumulation of bronchial mucus in the fauces; and, lastly, that during the half hour which preceded death, the face was purplish and bedewed with perspiration. On examination of the body there were found, in this case also, marked signs of asphyxia; the whole venous system was gorged with blood, the meninges and the brain-substance were markedly hyperæmic, and the lungs crepitated at the apex and in front, but were hard and less crepitant posteriorly, while their color was of a deep brownish-red tint. When incised, a good quantity of black blood exuded from them. In the posterior margin of the right lung were a few small apoplectic centres, and especially blood-imbibition.

Now, gentlemen, can it be said that individuals affected with hydrophobia must all necessarily die of asphyxia? I would not dare to state this positively, because of the rapidity, the suddenness with which death occurs in some cases. Yet, I believe that rapid asphyxia from closure of the glottis, or slow asphyxia from repeated spasm of the respiratory muscles, is the most frequent mode of termination of this complaint. The clinical reports and the anatomical details to which I have called your attention seem to me to leave no doubt on this point.

Before I describe the chief symptoms of hydrophobia proper, I wish to relate to you a few cases of mental hydrophobia, a special form of the complaint, which is brought on by emotion on seeing rabid individuals, or on hearing a description of real cases of hydrophobia.

In the spring of the year 1828, I was engaged with my colleague at the Academy, M. Leblanc, and with Dr. Ramon, in investigating the rot-disease which was rife among the sheep of the Sologne. He had just inoculated with the disease three hundred sheep belonging to M. Joupitre, mayor of the department. Whilst talking of virulent diseases in general, this gentleman told us that he had been affected with hydrophobia. A farm-dog had tried to bite his arm, and about the same time had bitten a good many beasts which died of rabies. A few months afterwards, on Easter Sunday, after service, and at a breakfast at which every one had done his best to make up for the rigid abstinence of the past Lent, M. Joupitre exclaimed suddenly that he was seized with hydrophobia. He could not eat or drink any more, and our host was already beginning to rave, when his wife, who only believed that he had eaten too much, persuaded him to tickle his throat with his fingers. The advice was good, for copious sickness was brought on by the manœuvre, and nothing more was said about rabies.

That same year I happened to relate M. Joupitre's case to a presiding judge in chambers, who, in his turn, told me that he also had once believed himself to be seized with hydrophobia. He used to go out riding frequently, and a sporting dog which generally accompanied him, often jumped to kiss the hand with which he held his whip. During one of these rides they once met a flock of sheep, after which the dog ran, biting those he could catch. The animal still heard and obeyed his call, but he had a strange aspect.

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* Archives de Médecine, 1862.

Again he ran after and bit dogs, cows and oxen, and lastly swam across a river; a few hours later, he died. A short time after this, the judge heard that many of the beasts that had been bitten by his dog, had died of rabies. This news alarmed him because he recalled to mind that on the same day the dog had licked his right hand several times. On examining his hand, he found several small scars on it, and seized with terror upon this, he no longer dared touch water to shave himself, and fully believed he had hydrophobia. A medical man, who was sent for from Orleans, tried in vain to calm his fears: for several days he was excited and delirious. At last, being told over and over again that persons seized with rabies died very rapidly, and that he could not therefore be rabid since his dread of water dated already ten days back, and after reading in books about the duration of confirmed hydrophobia, he allowed himself to be persuaded, and his dread of water vanished as soon as he became convinced that he should have died long ago if he had been rabid.

You see then, gentlemen, that a nervous kind of hydrophobia may develop itself under the influence of intense mental emotion, or when certain excesses or special conditions induce dysphagia or a disgust for food; and medical men may themselves be deceived, if they do not bear in mind the length of the period of incubation of real hydrophobia, and the course of this fearful malady, which invariably kills in the space of three or four days from the first manifestation of the symptoms. It is important that one should be aware of this cause of error, which might have fatal results, because I have known medical men—men of strong minds and of courage, who although well aware of the conditions needed for the development of rabies, for several months, and even years, after having attended persons suffering from hydrophobia and dissected their bodies, were seized with more or less continued dysphagia on the mere thought and recollection of the awful scene which they had witnessed. Time alone got rid at last of this nervous susceptibility, which manifested itself in the shape of spasm of the pharynx, and they cured themselves of it by appealing to their knowledge of the disease, and by forcing themselves to drink some liquid whenever they felt the sensation coming on.

I have related to you, gentlemen, several cases of true rabies occurring in man, and I might easily swell their number by telling you of cases of young children bitten by mad dogs, who came under my observation. I have also given you instances of nervous hydrophobia, so that we might now review together the chief symptoms of human rabies; but before doing so, I wish to give you a sketch of canine rabies. My intention is not merely to draw a parallel between rabies occurring in man and rabies developed in dogs, but I am anxious also to teach you how to recognize canine madness, and thus furnish you with the best preservative against human rabies. For if hydrophobia could be always recognized in a dog, the creature would be immediately sacrificed, and the chances of inoculating the disease to human beings would be thus lessened.

In one of the late discussions on rabies at the Academy of Medicine, M. H. Bouley, clinical professor to the school at Alfort, drew a striking picture of rabies canina, based on what he had seen himself and on quotations from Youatt's work.* There are three well-marked stages of the complaint in the dog. The first is characterized by melancholy, depression, sullenness, and fidgetiness; the second by excitement, by rabid fury; and the third and last, by general muscular debility and actual paralysis.

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* Bulletin de l'Académie Impériale de Médecine, Paris, 1863, t. xxviii, p. 743, *et seq.* See also, Rapport général fait à la demande du gouvernement sur divers remèdes proposés pour prévenir ou pour combattre la rage, by Bouchardat. Bulletin de l'Acad. Imp. de Méd., Paris, 1852, t. xviii, pp. 6 to 30, and 1855, t. xx, pp. 714 to 727.

Whether the disease originated *de novo*, or was communicated, the dog looks ill and sullen after a period of incubation of very variable length: he is constantly agitated, turning round and round inside his kennel, or roaming about if he is at large. His eyes, when turned on his master or friends of the house, have a strange look in them, expressive of sadness as well as of distrust. His attitude is suspicious, and indicates that he is not well; by wandering about the house and the yard, he *seems to be seeking* for a remedy to his complaint. He is not to be trusted even then, because although he may still obey you, yet he does it somewhat slowly, and if you chastise him, he may, *in spite of himself*, inflict a fatal bite. In most cases, however, a mad dog respects and spares the persons to whom he is attached. But his agitation increases; if he is in a room at the time, he runs about, looking under the furniture, tearing the curtains and carpets, sometimes flying at the walls as if he wished to seize a prey. At other times, he jumps up with open jaws as if trying to catch flies on the wing; the next moment, he stops, stretches his neck, and seems to listen to a distant noise. He probably has then hallucinations of sight and hearing, seeing objects that do not exist, and hearing sounds which are not emitted. This delirium may still be suddenly dispelled by his master's voice, and, according to Youatt, "dispersed by the magical influence of his master's voice, all these dreadful objects vanish, and the creature creeps to his master with the expression of attachment peculiar to him. There follows then an interval of calm; he slowly closes his eyes, hangs down his head, his fore-legs seem to give way beneath him, and he looks on the point of dropping. Suddenly, however, he gets up again; fresh phantoms rise before him; he looks around him with a savage expression, and rushes, as far as his chain allows him, against an enemy which only exists in his imagination." By this time, already the animal's bark is hoarse and muffled. Loud at first, it gradually fails in force and intensity, and becomes weaker and weaker, apparently indicating incomplete paralysis of the muscles of the jaws, just as the dropping down pointed to paralysis of the muscles of his fore-legs. In some cases the power of barking is completely lost; the dog is dumb, and his tongue hangs out through his half-opened jaws, from which dribbles a frothy saliva. Sometimes his mouth is perfectly dry, and he cannot swallow, although in the majority of cases he can still eat and drink. When he has vainly attempted to swallow, he probably believes that it is because some foreign body sticks in his throat, for he puts his muzzle between his paws, and works with them as if he wanted to get rid of this.. Although he can no longer drink, people are misled into the belief that he does so from his lapping fluids with great rapidity. On close examination, however, the fluid is found to keep the same level in the vase which contains it, and one can see that the dog does not in reality swallow, that he does not drink, but merely bites the water. Although he cannot swallow fluids, he can still, in some cases, swallow solids, and he may then swallow anything within his reach, bits of wood, pieces of earth, the straw in his kennel, &c. This circumstance is one of very great importance to bear in mind, because when the body of a mad dog is dissected, a good many substances which have not been digested may be found in his stomach, and do thus furnish a proof of his complaint.

One period of the disease does not pass suddenly into another, but by an easy transition. Even in the first stage, that of depression and melancholy, the animal is from time to time very agitated, and shifts his posture. This agitation increases to a considerable degree, and in the second stage constitutes the rabid fury which characterizes this period, together with the hallucinations of sight and hearing. During this second period the animal drops down in a state of exhaustion, after paroxysms of rage; he seems completely prostrate, his head hangs down, his limbs give way under him, and he can no longer swallow. These are signs of incipient paralysis.

Towards the close of the second stage, the dog often breaks his chain, and runs far away from his master's house; he wanders about in the fields, seized from time to time with paroxysms of fury, and then he stops, from fatigue, as it were, and remains several hours in a somnolent state. He has no longer the strength to run after other creatures, although if he be worried, he can still gather strength to fly at and bite an individual. If he be not destroyed, as he wanders about, he generally dies in a ditch or in some retired corner. He apparently perishes from hunger and thirst and intense fatigue; but veterinary surgeons do not say that he dies from asphyxia brought on by spasm of the pectoral muscles or by convulsions.

Such, gentlemen, are the chief symptoms of rabies when occurring in dogs. It has not been my intention to give you a complete description of rabies in the lower animals, but merely to teach you how to recognize it in the dog. Van Swieten had already divided into three stages the course of confirmed hydrophobia in the human subject, the prominent characters of which were melancholy, furor, and asphyxia. When we inquire into the course of the symptoms of rabies in man, we shall see that this clinical division is founded in fact. This terrible malady is always inoculated to the human subject. It may have an incubation-stage, varying from a few days to a year; but the disease generally shows itself from one to three months after the infliction of the bite. The cases are rare in which it developed itself after three months, and still more rare in which it came on from the sixth to the twelfth month, and one is almost authorized, from the statistical observations that have been made, to question the authenticity of the cases in which the disease set in a year after the person had been bitten. *A fortiori*, must one regard with suspicion those instances of the disease in which the incubation is stated to have been more prolonged. These latter may not have been cases of true rabies, but of nervous hydrophobia, similar to those which I related to you, and in which the mere recollection of this awful complaint sufficed to bring on a more or less prolonged dysphagia.

During the incubation-stage there is no disturbance of health, no symptom which may excite suspicion, and, according to Van Swieten, persons who afterwards die of hydrophobia may contract diseases of various kinds, and even virulent diseases, such as variola, without the course of the rabies being thereby modified in the least. The virus of variola has, therefore, no influence on that of rabies, since it does not retard the evolution of the latter complaint, if it shows itself during the incubation-stage.

After the incubation-stage has lasted two or three months, the person who has been bitten suddenly becomes unusually sad; he either does not suspect his complaint, or, if he remembers having been bitten, carefully avoids to mention the circumstance, and seeks amusement away from home. But wherever he may be, his sleep is disturbed, and he often starts up; he feels constantly fidgety, sighs deeply, shuns his friends, seeks solitude, and begs that perfect silence be observed about him; any attentions shown him increase his restlessness and agitation.

Aggravation of these symptoms indicates the beginning of the second stage of the disease. Other phenomena show themselves also. The patient complains of a sense of discomfort about the precordial region; his respiration is sighing, his pulse irregular, as was noted in M. B. and in several of Van Swieten's cases. These disturbances of respiration and circulation, and the sadness and agitation, point to an already marked modification of the nervous system. This goes on increasing, and rigors supervene, which are true convulsions of all the muscles of the body. Then a symptom shows itself, which is nearly constant in confirmed rabies attacking the human subject, namely, *a dread of water*.

The sight of water is frequently sufficient to bring on shuddering; but it is when the patient carries water up to his lips that he is seized with the terrors characteristic of the disease, and with those convulsions of the face and of the whole body which make so deep an impression on the bystanders. A rabid individual is perfectly rational: he is thirsty, tries to drink, and commands his hand to raise to his lips a glass of water; but the liquid has no sooner touched his lips, than he draws back in terror, and sometimes exclaims that he cannot drink; his face expresses pain, his eyes are fixed, and his features contracted; his limbs shake, and his body shivers. The paroxysm lasts a few seconds, and then quiet seems to be gradually restored; but the least touch, nay, mere vibration of the air, is enough to bring on a fresh paroxysm, so great is the cutaneous hyperæsthesia in some cases. The patient cannot wash his hands or face, or comb his hair, without being at once threatened with convulsions.

During the intervals of calm, he sometimes complains of pain in the stomach and of nausea; when he is actually sick, he brings up greenish, porraceous matters. I have already mentioned priapism. This peculiar condition of the genital organs is exceedingly painful, and patients usually express their sufferings in terms which cannot be repeated here. In some cases the patient is seized with sudden terror; he turns abruptly round, fancying that somebody calls to him; there are hallucinations of sight and of hearing. Dr. Bergeron's patient heard the ringing of bells, and saw mice run about on his bed.

You must have been struck, gentlemen, with the many points of resemblance between these two first stages of human rabies and canine madness. In the human subject and in the dog the same symptoms show themselves: melancholy, sadness at the onset of the complaint, a desire to go away from home and to shun friends, agitation, restlessness, and hallucinations.

But as the agitation and the sadness increase, the second stage begins. In man, however, the disorders of innervation may differ from those in the dog, although satyriasis and hallucinations may be present, and the nervous system be deeply affected in both. In the human subject there is cutaneous hyperæsthesia; in the dog, on the contrary, sensibility seems to be abolished, for rabid dogs have been known to seize between their teeth a red-hot poker without evincing any sign of pain, and they scarcely move away when fire is set to the straw or the tow on which they may be lying. Lastly, whereas a rabid man shows a dread of water, a mad dog seeks water, and jumps into rivers, and bites the water; but he cannot drink, because he cannot swallow, and as in man, the dysphagia is probably dependent on spasm of the pharynx. He has hallucinations also, for he flies at imaginary objects, and hears imaginary noises. In the human subject there are at first clonic, and then tonic convulsions of the muscles of the life of relation, of those of deglutition, and of respiration, during a paroxysm; his aspect strikes one with fear, but he has no desire to bite, and does not strike the persons about him. A mad dog, on the contrary, flies at all the beasts which he meets with, and especially at dogs. In his rage he bites, while a horse kicks and tears with his teeth, and a ram or bull butts with his head. A dog bites because his teeth are his weapons of defence and offence; but we may well wonder why a rabid man does not strike with his arms. The reason is that during a paroxysm of rabies, a man is not in a state of furious anger, but is merely convulsed.

In the third stage of human and canine rabies, there are many points of resemblance, but as marked differences also. In both cases, each period of the disease is not abruptly separated from the others; but, as Van Swieten pointed out, the symptoms become more serious, and as fresh ones show themselves, there are degrees, rather than stages, of the disease.

In the third and last stage of the complaint, there is more intense thirst, while there is greater incapacity for drinking: the voice becomes hoarse, at first intermittingly, and then continuously, probably in consequence of spasm or paralysis of the laryngeal muscles. In the last hours preceding dissolution the patient's mouth is often full of a whitish froth, which he spits out constantly. Now, is this froth merely the result of the constant agitation of the saliva through the movements of the cheeks, the lips, and the tongue? or is it due to the patient's inability to swallow his saliva? or, again, does it consist of a mixture of saliva with a variable amount of bronchial froth, driven into the mouth by spasm of the pharynx?

The sight of this froth, and the constant spitting, sometimes alarm the patient himself: he thinks that the matter which he expectorates may do harm to the persons about him, and, as in the case reported by Dr. Bergeron, he begs that nobody is to come near him, and dreading for others the contact of his lips, he refuses to kiss his friends, he dreads to communicate his complaint to them, *aliis à se metuens*, as Boerhaave has it. Van Swieten mentions that a man communicated rabies to his two sons by kissing them, although he does not wish to draw absolute conclusions from the case. Medical men of the present day, however, are little inclined to believe that any danger can accrue from the contact of the saliva of a rabid individual with unbroken skin; but even those who think that there is then no cause for fear, take very good care to wash thoroughly the parts which may have been in contact with the saliva of a rabid individual. I am of opinion that one should prudently avoid all contact with the patient's saliva, as the dog's saliva is capable of imparting the disease to man, and as it has been shown by experiments made by Magendie and Breschet, in 1813, and afterwards confirmed by Renault, at Alfort, that dogs have become mad after being inoculated with the saliva of a rabid man.

As the voice becomes hoarse, and the patient keeps constantly spitting, the convulsive seizures get more and more frequent, and recur spontaneously, without any determining cause. The close of each seizure is attended with spasm of the respiratory muscles, and signs indicating some obstruction to the breathing. On this spasm lasting a long time in one of the seizures, the patient dies asphyxiated, *mors convulsiva cum summâ in respirando angustiâ.* Rabies, in the dog, often lasts several days; in the human subject death always occurs within four days after the first rigor and difficulty of deglutition have set in. The mad dog apparently dies palsied, while the human patient dies from a tonic convulsion of the respiratory muscles. This is an important difference which should be noted in the final stage of canine and human rabies. Van Swieten, however, in his comments on Boerhaave's 1138th aphorism, relates a case of hydrophobia occurring in man, in which death was not preceded by convulsion, or even by struggles, and seemed to result from general paralysis, *ac si universalis paralysis mortem induxisset.*

There is no symptom during the period of incubation of the disease which indicates that the individual has been inoculated with the virus of rabies. In a great many cases, no ill effects have followed a bite inflicted by a rabid dog. We may then suppose that no virus was deposited in the wound; or, if the view be not admitted that some individuals are not susceptible of the poison, it may be conjectured that from some special conditions, no absorption of the poison took place. Whatever hypotheses may be started on this point, facts prove that of several persons and several animals bitten by the same mad dog, a few only become rabid. During the period of incubation, which may extend over several months, no sign will indicate which individuals out of the number will fall victims to the bite. No change in their habits or their functions awakens suspicion; and yet I wish you to remember that one of my patients, some time before the invasion of the complaint, exhibited a return of sexual aptitude which he seemed to have lost a long time ago. If, during the period of incubation, doubts and fears may exist, all uncertainty comes to an end when the stage of invasion begins. The muscular debility complained of in many cases, the restless sleep out of which the patient starts up, his continual fidgetiness, his suspicious breathing, his sadness and search after pleasure, and then his love of solitude, must awaken terrible fears in the practitioner, especially if there be no moral causes or no organic lesions to account for these symptoms satisfactorily. The intense thirst, general muscular pains, and rigor which might at first be ascribed to some grave febrile affection, are followed by a symptom which is almost pathognomonic of rabies, namely, a sudden difficulty in swallowing liquids, water in particular. When there is complete inability to drink, and when this dysphagia is immediately succeeded by tremor on the patient carrying some liquid to his lips, all illusion is dispelled, and it becomes clear that the patient is under the fatal influence of the virus of rabies. There is such a thing as nervous hydrophobia, true dysphagia brought on by a dread of rabies, and I have related to you examples of it; but the sudden invasion of this complaint, generally coming on through the person recalling to mind or hearing the relation of a case of true hydrophobia, and the duration of the dysphagia over the period of four days, are amply sufficient to characterize the complaint, and to enable the practitioner to persuade the patient that he is suffering from mere nervous symptoms which will vanish as soon as he ceases to fear. Besides, in nervous hydrophobia, there is dysphagia only, but no general convulsions, the spasm affecting the pharynx alone, while the breathing goes on with regularity.

Maniacs sometimes also evince a dread of liquids, and refuse to drink, and like persons suffering from rabies in the second stage, they are exceedingly agitated and loquacious, and have hallucinations, but they never have general rigors and spasmodic convulsions. They are, besides, delirious on all subjects, whilst a rabid individual retains all his reason, although he may occasionally have transient hallucinations. He is anxious to get well, and believing that his complaint is merely due to his inabilty to drink, he submits to any treatment; and once the paroxysm of excitement over, he allows a strait-jacket to be put on him without offering the least resistance. A maniac has lost his reason; in rabies, on the contrary, as Boerhaave stated long ago, even in the last stage of the disease, the patient retains his firmness and common sense, and requests the persons about him to keep some distance from him, because he dreads lest he should communicate his complaint to them.

I may here mention, that in the beginning of this century, a Russian physician, Dr. Marochetti, in a memoir on hydrophobia, and Dr. Xanthos, of Siphnos, in a letter to Hufeland, called attention to the presence, on the under surface of the tongue, near the frænum, of pustules or vesicles of a special character, during the stage of incubation of rabies. These had been long known in Greece under the name of *lyssi*. Drs. Marochetti and

Xanthos did not claim for themselves the credit of this important discovery: it was traditionally known, they said, in Russia and in Greece; and they had been told that if these vesicles or pustules were laid open in time and cauterized, all manifestations of rabies were prevented. Dr. Marochetti, in particular, frequently put this plan to the test, and succeeded completely; he recommends at the same time, it is true, the use of a ptisan of *genista-tinctoria* (Linnæus) which has for a long time been used in Ukraine against rabies. The presence of an eruption under the tongue subsequent to inoculation with the virus of rabies, seems to me to be a fact of such importance, that it is my duty to call your attention to it, as it has been observed by men whose scientific honesty we have no right to suspect. I must observe also, that if the presence of this vesicular eruption has not been ascertained in France since the publication of Dr. Magistel's work,* it is because practitioners have not, in general, looked for it during the period of incubation of rabies, although Drs. Marochetti and Xanthos have particularly stated that it showed itself during the first few days after the inoculation. Dr. Magistel says, in his memoir, that he saw the *lyssi* in different individuals on the sixth, the eleventh, and the twentieth day, and that, after the twenty-second, he never succeeded in finding them, although he looked for them until the thirty-fourth day after the inoculation of the virus. It would seem, therefore, that this peculiar sublingual eruption may be met with in a certain number of cases of rabies, but that one must know how, and particularly when, to look for it, namely, at the commencement of the incubation-stage, and not in the period of invasion or of confirmed rabies, since the eruption has disappeared by that time, without leaving any trace behind. In the present state of science, we may not perhaps have the right to deny, as has been done, the presence of lyssi in rabies. This eruption should, therefore, be searched for in persons who have been recently bitten by a rabid animal, and whose wounds have not been cauterized.

The accuracy of the statements made by Drs. Marochetti and Xanthos may easily be tested, because the wounds inflicted by rabid animals are rarely cauterized sufficiently early and to a sufficient depth so as to prevent absorption of the virus, so that in a certain proportion of cases, the presence of lyssi ought to be made out from the third to the twentieth day after the inoculation.

I need not dwell on the advantages that would be obtained if the statements made by Drs. Marochetti, Xanthos, and Magistel were confirmed. Rabies could then be diagnosed during its incubation-stage, and if by laying open and cauterizing these vesicles the ulterior manifestations of the disease can be prevented, the complaint could be cured, as soon as the sublingual eruption was detected.

I cannot, therefore, too strongly recommend practitioners to look out for the presence of lyssi in individuals who have been exposed to the risk of being inoculated with rabies. The examination should be made regularly twice a day, according to Marochetti, because the eruption does not show itself on a fixed day, and the vesicles break easily. The same authority adds, that the eruption comes out at an earlier period in proportion to the amount of poison deposited in the wound, and that the invasion of the confirmed disease comes on also earlier, according to the early date of the appearance of the eruption.

There seems to have been of late a disinclination to attach any importance to the presence of this eruption, and it has been said that it was a very extraordinary circumstance, without its analogue in pathology, that the virus of rabies should be localized—stored up in a particular region. I cannot entirely concur in this opinion; and without desiring to prove that this localization of the virus is a perfectly natural phenomenon, which might have been foreseen, I will merely call attention to the fact that in most virulent diseases, a primary localization of the virus may be detected, in a particular tissue or organ, and that the disease gives rise to general manifestations only secondarily. Thus, in eruptive fevers, we see the morbid principle affect the skin primarily, and the lumbar portion of the spinal cord in small-pox, the bronchial and laryngeal mucous membrane in measles, and the kidneys in scarlatina. We see syphilis limit itself in the first instance to the lymphatic ganglia in the groin and in the occipital region, and station itself for a time in the lymphatic system before giving rise to secondary manifestations in mucous membranes and in the skin. Again, we see the poison of glanders affect the mucous membrane of the nose in the beginning, and it is only after a time that other mucous membranes are involved, together with the skin, the cellular tissue, the joints and viscera.

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* Mémoire sur l'hydrophobie, or Journal de l'hôpital de Burley, Paris, 1824.

If in virulent diseases in general, therefore, the virulent principle has a primary elective seat, why should one refuse to admit, on *à priori* grounds, that the virus of rabies, when inoculated into any part of the body can act primarily on a determinate and localized region? Why should any surprise be felt at its selecting the free extremities of the excretory ducts of the salivary glands, when no doubt is nowadays entertained as to the saliva itself being the vehicle of the poison? Again, why wonder that a special eruption should be confined to the region where the excretory ducts of the sublingual and submaxillary glands terminate, when experiments by Claude Bernard have proved that all the salivary glands do not possess similar properties? Lastly, when it has been experimentally shown that certain substances, such as iodine and the iodides of potassium and of iron, are more rapidly eliminated by the salivary glands than by any other organs, why should it not be admitted that these glands may eliminate an organic poison, a virulent principle, which, after a definite time and by virtue of special circumstances, lodges and is stored up in the extremities of the excretory ducts of these glands, or in the salivary follicles in their vicinity?

Marochetti thought that the virus which had been thus deposited was after a certain time absorbed, and then gave rise to all the symptoms of confirmed rabies. He, therefore, followed the traditional practice of the inhabitants of Thessaly and Ukraine, and made an early incision through the vesicles, so as to give issue to the virulent matter, and then cauterized them with a red-hot iron. He affirms that this plan always succeeded in the numerous cases which came under his observation in Ukraine. One cannot take too much pains to look for this peculiar eruption, since it would seem to be the only sign by which the incubation-stage of rabies can be diagnosed, and since by cauterizing it, one may arrest the fatal progress of the disease.

In a great many old books, you will find that the first symptoms announcing the invasion of the disease, show themselves at the seat of the bite. Boerhaave himself states that the wound becomes painful again, and that vague pains subsequently show themselves in the neighboring regions also. It has been further said by some that wounds which had healed opened afresh; and Salius Diversus, who fancied that he had discovered an infallible sign of threatening rabies, asserted that a peculiar pain was set up at the seat of the bite, and from there ascended by insensible degrees to the brain in the space of three or four days, and gave rise to vertigo. This pain would be, therefore, a kind of aura, analogous to the aura of epilepsy and hysteria, with this difference, however, that its progress upwards is very slow.

When men like Boerhaave and Van Swieten declare that such phenomena have been noted, no one can refuse to believe that they may occur, although it should be observed that in cases recorded in modern times by careful practitioners, no allusion is made to any such phenomena. Thus, in Dr. Bergeron's case, which is given with such full details, it is distinctly stated that the scar left by the bite *had undergone no change* and was not painful. In the case under my care, in St. Agnes Ward, no painful scar could be found on the hand that had been bitten. Lastly, in the cases reported by Dr. Peter and by Dr. Eug. Fournier, not the slightest trace of the bite could be seen; yet, I must recall to your memory that two days before he was brought to the Beaujon Hospital, the subject of Dr. Fournier's case complained of pain in his right arm, and said that he was being probably *seized with rabies in the arm* which had been bitten.

The *prognosis* of rabies, in the stage of invasion, when dysphagia and convulsions have shown themselves, is always unfavorable. Nothing that has been tried has ever succeeded in arresting the fatal progress of this fearful complaint, and death occurs more rapidly in proportion to the frequency of the convulsions.

I now pass on to the *etiology* of rabies in the human subject.

The last discussion which took place at the Academy on rabies, and in which Messrs. Vernois, Bouley, and Tardieu joined so brilliantly, referred chiefly to the etiology of the disease in the dog and the human subject. From the facts stated in the course of that discussion and the results of statistical investigations, an important inference can be drawn, namely, that rabies very rarely occurs in the human subject. In France, with a population of more than 36,000,000, there are only every year, on an average, from 20 to 25 cases of rabies, that is, less than two cases to a million of people. And yet there is a certain proportion of bites inflicted every year by rabid dogs, and statistical researches have shown that the bite proves fatal fifty times out of a hundred. These facts would seem to indicate that certain individuals are not susceptible of the influence of the virus. But if we bear in mind that viruses when inoculated take effect in nearly every case, it would perhaps be more rational to believe that every bite does not inoculate the virus of rabies, either from the virus not having been secreted by the salivary glands at the time, or from its being wiped off from the tooth in its passage through the clothes. This last hypothesis is supported by the fact that the most dangerous bites are those inflicted on exposed parts of the body, such as the face and hands.

Rabies is communicated to man by different animals in the following order with regard to frequency: first comes the dog, next the cat, and then the wolf and the fox; in very exceptional instances it has been transmitted by cows and by horses. The chief cause of the difference in the results following the bites of these animals lies in the fact that the first on the list make use of their teeth when they attack man, whilst the last strike with their head or feet. A horse may, however, inflict terrible wounds with his teeth, so that one must be very cautious when he gets near a rabid horse, because his teeth may be charged with virus in a fit of anger.

It is generally said that the period of childhood favors the rapid development of rabies. But this saying seems to me to be one of the results of misinterpreted statistics. If it be true that children are more frequently seized with rabies than adults are, the probability is that this does not depend on a greater morbid susceptibility arising from their age, but on their frequently playing with dogs who bite them when they are rabid, and on their being too inexperienced to recognize the strange aspect of a rabid dog, and too weak to defend themselves or to avoid danger. Can the bite of an angry dog, who is not rabid, give rise to rabies? One cannot conceive how an animal can communicate a virus which he himself has not about him, and if this were unfortunately the case, the number of cases of rabies would be infinitely greater, for there are very few individuals who through life escape being bitten by a dog. Or "we should be compelled to admit," as Mr. Bouley remarks, "that there may exist in the dog a perfectly transient and evanescent rabid condition, during which the animal's saliva is virulent, but after which it becomes normal again." Such a view would be a pure hypothesis, while facts show, on the contrary, that a dog who has communicated rabies dies himself of the disease. I must not, however, omit to mention a case recorded by Dr. Camille Gros, as having been under Dr. Tardieu's care in the Lariboisière Hospital, and which is one of the rare instances apparently pointing to this as an exceptional cause of rabies.*

Van Swieten reports that an old woman died with all the symptoms of rabies, after a wound inflicted on her by an irate cock; but, as he could not admit that a virus not present in an animal could be communicated by that animal, he conjectures that the cock was suffering from rabies which had been imparted to it by a fox. He adds besides, that if rabies could be spontaneously generated in the cock, we ought to be surprised why it does not more frequently occur in England, where this irascible and quarrelsome bird is trained to fight.

Malpighi declares also that his own mother died of rabies a few days after being bitten by an epileptic. But, in spite of the authority of these writers, I believe that the cases which they have recorded are very questionable. We should be equally incredulous as to the cases of spontaneous rabies said to have been observed in the human subject. M. Vernois† cited, in proof of the spontaneous origin of rabies in man, cases observed by Dr. E. Gintrac‡ of Bordeaux, and by Dr. Barthez; but as M. Velpeau remarked, and justly in my opinion, there was no absolute proof that there had been no contagion in these cases, for it is not necessary that there should be a bite for rabies to develop itself in man. Some portion of the body, denuded of epidermis, need only be in contact with the virus of rabies; and this may occur on the dog licking a person's hand. Van Swieten also mentions that a young man died of rabies after having bitten his own finger in a fit of passion. It may be that, in this case as in many others, traumatic tetanus was mistaken for rabies.

I myself am of opinion that rabies in the human subject is always the result of inoculation with the virus of rabies, and that those cases in which the disease is said to have been communicated by dogs that were not mad, or to have been generated *de novo*, must be regarded as instances of traumatic tenanus or of nervous hydrophobia.

The pathological changes found after death, in cases of rabies, are only those dependent on the asphyxia which occurs in the last stage. Morgagni has studied with considerable care this part of the subject of rabies in his eighth letter, and the conclusions which he came to are similar to those which have been arrived at from dissections made of late years.

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* Thèses de Paris, 1860. Considérations sur la rage, by Camille Gros.

† Etude sur la prophylaxie administrative de la rage (Annales d'hygiène publique at de médecine légale. Paris, 1863, t. xix, p. 52).

‡ Journal de Médecine de Bordeaux, August, September, and October, 1862.

Hyperæmia of all the parenchymatous organs is alone met with, as a consequence of the final convulsion.

Dissection gives no clue, therefore, to the nature of the complaint, but an analysis of the symptoms and the etiology of the disease lead one to regard it as a virulent malady. The virus contained in the saliva of rabid animals is the sole source of the contagion of rabies, as has been shown by the experiments made by Professor Renault (of Alfort), and by the circumstances under which rabies affects the human subject after a bite inflicted by a dog, a wolf, or a cat, or after the virulent saliva has been in contact with a denuded portion of the integuments. Rabies is, therefore, a virulent disease which should be placed by the side of glanders, another virulent complaint communicated to man by horses. The virus of rabies remains for a variable time in the system, after its introduction into it, without giving rise to any appreciable lesion, except the sublingual eruption, the presence of which is to be regarded as doubtful until confirmed by other observers. It should be remarked, however, that many authors, previous to Marochetti, had spoken of these small tumors under various names, and as being of very various nature; so that, whatever conclusion may be arrived at by and by, concerning them, it must be admitted that the numerous discussions to which they have given rise seem to tell in favor of their existence.

Ettmuller states that up to the 17th century a good deal of attention had been paid to the presence of these sublingual swellings, and he adds, after quoting statements made by many writers, "Others are of opinion that there is no small worm concealed beneath the tongue of a mad dog, but that the swelling which is found there consists of some of the granular blood which stagnated underneath the tongue, in the ranine veins. I have not come to any conclusion on the point, because I have not sufficient data to go upon." This great reserve, which Morgagni showed also, should be imitated until further light is thrown upon the subject.

I believe that rabies is never generated *de novo* in the human subject, but is communicated by the dog. We should therefore learn to recognize and guess it even in that animal, as he can so easily transmit it by his caresses or bites. Lastly, after inoculation has occurred, measures should be unhesitatingly employed which destroy at once all property in the virus, and prevent the fatal evolution of the disease.

Cauterization is, after all, the only measure from which a successful result may be anticipated, and, in order to insure success, it should be done immediately after inoculation of the virus. Delay allows absorption to take place, and the part should therefore be at once and deeply cauterized. By going beyond the area of the virulent inoculation, a more or less extensive wound will be produced, but which gives rise to no risk, while imperfect cauterization exposes the patient to the risk of dying.

A red-hot iron suffices for destroying the tissues of the bitten part, and it has the advantage of acting quickly, and of leaving behind sloughs which take some time to come away. Récamier recommended the acid nitrate of mercury, because it penetrates the tissues deeply, and rapidly disorganizes them. Caustic potash and corrosive sublimate also answer the purpose of destroying the tissues, and rendering them inapt to absorb the virus. Either of these caustics may be used, the first and chief indication being to act quickly and deeply.

Rabies will not be developed if the wound has been cauterized sufficiently early and deeply; but if this has been done inefficiently, is there, we may ask, while the disease is incubating, a symptom which warns us of the threatening peril, and which may be used as a guide to treatment?

The generally accepted opinion is that there is no special symptom to be detected during the incubation-stage, however prolonged it may be. Still, we should bear in mind the facts handed down by tradition, and we should take into account the extreme reserve shown by Ettmuller, who admits the facts, although he adds that he is not in a position to decide on the nature of the tumors which are developed under the tongue of persons inoculated with the virus of rabies; and lastly, we should, until further observations are made on this point, credit the statements made by Xanthos in his letter to Hufeland, and by Dr. Marochetti in his memoir. Besides, we have no cause for doubting the value of the cases recorded by Dr. Magistel. Attention should for the future, therefore, be directed to this point, and I cannot too strongly recommend to you to look for these sublingual swellings in all persons that have been bitten by mad dogs. If such an eruption does really exist, and if by laying open and cauterizing the vesicles the disease can be arrested, our fears will be set at rest.

At the same time that the lyssi are cauterized the treatment recommended by Dioscorides and Celsus should be put in force. Mr. Gosselin has lately advocated the same plan again, which he tried in the case of a girl who had been bitten by a mad dog, but who did not become rabid.* Celsus aimed at renewing the fluids in the body by calling into exaggerated action the skin, the liver, the kidneys, and the intestines, and in facilitating repair by a highly nutritious diet, open-air exercise, and prolonged immersion in water. Mr. Gosselin had probably the same end in view, by inducing abundant perspiration, by repeatedly purging the patient, and by recommending violent muscular exercise, and daily sulphur baths. This debilitating treatment produced very rapid emaciation, notwithstanding the amount of food taken by the patient. She was discharged well, however, from the hospital, and no symptom of rabies showed itself in her afterwards, although her wound had not been cauterized. We cannot draw any conclusion from a single case of this kind, especially as about one-half only of the individuals bitten by a mad dog become rabid; but as this method is not attended with any serious risks, while it may prove successful, I believe that it should for the future be put in force during the incubation-stage. But if, in spite of all that has been done, rabies should set in, what is the practitioner to do? He may do anything, since the patient is doomd to die. Venesection pushed to syncope has been advocated, with the view of emptying the vascular system, and of getting rid of the virus with the blood at the same time. This treatment has not been attended with good results, however; and when the patient did not die from the prolonged hemorrhage, he died a few hours afterwards during a spasmodic seizure.

The ancients had a celebrated method of treatment for rabies, called the *sailors' method*. The rabid subject was dipped into the sea, into a river, or a bath simply, until he was nearly asphyxiated. Van Swieten mentions cases in which a cure was obtained by means of this double action of water and asphyxia. Euripides is said to have been cured of rabies by this method, and to have from gratitude written that the sea washed away all the ills of man. But the medical men who sanctioned the sailors' treatment probably meant to act with energy on the nervous system of their patients, for these unfortunates were thrown into the sea when they least expected it, and force was employed when they made any resistance. This is a barbarous plan, which could only be excused if it were always successful. Tulpius had great faith in this treatment, and he affirms that "in the populous city of Amsterdam, where rabies was common, he never saw a fatal case of this disease when the patient had been dipped into the sea at a proper period."

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* Bulletin de l'Académie Impériale de Médecine. Paris, 1863, t. xxix, p. 22.

This plan was chiefly recommended during the incubation-stage, and at the beginning of the period of invasion. But what is to be done when the mere contact of water with the lips brings on spasms, and do the chief symptoms of this last stage of the complaint, the extreme agitation, the convulsions, the abundant secretion of saliva, furnish us with some indications of treatment? As sleep calms all nervous excitement, and suspends the convulsive paroxysms, it seems very rational to me to treat the symptoms, as we cannot act on the morbid cause itself. Opium in large doses would, by inducing profound sleep, answer the double purpose of quieting the nervous excitement and of delaying the convulsions. Mixtures containing opium are of course out of question, since at this period of the disease the patient cannot swallow; but morphia can be used by the endermic or the hypodermic method. Large doses of this salt can be very rapidly introduced into the blood, either by sprinkling with it the raw surface of a blister raised by strong liquid ammonia, or by injecting it under the skin. When once sleep has been procured in this manner, it should be kept up as long as necessary, that is to say, until the disappearance of all spasm as the patient wakes up.

Chloroform might be also used against the spasms of rabies, for the power of chloroform inhalations to stop all convulsion is well known; but in order to obtain satisfactory results the convulsions should be forestalled, and with that view the patient should be for several hours every day kept under the influence of the anæsthetic, as has been successfully done in cases of eclampsia.

Could the *curara* poison, if injected into the veins, or into the subcutaneous cellular tissue, in sufficient doses, frequently repeated, modify the convulsive influence of the virus of rabies, by acting on the nervous system intermittingly? Curara has, however, been used without any satisfactory result in tetanus, although the failure may perhaps, in great part, be ascribed to the manner it was administered. We have seen how far opium and morphia could, by inducing sleep, quiet the nervous excitement, and prevent the return of the convulsions. We have seen how chloroform inhalations and curara could arrest the spasmodic convulsions from their special action on the nervous system. But by so doing, we only follow the indications furnished by the principal symptoms of the disease. Let us inquire, however, whether there be not a specific remedy, an antidote of rabies. In this complaint the saliva seems to be the sole vehicle of the virus, as shown by experiments made by veterinary surgeons. Attempts have naturally been made to modify, by means of mercury, which acts on the salivary glands specially, the salivary secretion and the composition of the blood. According to Van Swieten, mercurial preparations have been of service in hydrophobia. The Chinese believed the following formula to be *infallible:*

℞. Musk, half an ounce.
Native cinnabar, } of each, five drachms.
Artificial cinnabar, }

These substances were rubbed down together to an impalpable powder, and were then given suspended in a spoonful of rice-spirit. Calm sleep and copious perspiration came on after two or three hours; otherwise, a second dose of the powder was given, and a cure was considered as sure to follow.

Van Swieten tells us also that mercurial preparations alone, without musk, have proved useful in rabies, and mentions as a proof of his assertion the fact, that of two hundred beasts bitten by mad dogs, and to which turpeth mineral was given in doses of from twelve to twenty-four and forty-eight grains, not a single one died. He relates also, that a young man who exhibited all the symptoms of confirmed rabies after being bitten by a mad dog, got well by taking every night, for three nights, a drachm of turpeth mineral (the yellow subsulphate of mercury) and a small quantity of theriaca.

I mention these cases because they seem to me to possess great value, as they are given in the work of Boerhaave's commentator; and they should induce us to try again, in dogs that have been inoculated with rabies, the effects of mercurial preparations. Should some improvement follow their use, we should not hesitate to prescribe during the incubation-stage, and at the outset of the period of invasion, mercury in large doses, in order to arrest in the human subject the progress of rabies.

You may perhaps be surprised, gentlemen, that I should dwell so much on the treatment of rabies, especially at a time like the present, when no faith is put in the measures which used to be vaunted in this complaint. But the fact that it is almost universally regarded as incurable, compelled me to bring to your notice methods of treatment recommended by trustworthy practitioners. And, rather than I should authorize you, by my excessive reserve, to remain perfectly inactive in a case of rabies, I have preferred to bring before you, and thus induce you to repeat, trials made by our predecessors, and even to encourage you to make fresh trials, by pointing out to you the way which seemed to me the best.

In presence of a complaint which terminates constantly in death, the practitioner's duty consists in boldly trying everything.

LECTURE LV.

ASTHMA.

Its Characters differ according to the Individual affected, and according to his Age.—A peculiar Coryza may be the only Manifestation of the Disease.—The same holds good with the Catarrh, which is usually one of the Elements of Asthma, and comes on at the Close of the Fit, but may in some cases be exclusively Predominant, when it then presents Peculiar Characters.—Exciting Causes of Asthma; they are often Absent; when Present, they are exceedingly Variable, and occasionally very Singular.—Influence of External Circumstances: Climate, Seasons, and Temperature.—Opinion of Authors on the Nature of Asthma.—Dyspnœa Symptomatic of an Affection of the Heart or of the Great Vessels, of Pulmonary Emphysema, of Bronchial Catarrh.—Asthma is a Neurosis, and the Manifestation of a Diathetic Condition, Gout, Rheumatism, &c.—It is also a Manifestation of the Tubercular Diathesis.—Treatment.

GENTLEMEN: Through a fortuitous combination of circumstances, which will sometimes occur in an inexplicable manner, we have had at the same time in our wards several individuals suffering from *idiopathic asthma*, a disease which is common enough, but is rarely seen in hospitals. As the epithet *idiopathic* indicates, this complaint occurs independently of all demonstrable organic lesion, in paroxysms of dyspnœa and oppression, which recur at more or less regular, more or less distant periods, in the intervals between which the respiratory functions generally recover their usual regularity.

Thus, an individual in perfect health goes to bed feeling as well as usual, and drops off quietly to sleep, but after an hour or two, he is suddenly awakened by a most distressing attack of dyspnœa. He feels as though his chest were constricted and compressed, and has a sense of considerable distress; he breathes with difficulty, and his inspiration is accompanied by a laryngo-tracheal whistling sound. The dyspnœa and sense of anxiety increasing, he sits up, rests on his hands, with his arms put back, while his face is turgid, occasionally livid, red, or bluish, his eyes prominent, and his skin bedewed with perspiration. He is soon obliged to jump off his bed, and if the room in which he sleeps be not very lofty, he hastens to throw his window open in search of air. Fresh air, playing freely about, relieves him. Yet the fit lasts one or two hours or more, and then terminates. The face recovers its natural complexion, and ceases to be turgid. The urine, which was at first clear, and was passed rather frequently, now diminishes in quantity, becomes redder, and sometimes deposits a sediment. At last, the patient lies down and again falls off to sleep. On the next day, he transacts business and leads his ordinary life, often having a mere recollection of his past sufferings. In some cases, however, he continues to feel a more or less undefined sensation of thoracic constriction, which is liable to be increased by movements of the trunk, and to then render breathing more laborious and difficult. In other cases, the patient complains of flatulent distension of the stomach after eating, and of an unusual tendency to doze. In the evening, about the same time nearly, a fresh paroxysm comes on, perfectly similar to the one of the previous day, which, like it, yields, to recur on the following night, and again for three, four, five, ten, twenty, and even thirty nights. These paroxysms constitute a true *fit of asthma*, which sometimes terminates in slight bronchial catarrh, which in its turn disappears spontaneously, after some time. The return of the attack is not under the dependence of any law, and in some cases it takes place after the lapse of four or five years, but in others, every year, and even oftener.

You will meet with individuals who suffer from nearly constant fits for several weeks or months. During the day, somewhat more active exercise than usual, a slightly brisker walk, mental emotion, some annoyance, will bring on difficulty of breathing, amounting almost to suffocation, and a disastrous sense of anxiety. In the evening, the paroxysms return regularly with greater or less intensity, without being brought on by any cause. During the night, the dyspnœa is so great that the unfortunate patient is unable to lie down on his back or on his side, and is obliged to sleep in the most varied, and sometimes the queerest attitudes. Sometimes he can only find sleep by kneeling on his bed and resting his head on his knees, or by spending the night in an arm-chair, or by propping himself up in bed in the sitting posture; sometimes again, he can only sleep standing, resting on a piece of furniture or on the mantel-piece.

Whether an asthmatic individual be in bed or up and about, he is generally seized during the night, and, as a rule, also in the early part of the night. There are, however, exceptions to this general rule, for asthma, like all nervous disorders, is capricious, and affects very marked individualities. In every case, the attack returns at the same hour generally, but this may be in the second, and not in the first half of the night.

Thus, I have myself been long subject to asthma, and my fits used to return about three o'clock in the morning. I was then invariably awakened by a sense of oppression, and I heard my clock strike three.

In some cases, the attacks are *diurnal* instead of *nocturnal.*

My mother, from whom I no doubt inherited my asthma, used to be seized between six and eight o'clock in the morning. During the rest of the day, she was as active as possible, and she had good nights.

The master-tailor of a regiment of carbineers, then stationed at Saumur, whom I knew, was regularly seized at three o'clock in the afternoon. His attacks recurred so punctually at the same time that, on account of the period of the day at which they occurred, I ascribed them to paludal influences and diagnosed larvated intermittent fever. I gave him quinine, but without any good results.

Many instances could doubtless be found of diurnal asthma, similar to the above; but they are only exceptions, and by no means invalidate the law that the paroxysms generally return at night.

In some cases, instead of manifesting itself at once by fits of oppression at the *chest*, this singular malady sets in with *coryza.* All at once, and often without his having been exposed to any of the causes which generally bring on a cold in the head, the patient begins to sneeze with extreme violence, and in the most strangely obstinate manner. His nose runs profusely; his eyes swell, and fill with tears; then, after a few hours, these symptoms disappear as rapidly as they set in, and in the course of the evening, more commonly during the night, asthma comes on with its usual characters. During four, five, or six days in succession, and even more, and nearly always at the same time, the same phenomena repeat themselves, and terminate in the same way.

In other instances, the whole paroxysm is exclusively constituted by this paroxysmal coryza, occurring independently of all appreciable cause, or under the influence of causes which are as varied and as curious as those which, as I shall tell you presently, induce an attack of genuine asthma.

At the end of January, 1863, a lady consulted me on account of fits of asthma with which she was seized under singular circumstances. She lived at Narbonne, and whenever the wind blew from the sea, she had a violent cold in the head, which lasted from twenty-four to forty-eight hours, but she had no difficulty of breathing.

She added that one of her children, who was five years old, was also subject for eight or nine months of the year to coryzas, which began with endless sneezing, whenever he exposed his full face to the early rays of the sun or to a fresh breeze.

This year, again, May 19, 1863, I was consulted by an engraver, residing in St. Martin's Street, in Paris, who for the last five years, from the month of March (when he is in the habit of going every Sunday to the country), has been subject to fits of sneezing, accompanied by lachrymation, which recurred two, three, and four times a day. This went on for two or three months, and in the interval between the attacks, his general health was not in the least disturbed. Ten years ago, he had had fits of asthma, and even now he had them every year in the month of February; these fits come on at night only. He had suffered from bleeding piles until five years ago, and his attacks of sneezing had only come on since his piles had ceased to bleed. He had never had a fit of the gout, and although he had never suffered from any skin eruption (with the exception of some pityriasis capitis), he yet had been subject every two or three months for the last five years to a kind of slight eczematous eruption, which lasted from ten to fifteen days. When this eruption showed itself, the sneezing disappeared.

I have often predicted to individuals suffering from this curious form of coryza, who had never felt anything about the chest which could justify my assertion, that they would sooner or later become subject to asthma, and they have subsequently come back and told me that my suspicions had turned out to be true.

It is a fact clinically established, however singular it may appear, and however inexplicable it may be, that, although fits of asthma come on in most instances at night, this paroxysmal coryza, which I regard as one of the manifestations of the same disease, is diurnal, and mostly occurs in the first half of the day. This was the case, as you may remember, in the instance of the man who remained a pretty long time at No. 3, in St. Agnes Ward.

Asthma sometimes, again, assumes the *catarrhal form;* and then the bronchitis, which, as I told you at the commencement of this lecture, occasionally and even habitually ends the attack, seems to be the sole manifestation of the disease.

This occurs in children chiefly, although it is not of very uncommon occurrence in adults.

In the month of January, 1861, I had under my care a lady, who had come from the provinces, and who was seized two or three times a year with a violent catarrh, of which I have never seen another instance. There was continued orthopnœa, with nocturnal exacerbations that were perfectly frightful, and yet the severity of the dyspnœa was by no means proportionate to the signs revealed by auscultation. Sonorous rhonchi were alone heard, and scarcely a few fine scattered mucous rhonchi: the vesicular murmur was nowhere audible. These symptoms sometimes lasted from one to two or three months without intermission; at rare intervals only were there glimpses of improvement, which lasted a very short time, until these prolonged paroxysms ceased somewhat suddenly, leaving behind them no appreciable traces of the disturbances which they had caused.

I repeat, it is chiefly in children that this occurs. Asthma assumes such singular forms in them that it is often unrecognized. I believe I was one of the first to point out, if not its existence in young children, at least the strange forms under which it manifests itself. It is only exceptionally that they are affected exactly in the same way as adults are, and, for my part, I remember only one instance of the kind.

The patient was a Moldavian boy, aged five, who had very distinct and well-characterized fits of asthma, together with some pulmonary emphysema. In his family history, there was no mention of any hereditary taint of gout or of rheumatism. I saw him again two years afterwards; he had then a most characteristic fit of the gout, with redness, swelling, and pain in the big toe. This was the first, and has been the last, instance I have ever seen of gout at such an early age. The gouty arthritis attacked the knees, and had not the slightest resemblance to acute articular rheumatism. During this attack of gout, the boy had not a single paroxysm of asthma. The disease ran its usual course, for, as I will tell you by and by, gout and asthma are often manifestations of one and the same diathesis, and they may alternate in the same individual, as they did in my Moldavian patient.

This form of asthma, which occurs in adults, affects children only exceptionally. In the latter, the catarrhal form is the predominating one, and presents numerous varieties. I will give you cases in proof of this, which will be better than any statement.

One of my confrères, a man of a robust frame, had two children whose health was very delicate. Their mother was hysterical, but was a sensible person notwithstanding, as her sympathetic system of nerves was more affected than the nerves of the life of relation.

One of the children became one day affected with some pulmonary complaint, presenting all the symptoms of broncho-pneumonia. These came on with startling suddenness, as it were, and assumed at once an alarming aspect. I was summoned an hour after they had shown themselves. On ausculting the child's chest, I heard subcrepitant rhonchi in great abundance, and the extreme difficulty of breathing made me dread imminent suffocation. I advised a large flying blister to be applied to the whole chest immediately. Three days afterwards, the child was quite well. My treatment had been followed by too marvellous a success, and especially had been too rapidly successful for me to ascribe to it the credit of the cure. A few months afterwards, however, the same symptoms manifested themselves, but although no active treatment was had recourse to, they disappeared after lasting forty-eight hours. This time, still more than the first, I asked myself whether we had really to do with a peripneumonic catarrh. I recalled to mind what broncho-pneumonia was in infancy. For whilst I had learned from experience, both in hospital and in private practice, that no child dies of genuine lobar pneumonia, the disease yielding in general, if not always, without medical interference, I knew also that it was a different case with catarrhal pneumonia, and that, although a serious complaint at all ages, it was to be dreaded most in childhood, so much so, that of forty cases treated by me in hospital forty had proved fatal whatever treatment had been had recourse to. When I considered, therefore, that my confrère's son had recovered from such a fearful complaint on the first occasion in three days, and on the second in forty-eight hours, I doubted the accuracy of my diagnosis, or at least attempted to complete it by taking into account the family history. When I thought of the mother's hysteria, I inferred that, in the child's case, the nervous element must assuredly have played the principal part, if it had not been the sole agency at work. Hence, when, three months afterwards, I was again summoned to see the same boy, who, after having played as usual during the day, had about ten or eleven o'clock at night another paroxysm apparently as formidable as the previous ones, I advised that *stramonium* leaves should be burnt in his room, confining myself this time to combat the spasmodic element. On the following day, the child was up and about.

His complaint had therefore been a true pulmonary neurosis, complicated with a bronchial secretion, the presence of which had been revealed by the fine subcrepitant mucous rhonchi heard. In this it resembled many other neuroses, which, as I shall tell you on other occasions, are frequently accompanied by abnormal and exaggerated secretions. In a word, I had had to deal with fits of asthma.

This was the first time that I had seen such symptoms in a child, or rather it was the first time that I recognized their nature, because, when I appealed to my recollections, I remembered a certain number of instances which I had met with, but without understanding them. How often, gentlemen, has it not happened that very learned, intelligent, and attentive physicians have seen, without discerning them, disorders which another more careful and a better observer, perhaps more fortunate also, and better served by circumstances, has discovered and recognized afterwards! How many phenomena are there which we vainly try to interpret, until a day comes when we are more enlightened, and perhaps, also, are better inspired, and we discover their significance! Thus, in the present instance, this was the first time that I understood a fact which I had until then misunderstood, and that I recognized asthma under this strange form which I had not yet known how to diagnose.

I know a magistrate whose wife and nieces were the strangest type of the nervous temperament. His daughter, who was subject to catarrhal affections, went to Nice to spend the winter. In the month of May, she became affected with a catarrh of such violence that her friends got alarmed and brought her back to Paris as soon as she was able to bear the fatigues of the journey. On her arrival, she was seized in the same way, and Dr. Blache and I were sent for. We found her in a state of extreme dyspnœa, and we thought that asphyxia was imminent. But remembering the case which I related to you just now, and taking into account the hereditary history of the child, I was not frightened by this apparently very grave condition, for I foresaw that this violent conflagration would be soon extinguished. We prescribed stramonium fumigations, and, in order to calm the parents' anxiety, added a mixture the effects of which were to be insignificant. Our prognosis proved accurate. Two hours after the stramonium had been used, the symptoms disappeared. The next day, the patient was well, and when we called, the friends received us with marks of gratitude and joy, doubtless ascribing to our mixture the credit of the cure. Since then, this young lady has been seized with similar symptoms several times, and they have always been quieted by fumigations with stramonium.

Taught by these cases, and my attention once aroused on this point, I never again mistook this form of asthma whenever I happened to meet it, and I have often seen it, at least in proportion to the rarity of the complaint in children. Yet few years have passed by in which I did not see one or two cases of the kind.

In the above instances, the course of the symptoms was very rapid; but you will find, in general, that they are less intense, and that they then continue for seven, eight, ten, or twelve days, especially if they are not combated in time, or actively enough. Even then, under the catarrh which overlies the nervous element so as to mask it, the essence of the disease is always the same, and its nature has not changed.

This is so true that, if you adopt in time measures capable of removing the spasmodic element, the disease yields at once and more easily than a genuine pulmonary catarrh, even if the catarrhal symptoms were more intense and apparently more formidable in the former than in the latter.

Doubtless, when the catarrhal element has been of longer duration, it is more difficult to detect the asthma, although it is even then characterized by peculiar symptoms running a strange course. There are, on the one hand, paroxysms of oppression at the chest, of suffocation, recurring intermittingly, especially during the night, and often persisting, even after the catarrh has yielded, with a violence which is not in accordance with the improvement in the inflammatory phenomena. On the other hand, the general disturbances and febrile reaction which accompany this catarrh are slightly marked, and by no means proportionate to the severity of the local manifestations.

Lastly the paroxysms, however fearful they may be, terminate, in general, with surprising rapidity, although they recur, it is true, at more or less distant intervals. They yield to methods of treatment which are sometimes most insignificant, and it is in such cases that homœopathic practitioners obtain the marvellous good results of which they assume the credit. In very many cases, however, very active treatment is necessary in order completely to subdue the disease. Ipecacuanha, given at the onset in emetic doses, has then been in my hands of extraordinary and perfectly unexpected service, in adults as well as in children. Belladonna or atropia, followed on the ensuing days by the administration of spirits of turpentine, according to rules which I will lay down by and by, has been equally successful.

When the catarrhal element predominates, and when, as is pretty common, a slight cold caught on exposure has been the starting-point of the fit of asthma, one might be tempted to ascribe the difficulty of breathing and other phenomena to the bronchial inflammation entirely; but this would be a grave error. Without anticipating now what I shall have to tell you at length when I come to discuss the nature of asthma, I will repeat the statement which I made just now, namely, that the spasmodic element constitutes the essence of the disease. This is so little dependent upon the inflammatory catarrhal element that the same individual who may have had an attack on the occasion of a slight cold, frequently has not the slightest fit of asthma if he happen to have a severe attack of bronchitis, of capillary bronchitis, or even pneumonia.

An old friend and a patient of mine, a rich capitalist, has been subject to fearful fits of asthma since the age of twenty-five. They were so continuous and so violent in 1831 that for seven months he was unable to sleep in a bed, and was obliged to sleep standing, resting the whole night against the mantel-piece in his room. In 1840, on coming out of a theatre, he caught a cold, and had an attack of broncho-pneumonia, of a very serious character, which for a while put his life in danger. During the course of this complaint, he never had a single paroxysm of orthopnœa. Although he cannot even now sleep in a bed unless the mattresses be arranged so as to form a kind of arm-chair, he then slept flat on his back during the whole of his inflammatory attack. His colds have now peculiar characters, and make him very ill, but he never has a fit of asthma while they last.

In such cases, therefore, bronchitis plays a part in the development of asthma, but it only acts as an *exciting* cause, which finds the system in special conditions without which it could not have produced the same effects, and the latter (and this is the point on which I am anxious to lay most stress) are by no means proportionate to the former.

According to its *exciting causes*, asthma has its individualities and its fancies, as well as its peculiar modes of manifesting itself.

In the majority of cases, it comes on without any appreciable cause; in others, which are not very uncommon, the attacks are brought on by perfectly determinate causes, which vary indefinitely in different individuals, but are nearly always the same in the same individual, although their singular influence cannot be explained.

Allow me to give a few instances in illustration.

The lady, whom I mentioned just now *apropos* of coryza, told me that her mother has been asthmatic, and that she herself had been so since the age of ten, and that she never could be in a room where stalks of Indian corn were shaken without being immediately seized with a fit. She had been entirely free from asthma for five or six years, when, at the end of the year 1862, she had an attack which lasted a month, and was this time again brought on by the same cause. Whilst at Bagnères-de-Luchon, she had been suddenly seized with asthma in her bedroom, where a paillasse made of the leaves of Indian corn, on her children's bed, was being shaken.

A case was recently mentioned to me of an individual who could not pass the shop of a ropemaker without being at once seized with a fit of asthma; either the smell, or, what appears more probable to me, the dust from the flax brought on the attacks.

The worst fit of asthma which I ever had myself came on under the following circumstances.

I suspected my coachman of dishonesty, and in order to assure myself of this, I went upstairs to the loft one day, and had the oats measured in my presence. Whilst this was being done, I was all at once seized with a fit of dyspnœa and oppression at the chest so great that I had scarcely the strength to get back to my apartment; my eyeballs protruded out of their sockets, and my pale and turgid face expressed the deepest anxiety. I had only time to pull my tie off, and to rush to the window, which I opened in search of fresh air. I am not an habitual smoker, but I then had a cigar, and took a few puffs; in eight or ten minutes the paroxysm was over.

Now what had caused this fit? Doubtless it was the dust from the oats, which were being measured, that penetrated into my bronchi. But it was unquestionable also that this dust was not enough to bring on of itself such an extraordinary attack, or the cause at least was quite out of proportion to the effect produced. I have a hundred times in the streets, or on the boulevards of Paris, or on highroads, been exposed to an atmosphere of dust considerably thicker than the one which I had then breathed for a very short time, and yet I had never felt anything approaching to this. There must, therefore, have been something special in the cause, and it had besides acted on me whilst I was in a peculiar state. My nervous system was shaken from the influence of mental emotion caused by the idea of a theft, however trifling, committed by one of my servants, and a cause, very slight in itself, had acted on my nerves with extreme intensity.

You will find in books analogous cases.

Cullen's annotator reports that he knew a stout robust man who was seized with asthma whenever rice was thrashed in the neighborhood of his house.

Some of you may recollect a woman who was at No. 6, in St. Bernard Ward, and who had been admitted on account of rheumatic pains. She was forty-three years of age, and remarkably stout, and there was in her history, with regard to the present point, a peculiarity which struck you. Her father was living, and enjoying good health; her mother had died of dropsy, which, from her account, must have been due to a cardiac affection; she herself had enjoyed excellent health until the age of twenty-three. She married at that time, and became subject to asthma, which recurred in paroxysms at variable intervals for the space of two years, and disappeared entirely after she began to nurse her children. The fits used to come on regularly about ten or eleven o'clock at night, lasted the whole of the night and left her in a state of uneasiness and oppression, which continued till noon; from that time she was free, and went about her usual occupations. The peculiarity in the case, which you may recollect was this: whenever she happened to be in her bedroom when her feather bed was being shaken, she had a fit instantly, and she was never so bad as at such times. This cause of asthma has been mentioned to me in several instances.

The following are no less curious cases.

A chemist at Tours, who was slightly asthmatic, had a fit whenever powdered ipecacuanha was dispensed in his shop. It was not only when the root of this drug was powdered, the mere weighing of the powder sufficed to bring on a fearful paroxysm of dyspnœa which lasted an hour. Whenever ipecacuanha had to be dispensed, therefore, he was informed of it, and withdrew to his own apartments. No other powder, no other kind of dust, made him suffer in the same way.

I knew another chemist, of St.-Germain-en-Laye, who was all his life subject to asthma, which recurred under precisely the same circumstances as the above.

Dr. Massina published his case in the "Gazette des Hôpitaux."

The singular effect of ipecacuanha powder was noticed and mentioned long ago. Cullen relates that the wife of an apothecary was seized with asthma whenever ipecacuanha root was powdered in her husband's surgery, even if she happened to be at the time in the innermost part of the house. Murray, if I remember aright, has recorded a similar case in his "Apparatus medicaminum."

A chemist of the Chaussée-d'Antin stated some time ago that when linseed or scammony, as well as ipecacuanha root, were being powdered in his laboratory, he had a violent fit of asthma, which invariably commenced with coryza.

It is not only when they are in a state of powder that certain substances produce these curious effects; their smell alone is sometimes sufficient.

Floyer* cites the case of a lady whose paroxysms were brought on by the least scent.

I have myself had fits of asthma if I remained a few minutes in a room where there was a bouquet of violets; and I know other people who are affected in the same way by the smell of other flowers.

Other cases might be, doubtless, added to these, if one took the trouble of looking out for them; but those which I have mentioned are sufficient to give you an idea of the variety, and especially of the curious nature, of the exciting causes of asthma.

The influence exerted on the development of asthma by *atmospheric conditions*, by *climate*, *seasons*, *temperature*, &c., are no less interesting to know, and no less singular. Two years ago, a young man from Saint-Omer came to consult me. He was subject to frequent fits of asthma, and took advantage of a respite to come up to town. As soon as he got to Paris, he felt markedly better, his attacks became less violent, and after two or three days, he was nearly free from them. He seemed to me to have recovered with too marvellous a rapidity not to ascribe it to some special influence, perhaps to a change of air, and my suspicions soon turned out to be correct. The patient remained in town for three weeks, during which time he had not a single paroxysm. At last he came and took leave of me, informing me that he was going to Versailles. This trip was the test which I wanted to confirm my suspicions or not. The very first night which he spent at Versailles, at the very gates of Paris, he had a most fearful attack; he did not feel as well as before on the following morning, and in the evening of that day, he had another fit. On the second day, he started for Saint-Omer taking Paris on the way.

I had foreseen that the trip to Versailles was the test which I wanted from what the patient had himself told me. He became subject to asthma when he was nineteen years old; he was then residing in his native town; two years afterwards, he had gone to London with his father on business, and from that time, in spite of the London fogs, which are perhaps abused to an exaggerated degree, he had never felt anything of his complaint. Yet during the two years that he spent in England, he had led a young man's life, combining work and pleasure, exposing himself carelessly to inclemencies of weather, and to the habitual causes of catarrh. Although he had caught colds during that time, he had never suffered from asthma, and his colds had even got well more quickly than when he was in France. On his return to Saint-Omer, he was immediately seized in the same way as before, and after suffering for two years, he decided on coming and consulting me. I recommended an active treatment, and sent him back to his native town. A few months afterwards, he wrote to say that he was no better. I advised him to return to Paris, but his friends answered that it was perfectly impossible he could travel as he was so ill; I still insisted on his leaving Saint-Omer. He followed my advice, and was carried to the station; before he reached Paris, his oppression had already diminished to a marked degree, and a few days afterwards, as had happened on his first visit to the capital, he was again perfectly well. I was now sufficiently enlightened as to the course to pursue, and it was useless to make a third experiment. I therefore advised him to leave Saint-Omer, and to take up his residence in London. I must add, however, that he did not follow my advice, and that, when I saw him again in 1863, he told me that he had continued to live in his native place, and that he had been perfectly well for the last five years.

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* Floyer, Traité de l'Asthme.

A barrister, an old friend of mine, usually spends three or four months every year on his estates in Calvados. He enjoys excellent health in Paris, but no sooner goes to his estates than he has there fits of asthma, coming on usually about ten or eleven o'clock at night. His dyspnœa is so great that he is obliged to stay at his window until morning, in spite of the cold which begins to be felt during the autumn months. He is free from it in the morning, and can during the day attend to business.

I had under my care two brothers, twins, so exactly like one another that I could not tell one from the other unless I saw them side by side. This physical likeness went further, for they had, if I may be allowed to say so, a still more remarkable pathological likeness. Thus I was attending one of them at the Néothermes, in Paris, on account of an attack of rheumatic ophthalmia, and he said to me, "At this time, my brother must be suffering from an attack of ophthalmia like myself." As I had expressed doubt at this, he showed me a few days afterwards a letter which he had just received from his brother, who was then in Vienna, and in which the latter wrote, "I am suffering from ophthalmia, you must likewise." However singular this may appear, it is a fact, which has not been related to me, but what I have seen; and I have met with other analogous instances in practice. Now, these twin brothers were both asthmatic, and that to a fearful degree. They were born at Marseilles, but they could never stay in that town, where business often called them, without being seized with asthma; they never suffered from it in Paris. Furthermore, they had merely to go over from Marseilles to Toulon, to get rid of their asthma. As they were constantly travelling from one country to another, on business, they had noticed that certain localities were fatal to them, while in others they were free from all difficulty of breathing.

This, gentlemen, is a pretty general law, and it was necessary that you should know these facts. They will be of great use to me when I come to speak of the nature of asthma.

Dr. G. Vidal communicated to me a case which fell under his own observation, and may as well be mentioned here. He knows a merchant sea-captain who has been a sufferer from asthma for many years. Whenever he goes to Peru for guano, his fits become less violent, and cease entirely as soon as he gets to the Chincha Islands; but his complaint, from which he does not suffer at all, and which he might consider radically cured, during the voyage from America to France, returns as soon as he has left his ship, and he no longer breathes an atmosphere charged with guano exhalations. This case is only in a certain measure analogous to the preceding, because it is less explicable by a change of climate than by another influence. For you know what guano is, and what a penetrating ammoniacal smell it gives off. When I come to speak of treatment, I will tell you what part ammonia sometimes plays in calming paroxysms of asthma.

There is known in England, under the name of *hay fever*, an affection which is to a certain extent a variety of the disease of which I am now speaking. About the end of May, and during the month of June, and even at a more advanced period of the summer, some individuals are suddenly attacked with a coryza, accompanied with violent sneezing, and then with cough and oppression at the chest, especially during the night. This curious disorder lasts sometimes with a distressing obstinacy, until the patient changes his residence, when it ceases as rapidly as it came on. I confess that I have seen several cases of hay fever, and that I have never been able to distinguish it from asthma with periodic recurrences, these coming on much more frequently in summer than in winter. I question how far emanations from freshly cut hay has any share in the production of the symptoms of hay fever, and whether the influence of the season is not a much more potent one.

For asthma is a *summer complaint*, in this sense, that sufferers from it are much more frequently subject to it in the warmer portion of the year, from May to November, than in the colder, from November to May.

Asthma, again, is more common in equatorial regions than in temperate zones or in cold climates. This fact is all the more remarkable that everybody is aware how relatively rare thoracic complaints are in hot climates, where diseases of the liver and of the digestive organs predominate.

Let us inquire into what passes before our eyes with regard to the influence of temperature.

Whilst an individual liable to catarrhs dreads cold, which easily brings on his attacks, and takes good care to clothe himself warmly; whilst in winter he goes rarely out of doors, and remains by the fireside, an asthmatic person, on the contrary, likes the open air, detests small rooms, and finds that low ceilings seem to press on his chest, as it were. However wealthy he may be, you will find him generally in a room without curtains, or with curtains of light material only; heavy wool or silk hangings give him a sensation of choking and of oppression; he must often have the windows open in the depth of winter as if it were summer; in a word, he wants a considerable quantity of air. Whether this want be real, or whether it be the patient's fancy, a kind of mania, if you prefer, you will notice it, and I wished to point it out to you.

Now that I have briefly spoken of some of the circumstances under the influence of which fits of asthma arise, I will review the opinions which have passed and still pass current in the profession on the nature of this singular complaint. I will speak of the theories propounded by my esteemed colleagues, Drs. Rostan, Louis, and Beau. I will try to discuss them, and will tell you at the same time what my own views are, and how I interpret the facts.

Professor Rostan admits now the existence of idiopathic nervous asthma, but he did not always do so. There was a time when he did not believe in this curious neurosis of the respiratory organs, and when he regarded it as being always a symptom of some *disease of the heart or great vessels*. Influenced by the remarkable investigations which he had made on asthma in the aged, when he was physician to the Salpêtrière, he made no difference between asthma and dyspnœa. He regarded those words as synonymous, but I am far from doing so. Asthma is, in my opinion, a special and complete disorder, a manifestation, a peculiar form of a general complaint, having very variable local expressions, sometimes giving rise to paroxysms of dyspnœa, of oppression at the chest, to a curious kind of coryza, and to peculiar catarrhal attacks, which, as I took care to tell you, may constitute the whole paroxysm; but at other times, also, manifesting itself by attacks of articular or of wandering gout, by fits of the gravel, by rheumatism, or by hæmorrhoidal affections. Asthma does not consist in oppression at the chest, because we should have to give that name not only to the dyspnœa, which is a symptom of diseases of the heart and of the great vessels, but also to the difficulty of breathing, which is so great, and which increases to suffocation in cases of œdema of the glottis, of croup, of tubercular disease, or of albuminuria. This confusion is avoided by all: there is an immense difference between dyspnœa and asthma. Although asthma is a dyspnœa of special form and nature, every paroxysm of dyspnœa is not asthma. Have you ever seen paroxysms of dyspnœa occurring in an individual suffering from disease of the heart diminish on the patient taking exercise? Do you not see the reverse every day? You may, at will, as it were, bring on a fit of asthma, or, to speak more correctly, a paroxysm of dyspnœa, in an individual suffering from an affection of the heart which is, in the least, serious. A brisker walk than usual, going up a staircase, are sufficient to bring on more or less difficulty of breathing, which is in some cases so great as to give rise to a sensation of choking. I must add, however, that these attacks of symptomatic asthma may also come on independently of such exciting causes as the above; in some cases they occur under the influence of a somewhat keen mental emotion, and in others they appear apart from all appreciable causes.

Recall to mind the case of that woman who died a few days ago in one of my wards of hypertrophy of the heart complicating an aneurism of the arch of the aorta. You saw her on several occasions in fearful paroxysms of dyspnœa which had come on suddenly, and which towards the last recurred frequently night and day, and without any exciting cause.

If symptomatic dyspnœa may show itself, like idiopathic asthma, independently of all appreciable causes, it is important, in order to distinguish them one from the other, that their course should be investigated.

A fit of asthma runs a course analogous to a paroxysm of fever, that is to say, it begins with a certain degree of slowness, although in some cases its access is somewhat sudden; it increases by degrees to its maximum, like all neuroses, and then decreases in the same way, until it ceases ultimately, leaving the person in perfect health, for a more or less prolonged period, until a fresh attack supervenes. This is surely not the course and aspect of a dyspnœa which is dependent upon disease of the heart. The access of this latter is generally sudden, and it never ceases so completely and so thoroughly as the feeling of oppression of asthma. It is always imminent, and does not leave the individual after a fit in a condition of perfect health, like that of the asthmatic subject, who, when his fit is once over, is not exposed to a return of it in consequence of the slightest emotion, or of a little more active exercise than usual. Until a fresh attack, which often supervenes without his being able to account for it, interferes with him, he will resume his usual mode of life and occupation, and be as free in his movements as though he were not ill. Should he, however, have secondary pulmonary emphysema, he will suffer from habitual oppression, which greatly differs from fits of asthma. An individual laboring under heart disease will always be exposed to the risk of a fresh attack under the influence of the slightest causes. There is no doubt, and you should be aware of the fact, that paroxysms of true asthma may complicate diseases of the heart and lungs.

Let us inquire into what happens in such cases, and allow me to enter into more general considerations, to which I shall have occasion to revert more than once.

A woman, say, has cancer of the uterus, and complains of pain in the loins, and in the hypogastrium, which increase at the menstrual periods, during digestion, or when she goes to stool. Another woman similarly affected has no pain at all, while a third has uterine or sciatic neuralgia recurring daily, exactly at the same hour, with such periodic regularity that she can predict their return within a few minutes. In the case of two ladies whom I attended with Récamier and with my friend, Dr. Lasègue, respectively, these paroxysms of pain lasted five or six hours. In one of them, they continued for several years, and were of an excruciating character. When in pain, the patient rolled about on the floor. In the interval between the attacks, she had only a sensation of heat in the affected side. In these various instances, whether the pain was permanent or not, or was intermittent, the lesion was always the same; when it was intermittent, a neuralgic disorder was superadded to it; the cancer became complicated with the painful neurosis, the existence of which it does not exclude.

In the same manner, the existence of a disease of the heart does not exclude the possibility of asthma. Some persons may be affected with most serious complaints of the central organ of circulation, without suffering from proportionately grave symptoms, whilst others, with lesions that are much less marked than those of the former, are distressed by fearful symptoms. In other cases, again, a neurosis may be superadded to an organic affection. In a word, each person, if I may be allowed the expression, has his own way of carrying his complaint. The system of this one will seem to be indifferent to the lesion, the irritability of the nervous system of that other will show itself by phenomena recurring in paroxysms, and of a peculiar character, according to the nature of each individual. These are facts which one must be aware of, and you may conceive how important it is in practice to know how to distinguish a nervous from the organic element which it complicates.

What I have said of asthma and neuralgia accompanying uterine affections is also applicable to other pathological conditions.

The patient of whom I spoke just now, and who had an aneurism of the aorta, complicated with hypertrophy of the heart, had exhibited the most characteristic symptoms of angina pectoris. Now, what is *angina pectoris?* In a great many, in most cases, in fact, it is a neuralgia dependent upon an affection of the heart and great vessels, as in this woman; but it sometimes is a neurosis perfectly independent of all organic lesion of the central organ of circulation, and even of all appreciable organic lesion. It is a true epileptiform neuralgia, it is a something analogous to epileptic vertigo, and is a mode of this dreadful form of epilepsy of which I have already treated at great length. Like epilepsy, it comes on suddenly, runs a rapid course, and ceases suddenly, and it is not very uncommon for persons who have in former years had attacks of angina pectoris to become subsequently subject to epileptic seizures.

Neuroses may, therefore, be superadded to organic diseases, but they remain independent of them, and these only serve to determine their development. They are not dependent upon them, since the organic lesion does not generally accompany them, and cannot consequently be regarded as the essential condition in the production of the nervous elements of which we speak. In the case of asthma, when it comes on in individuals suffering from diseases of the heart or lungs, it is from the organic lesion determining its manifestation. I would not yet leave on your minds an idea which I do not hold, but which I might seem to have, from what I have just stated. In asthma, the lesion may not be such as to be appreciable to an anatomist; but there is not the less a modification in the condition of the tissues, whether this modification be seated in the cerebro-spinal axis, or primarily in the respiratory apparatus, which does not perhaps alter its structure any more than an overcharge of electricity alters the glass or metallic layer of a Leyden jar.

Dr. Rostan asserts also that asthma may be due to *pulmonary emphysema*, and Dr. Louis holds the same opinion. This view is more specious than the other. For emphysema is nearly always present in asthmatic individuals, and from this the inference has been drawn that this organic lesion was the cause of the complaint, but in this as in the former instance, dyspnœa has been confounded with asthma. When cases of idiopathic nervous asthma are shown to physicians who advocate this view, they diagnose emphysema, the existence of which is often revealed by auscultation and percussion. It would be easy, however, to show them instances in which the nervous disorder does not in the least coexist with emphysema of the lungs. Thus, the patient at No. 10, in St. Agnes Ward, who has been asthmatic for many years, has, it is true, both emphysema and bronchial catarrh, but this is not the case with the woman at No. 6, in St. Bernard Ward. She has none of the signs of emphysema, as many of you have been able to ascertain like myself, and all over her chest vesicular breathing may be heard of normal character.

The facts which Dr. Louis adduces in support of his views have been accurately observed, no doubt, but their importance has been exaggerated; and I will explain to you how this learned physician has been led to adopt the conclusions which he has laid down.

What are the conditions which give rise to emphysema? Is it a primary or a secondary affection? For my part, I cannot conceive it to be a primary complaint, and in order to show you how it is an effect and not a cause of asthma, I must enter into some details regarding the mechanism of its production. But, in the first place, what is the mechanism of cough? The glottis closes, after a more or less deep inspiration, and the expiratory muscles contract in order to expel from the bronchial tubes the air or mucus, the blood or pus, which they may contain. It is only after efforts which are often violent that the expiratory powers overcome the resistance opposed to them. But what takes place during this contest? Pressure is exercised from within outwards on the bronchial tubes and the pulmonary vesicles. This pressure manifests itself outwardly by turgidity of the vessels of the face and neck, towards which the blood is driven in consequence of the compression of the vascular branches distributed through the lungs. The air which is imprisoned inside the bronchi resists the elasticity of the walls of the air-cells, and when the pressure is sustained and powerfully repeated for a long time, when the resistance from the obstacles to the free exit of the air imprisoned within the chest is too great, the walls of the air-cells get distended, the chest expands, the lung dilates, and emphysema is the consequence. In some cases even the air-cells burst, and interlobular emphysema, into which we need not enter here, is the result.

When this mechanism of the production of vesicular emphysema is taken into consideration, no surprise need be felt at its being found in children who have had severe hooping-cough, and in individuals subject to catarrhs. Now pathological anatomy furnishes us with arguments against Dr. Louis's view, since emphysema is a very common affection, much more common than asthma, and found in the bodies of individuals who often never experienced anything during life like asthma, or, at the outside, who suffered habitually from slight dyspnœa.

Everything, therefore, goes to prove that emphysema has nothing to do with asthma. On the one hand, there is no relation between the organic lesion, which is necessarily persistent, or at least does not disappear within a few hours, and the transitory phenomena which constitute a fit of asthma; on the other hand, these phenomena show themselves without the least sign of emphysema being detectable, while again the latter may be, and is, indeed, often present without the former being ever produced.

Although emphysema is not a cause of asthma, it may yet be an effect of it, and you will see how.

An asthmatic individual inspires more slowly and more deeply than the man who breathes freely, while, instead of expiring passively, as in the physiological condition, in virtue of the elastic force of the lungs alone and of the relaxation of the muscles which contracted during inspiration, he expires actively, and in a more violent manner. In spite of the efforts which accompany expiration, the air is expulsed more slowly than in the normal condition, on account of the obstacle to its passage produced by the spasmodic constriction of the bronchi which it traverses. It is conceivable then, that if asthma has existed for a long time, pulmonary emphysema may result from the efforts at expiring, recurring at each paroxysm, and being frequently also attended with cough, which consists in still more energetic expiratory efforts. According to Dr. Beau,* asthma is the consequence of a *chronic catarrh of the small bronchi*, accompanied by a secretion of sputa having a density and a viscous character which are only met with in this complaint. Dyspnœa arises from the presence, in the ultimate ramifications of the bronchi, of the thick mucus which prevents the exit of the air imprisoned inside the air-cells. Laennec had called attention to this kind of sputa, which he calls *pearly*, in the variety of catarrh which he termed *dry*. The expectoration of an asthmatic subject after a fit consists indeed of globules of mucus of the size of a hemp-seed. It is never mixed with air, is semi-transparent, of a grayish and occasionally blackish color, while sometimes it is neither globular nor dense, and has somewhat the aspect of mother-of-pearl. Dr. Beau, who knew the view held by the illustrious discoverer of mediate auscultation, and who had personally observed cases which seemed to be in accordance with it, grounds on the existence of this peculiar expectoration of asthmatic subjects his opinion that this exceedingly plastic secretion accumulates in the bronchi. We need not, he thinks, be surprised that the patient is oppressed in his breathing, because the products of this secretion act like valves inside the bronchial tubes, just like false membranes in croup, or like foreign bodies which get into the respiratory passages. The rattling sonorous rhonchi which are then heard on ausculting the patient's chest are occasioned by the vibration of the column of air as it passes the mechanical obstacle thrown in its way by this thick mucus.

Like the previous theory which I discussed just now, this one is somewhat specious, although I think I can easily upset it.

Take a case of croup, in which false membranes have formed inside the bronchi. Although the obstacle to the free circulation of air through the lungs be then much greater than in the class of cases described by Dr. Beau, yet the paroxysms of dyspnœa from which the patient suffers have no resemblance whatever to fits of asthma. See again what takes place in the man lying at No. 10, in St. Agnes Ward, who has a chronic catarrh with very profuse muco-purulent bronchial secretion.

Doubtless this muco-pus which he brings up in considerable quantities, filling several spittoons, remains in the bronchi for some time, but although the patient suffers from dyspnœa, this has none of the characters of asthma.

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* Traité clinique et expérimental d'auscultation appliquée à l'étude des maladies du poumon et du cœur. Paris, 1856, p. 156 and following.

Now it may be said that in this case the muco-pus is secreted by the large bronchi, and that there is no obstacle to the passage of air, because the calibre of the tubes is sufficiently large to allow of the free circulation of air in spite of the presence of this mucus. My answer to such an objection is that the secretion, and consequently the accumulation of mucus, takes place in the smallest bronchial tubes as well as in the large ones, and we have a proof of this in the fine mucous bubbling rhonchi which may be heard by applying the ear to the patient's chest. The expectoration is so copious that at a given moment the bronchi in this instance are unquestionably much more completely obliterated than in the case of persons who bring up a few small pearly sputa only. Yet this man, I repeat, has nothing analogous to the paroxysms of dyspnœa which characterize asthma.

Granted that these mucous pearly sputa occasion the difficulty of breathing in a fit of asthma, it will be conceded that this secretion takes some time to form, but a paroxysm of asthma comes on with a rapidity which bears no relation to the presence of this mechanical cause. When we see a fit supervene spontaneously under the influence of mental emotion, or in consequence of the inhalation of a few grains of dust, the nature of which varies according to individuals (ipecacuanha or oats), can we assume that these various causes, which suffice to awaken the nervous susceptibility of the patient, are capable of giving rise to a mucous secretion with the same rapidity? On the other hand, you will frequently hear in asthmatic as in emphysematous subjects loud sonorous mucous rhonchi, apart from any attack of asthma, or again before or after a fit.

There are individuals also who are subject to what Laennec called *acute dry catarrh*, and who bring up pearly sputa, with extreme difficulty, after most violent efforts of coughing. They complain of a sensation of obstruction and of pricking at the aperture of the larynx and all over the chest, but which does not in the least resemble the dyspnœa of asthma.

Lastly, and this is the counterpart of what I have just stated, the catarrh which commonly accompanies asthma may be absent, and there are cases, few in number, it is true, in which no symptom of catarrh is to be seen, and no physical sign to be detected, whether the patient be examined and auscultated in the beginning of, during, or at the close of an attack.

As regards the etiology of asthma, therefore, the catarrhal theory is as inadmissible as that which looks upon the disturbance of the respiration as an exclusive symptom of some disease of the heart or of the great vessels, and as the theory of emphysema. In a therapeutic point of view, these various opinions are not more acceptable. When I come to speak of treatment, I will tell you how inhalation of the fumes of stramonium or of burning nitre-paper sometimes suffices to stop the symptoms at once. Now would this be the case, I ask, if it were true that these phenomena were exclusively dependent upon material lesions and mechanical causes?

According to Dr. Duclos, nearly all asthmatic subjects present the herpetic diathesis. I have myself ascertained this to be a fact in a good many instances, but not in the proportion stated by Dr. Duclos. Now when asthma assumes for a few days the continuous form to which I have called your attention, and which is accompanied by an exaggerated bronchial secretion, Dr. Duclos believes that an eczematous eruption like the one which we so often see on other mucous membranes or on the skin takes place on the pulmonary mucous membrane. That theory explains to a certain extent the strange course of this form of asthma, but it does not more than the others account for the intermittent or remittent character of the dyspnœa, which is there to testify to the existence of a nervous element.

But what is, after all, *the nature of asthma?* When one passes in review the series of facts which I have rapidly and briefly laid before you, he is tempted to compare this complaint to other spasmodic diseases of the respiratory system. Hooping-cough, of which I shall speak in my next lecture, suggests itself from the very first as an analogous disorder. An individual, for instance, gets a bronchial catarrh which, for the space of seven or eight days, has apparently the same characters as the simplest catarrh; then convulsive paroxysms come on which nothing can stop, and which recur every two hours, every hour, and even more frequently; they scarcely last from a minute to a minute and a half. In the intervals between them, the patient has exactly the same sensations as in the simplest cold; his expectoration presents nothing peculiar. The case, therefore, is one of catarrh, but of catarrh to which is superadded a nervous element which will enable you at once to diagnose the form of disease. The catarrhal element is there; occasionally it is alone present, while in rare instances the spasmodic element shows itself exclusively throughout the attack.

The same thing occurs in asthma. Although it is more commonly accompanied by all the symptoms of catarrh, and occasionally of severe catarrh, these are yet absent in a certain number of cases.

It may be justly admitted, therefore, with Willis and Cullen, that asthma is a nervous affection; that the paroxysms of dyspnœa which characterize it are probably the result of a spasmodic constriction of the bronchi, which, by narrowing for a time the calibre of these tubes, prevents the free circulation of air through the lungs, and gives rise to all the phenomena which follow.

The researches of Reisseisen, which have been confirmed by more recent investigations, and in particular by those of Gratiolet, who had the opportunity of studying the anatomy of the lungs in the elephant that died at the Ménagerie, have demonstrated the existence of muscular fibres in bronchial tubes of a smaller diameter than those in which there are no cartilaginous rings. Why should it be denied that these muscular tubes may be the seat of spasms when it is admitted that spasms may occur in other organs having a similar structure? Why should bronchial spasms be denied when no one questions the existence of vesical, intestinal, gastric, or urethral spasms?

If physiology leads one to infer their existence *à priori*, they cannot be denied when pathological cases are studied. The patient has a sensation of constriction within the chest. The energetic action of his inspiratory muscles cannot accomplish the act of breathing. It seems as if there were, and there is evidently, indeed, an obstacle to the entrance of air into the bronchi; because, if you apply your ear to the chest of an asthmatic subject during a fit, you will neither hear the sound of pulmonary expansion nor the bronchial respiratory murmur, which becomes audible after the paroxysm is over. And yet the muscles contract violently enough to create a vacuum inside the chest into which the air does not penetrate; the obstacle to the entrance of air is therefore in the bronchial tubes, not at the larynx, since the air passes through the glottis and traverses the trachea freely. Now, this obstacle which exists in the ramifications of the bronchi is not due to morbid secretions, as I have attempted to show, and must therefore be dependent upon spasmodic contraction of the bronchi themselves.

While admitting the nervous nature of asthma, some physicians have suggested another explanation of the dyspnœa than that of spasm. Thus, Bretonneau thought that the difficulty of breathing was due to violent congestion of the lungs. According to him, something analogous to what takes place in an epileptic seizure, attended with congestion, occurs in asthma. For if in some cases an epileptic aura is merely attended with pain, and consists in an unpleasant sensation which starts from some spot, the thumb, for example, and, ascending rapidly towards the head, is more or less immediately followed by the convulsive attack, in others the aura is accompanied by a congestive process which we can often detect. When the aura begins in the hand, this part swells, and the fingers are tightly squeezed by the rings on them. This lasts for one, two, or three minutes, and then an epileptic fit comes on. This congestion is as essentially nervous in its nature as that which causes flushing of the face during a mental emotion. Bretonneau admitted that a similar congestion occurred in asthma, and that the flow of liquids in the lungs obliterating the pulmonary vesicles and the bronchial ramifications gives rise to dyspnœa, and as a consequence to the mucous secretion which is commonly observed at the close of the paroxysms. You are aware that Cullen also ascribed to dilatation of the pulmonary vessels the production of asthma, but, contrary to what Bretonneau taught, Cullen believed that this vascular dilatation was the cause and not an effect of the spasm.

In spite of all my respect for the views of Bretonneau, who was my first and excellent teacher, I have always opposed this theory. I have never been able to understand the aura epileptica, but I do understand the mechanism of mere spasmodic constriction, and, more than this, I cannot conceive how it could be different.

Thus, gentlemen, asthma is a nervous complaint, a neurosis; and in order better to define the class to which it belongs, I will add, a diathetic neurosis; that is to say, it very rarely happens that it does not depend upon the existence of a diathesis. I will endeavor to prove this by facts, and allow me, therefore, to enter into some details which will not be devoid of interest.

An individual, say, becomes asthmatic when about thirty or forty years old. Until then, he had been entirely free from that complaint, and vain attempts are made to discover an exciting cause; but on inquiring into his previous history, it is made out that the patient exhibited in his youth symptoms of a different kind, which were evidently manifestations of a diathesis. These were eruptions of an herpetic, or, more commonly, of an eczematous nature; or he had rheumatic pains; or again, at a more advanced age, he had either fits of the gout or hæmorrhoids. Indeed, nothing is more common than to find herpetic, rheumatic, gouty, or hæmorrhoidal affections transform themselves into asthma. The fact was pointed out long ago. Truka[*] and Musgrave[†] have both recorded instances of the kind; and in reference to gout, I have known a case in which attacks of arthritis alternated with fits of asthma with great regularity: fits of the gout sometimes followed one another; at other times, paroxysms of asthma; occasionally, an attack of gout came on after one of asthma, or reciprocally, but the patient never had gout and asthma simultaneously.

Thus, eczematous eruptions, rheumatism, gout, and hæmorrhoids, and I may add, gravel, are complaints which may be replaced by asthma, and may replace it in turn: they are different expressions of one and the same diathesis. Hemicrania is another affection which I must not omit.

Many individuals who are subject to periodic headaches, are, or have been, affected with gout, rheumatism, or eczematous eruptions, or are the issue of parents who were so affected; or you may notice the reverse; that is to say, you may see eczematous and hæmorrhoidal affections, attacks of rheumatism or of gout, following on periodic headaches. Among other examples of these diathetic transformations, I will relate to you the following, which was the first case of the kind which attracted my attention at the commencement of my medical career.

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[*] Historia hæmorrhoidum. Windobonæ, 1794.

[†] Traité de la goutte.

I was intimately acquainted with a major of the English army, who for a long time had been subject to headaches that recurred so periodically—every other Wednesday—that he knew within an hour when he was going to be attacked. His seizures were so regular as to their course and duration that, more extraordinary still, he could tell when they would terminate. They indeed lasted a few hours, and then left him in a condition of perfect health. He had become subject to these attacks while staying in the West Indies; since then, they had never missed until the time when I made his acquaintance in Paris. He was quite tired of his complaint, and begged me one day to rid him of it. This was in the year 1824. I did not know then what hemicrania was. I asked the advice of some other practitioners, and prescribed blue pills in large doses. Under the influence of repeated purgation the attacks ceased to be periodical, but his general health was far from improving. Formerly, at the termination of the attacks, he felt perfectly well, and his sensations contrasted singularly with the *malaise* which he felt when the attacks were on the point of coming. In fact, he felt like all those who suffer from a gouty or hæmorrhoidal diathesis, and who feel such relief from the attacks, which are often preceded by an undefined *malaise*, that the seizures seem to be indeed necessary evils.

My patient went to reside at Fontainebleau during the fine weather, and I used to go and spend a few days with him there from time to time. One morning he had me called up in order to show me his foot, in which he had excruciating pain. I noticed swelling and considerable redness of the foot; in fact, he had a fit of well-marked acute gout. I did not at that time know how much one should respect such manifestations, and I was not aware that gout and headache are sister diseases: influenced also (in spite of myself and in spite of the principles which I had been taught when I began to study medicine) by the doctrines of Broussais, which were then in full vogue, I decided on adopting an antiphlogistic treatment. I ordered leeches, and emollient poultices sprinkled with laudanum, to be applied to the painful part. The arthritis yielded, but from that day the patient lost his former good health. He had a second attack, but of indolent, atonic gout, and not only did his general health fail, but even his mental and intellectual condition became deplorably affected. He lost his usual vivacious and merry ways, became dull, cross, and disagreeable; at last, he had an apoplectic stroke, and died two years afterwards of a second attack.

I might relate a great many similar cases, but will confine myself to three more which bear more especially on this point.

On July 15, 1861, a young man aged thirty, and residing habitually in the department of the Côtes-du-Nord, came to consult me in Paris. Dr. Blondeau, who happened to be in my consulting-room at the time saw him with me. He had the appearance of a man enjoying perfect health, and stated that no member of his family had suffered from gout. When a boy, between ten and fifteen years old, he had a moist eruption on his legs, which disappeared somewhat suddenly to show itself again ten years later. But from the age of seventeen to twenty-one, he had been subject to frequent paroxysms of nervous asthma which were so violent as to place him at death's door. He was only relieved by bleeding.

At the age of twenty-one, he had fits of regular gout, and from that time his asthma left him. However, as he was impatient of pain, and was anxious to get rid of it at any price, he had recourse to preparations of colchicum, and those fatal secret remedies, the Syrup of Boubée, Lartigue's pills, and Laville's Liquor, which are efficacious, but dangerous also. He got rid of his gout, but in less than three years, his health had become seriously impaired, and he looked prematurely old. He then went to Tours and consulted Bretonneau, who at once made him stop the dangerous remedies which he had been taking, and advised him to take the Carib ratafia (made of taffia and guaiacum root) and to take a good deal of exercise and live well. Under the influence of this plan of treatment, his acute gout returned and his health with it. Some time afterwards the patient went to Bagnères-de-Luchon, where he drank the waters and remained free from gout for two years and a half. The fits then returned, but were less violent; and when I saw him, he had had none for eighteen months. He complained however of headache, which returned every ten days or fortnight; always beginning in the right temple and stopping at the occipital region on the same side, and lasting about three or four hours. His health was excellent, with the exception of these headaches, which, if I may be allowed the expression, were the small change, as it were, of the fits of regular gout.

On March 2, 1863, I saw in my consulting-room again a man aged thirty-five, who had been subject to asthma since the age of sixteen. His attacks were never less than a fortnight's duration; they came on especially when he stayed in Paris and were very rare if he lived in the country. For the last six months they had grown less severe, and the last attack, which had set in with very moderate violence, had terminated on the third or fourth day in a fit of perfectly regular gout.

In the following case, which was communicated to me by my colleague, Dr. Hérard, it was not gout but rheumatism which was followed by asthma.

A lady about fifty years old had been attacked for the first time when about thirty years of age, by acute articular rheumatism a few weeks after confinement. She recovered, but had a relapse at the end of two months. From that time she became subject for several years to vague, wandering, rheumatic pains in the muscles.

These pains ceased. After this, other neuralgic pains showed themselves, and the patient became at the same time subject to periodic headaches, from which she had never suffered previously. She came under Dr. Hérard's care in 1858. She was then troubled with a spasmodic cough, which recurred regularly every night at the same hour. In the course of that winter, she had facial neuralgia, and the skin of her neck became covered with a dartrous eruption, of a papulo-vesicular character, which lasted a short time only.

She went through the next winter without illness, but in 1860 she had fits of well-marked nervous asthma, which came on in the evening and during the night. Dr. Hérard noted a peculiar circumstance, which has great analogies with what I told you of the influence of climates, namely, that the paroxysms of asthma came on especially when this lady stayed in a certain quarter of Paris. She lived in Cirque Street, and she had generally an attack when she went to spend an evening in the neighborhood at her brother's, in Ville l'Evêque Street. These paroxysms, which remained during fifteen or twenty days, never returned again, and were never accompanied by symptoms of catarrh.

These facts might, perhaps, be explained in the following manner. When persons suffering from gout or from piles do not in the proper season exhibit the usual manifestations of their diathesis (fits of articular gout and bleeding piles), they become, in a great many cases, and to a very high degree, subject to nervous symptoms, such as spasms of the stomach or the intestines, a condition of general *malaise*, which expresses itself by moroseness, sadness, or some change or other in their temper. These phenomena, it is true, often also precede, although to a less degree, the regular attacks. Now, it may be questioned whether asthma is something else than a form of these spasmodic affections which then attack the lungs.

This is the theory propounded by Dr. Duclos; and the following case, which resembles the preceding ones, supports it, in a certain measure.

I had under my care, a lady about thirty years of age, whose fits of asthma coincided with an eruption of urticaria. They lasted two months, and when the urticaria disappeared, the feeling of oppression at the chest increased invariably; so that it might justly be supposed that the asthma was caused by an exanthematous eruption in the bronchi.

There is another diathesis which differs from those to which I have just called your attention, and of which asthma may also be a manifestation, namely, the tubercular diathesis. Of the numerous instances of the kind which have fallen under my observation, I select the following, one of the most remarkable of them, the case of a lady, aged seventy, who enjoys very good health, with the exception of occasional fits of asthma. Her mother died of phthisis, and she has lost two daughters, one of whom was carried off by cerebral fever, and the other died of phthisis.

Tubercular individuals may, therefore, give birth to asthmatic children, and, on the other hand, asthmatic subjects may have tubercular children. It is certainly a very remarkable fact that asthma, which seems to be such a trifling affection, as regards the organic lesion which accompanies it, should apparently be, in some cases, a manifestation of a diathetic disease so marked in its localization as tuberculosis is. Bear these cases in mind, gentlemen, for they have reference to a great question, namely, the transformation of morbid affections into one another, a vast subject, which we cannot enter into here, and which might be treated in an important chapter of general pathology. In reference to the point which we have just been considering, remember that eczematous and rheumatic affections, gout, gravel, hæmorrhoids, periodic headaches, and asthma, which are varying expressions of one and the same diathesis, may replace one another. In proportion as you make further progress in the practice of your profession, you will but too often have the opportunity of verifying the accuracy of this statement.

Lastly, like all diathetic diseases, asthma is directly transmitted from parent to offspring.

The case of a man who has been for a long time an in-patient here, is sufficiently typical to serve as an illustration of this point.

He is thirty-one years of age, and has been subject to fits of asthma since he was thirteen. He had been perfectly free from them until then. He associated with other children of his own age, joined in their games, running and going through the same exercises as the others, without feeling the least distress, when he had a first attack of asthma, without his being able to assign any cause for it. The fit came on at three o'clock in the afternoon, and lasted from four to five days. From his description, it assumed the catarrhal form, and was so severe as to alarm his friends and the medical men who were consulted.

Five years afterwards, the symptoms became more regular; they no longer came on during the day, but always after twelve at night—about one or two o'clock in the morning. You already find here a fact on which I have laid much stress, namely, the period of day at which the paroxysms occur. The patient stated of his own accord, without being led by me, that these attacks were commonly of very great violence, if, previously to his getting into bed, he shook his paillasse, and he ascribed the intensity of the paroxysm to the presence of the dust which he raised about him in the room. There was generally but one paroxysm at each attack, and he was free for six months. At the present date, they return every six weeks, and last three days, that is to say, during the three days he has constantly a sensation of constriction and distress about his chest, which prevents him from working; this symptom becomes more intense at night, and generally diminishes in the early morning, although it occasionally increases then.

The patient mentioned also a circumstance to which I am desirous of calling your attention, namely, that his expectoration presents perfectly different characters before and after a paroxysm. During the fit itself he does not expectorate at all; before it, he brings up small, thick, globular sputa, which he compares to the germ of a hen's egg. You will recognize pearly sputa by his description. After the attack, his expectoration becomes mucous and purulent; you have yourself seen it in the spittoon, and it differed in no respect whatever from that of an individual suffering from the most genuine catarrh. This case, in which pearly sputa are brought up before a fit, where there is consequently no distress of breathing, no oppression at the chest, no asthma proper while the dyspnœa is unattended with expectoration, and the catarrhal expectoration occurs after the difficulty of breathing is over—this case, I say, is another instance which tells against Dr. Beau's theory.

This case illustrates also what I have told you of the various forms of the disease in infancy and adult age, and of diathetic transformations. The patient's mother was gouty, his father epileptic, and he was himself subject for some time to periodic headaches.

In the intervals between the paroxysms, he could go through the most violent gymnastic exercises without feeling the least distress of breathing. On several occasions he betted with his friends that he would go from Paris to the Place d'Armes at Versailles (a distance of eighteen kilometres, between thirteen and fourteen miles), and keep up with the Versailles omnibus, which, as you know, goes at a pretty quick pace.

He can run therefore for about an hour and three-quarters, walk pretty briskly up the rather steep ascent of Sévres and of Chaville, and reach the end of his journey without feeling more out of breath than a man might be who had just walked in measured step for the distance of half a kilometre. You may remember that during his fits he had all the physical signs of vesicular emphysema of the lungs: the inspiratory murmur was almost *nil;* expiration was forced and longer than inspiration; there were sonorous rhonchi and exaggerated resonance of the chest. When he had been free from asthma for a few days, this inspiration became longer, full, and easy; the vesicular murmur perfectly normal; expiration less prolonged than inspiration, performed without effort, and the thoracic resonance less than during the attacks. (In children and in adults suffering from hooping-cough, you will likewise frequently find signs of vesicular emphysema, which will rapidly disappear as soon as the neurosis itself shall have passed.)

Lastly, the history of this patient is again complete with regard to the treatment of the asthma. When a paroxysm is coming on the patient gets out of bed, heats some water and takes a foot bath, which generally relieves him; at other times, he is obliged to stand at an open window, however bad the weather may be, so as to allay his sense of anxiety by breathing the fresh night air. If asthma were a catarrh, would such a plan of treatment succeed? Stramonium gave him little relief, and ammonia, of which I shall speak presently, caused him much inconvenience. He derived, on the contrary, much benefit from arsenic. In this case, you have an illustration of the therapeutic fancies of asthma, as in others you had of its pathological caprices.

Indeed, like all neuroses, asthma often yields to measures which differ widely according to individuals; and experience alone teaches the patient and the practitioner what those measures are. I have already stated that asthmatic subjects commonly sought after fresh air; others, on the contrary, can find relief in their paroxysms only by standing with their back to a roaring fire. The last patient I alluded to, derived benefit from a hot foot-bath. If I were to enumerate all the plans, some more curious than others, which certain persons have recourse to, the list would be long before it came to an end. The brother of the Chancellor of the late Chamber of Peers had from four to six Carcel lamps lighted in his apartment whenever he had a fit of asthma, and felt immediately relieved after this. Another patient, whose attacks occurred during the day, got on horseback, and could only calm the paroxysm by galloping in the teeth of the wind.

These are surely singular and exceptional facts, but it was important that they should be mentioned, because they afford fresh proofs of the essentially nervous nature of the complaint.

I now pass on to the subject of *treatment*.

In certain countries in which asthma is a common disorder, its treatment was formerly empirical. In the East Indies, the popular remedy consisted in smoking the leaves of the *datura metel*. Dr. Anderson, who was in practice at Madras, recommended, and gave a quantity of them to an English officer who brought them to Europe in 1802, and made a present of them to Dr. Sims, of Edinburgh. The latter, on finding that the smoking of these leaves was followed by good results, tried to substitute for them *datura stramonium*. The trials answered, and stramonium has now become a popular remedy for asthma.

What I shall say of *datura stramonium* is applicable to the other species of datura, the *datura ferox, fastuosa*, or metel, although the first is the one generally used. Of all remedies which have been tried in asthma, stramonium generally answers best. The dried leaves of the plant are smoked either alone or mixed with sage, in a pipe, or rolled up in cigarette papers, or they are burnt in the patient's room. All asthmatic subjects are not however relieved, and habitual tobacco smokers often derive no benefit from it. This is conceivable since tobacco is a solanaceous plant like stramonium, and a man used to tobacco may not be amenable to the stupefying influence of stramonium. I know, however, tobacco-smokers whose paroxysms are calmed by stramonium; so that this would show that the latter has a specific virtue somewhat different from that of nicotine. Although one of these drugs does not, consequently, replace the other completely, there are still individuals who are not habitual smokers, and who obtain relief by smoking tobacco; I do so for my part, and I have already told you that when I had a fit of asthma, I had only to draw a few puffs from a cigar to remove the dyspnœa. Generally speaking, *all solanaceous plants*, stramonium, tobacco, hyoscyamus, and belladonna, are therefore more or less endowed with the same properties; they are all used in the preparation of the Espic cigarettes, which have long enjoyed, in the treatment of idiopathic nervous asthma, and of pulmonary catarrh complicated with nervous symptoms, a reputation which is still trumpeted by newspaper advertisements. They are prepared according to the following formula, which you will find in some of your text-books:

℞. Folii Optimi Belladonnæ,	gr. vj.
" " Hyoscyami,	gr. iij.
" " Stramonii,	gr. iij.
" " Phellandrii Aquatici,	gr. j.
Extracti Opii,	gr. ¼.
Aquæ Destillatæ Laurocerasi,	q. s.

The leaves, after careful drying and removal of their veins, are cut up and thoroughly mixed. The opium is dissolved in the cherry-laurel water, with which the leaves are wetted. The paper used for the cigarettes is also soaked in the solution and then dried.

You may conceive the efficacy and success of such a combination of remedies. Yet, when stramonium and other solanaceous plants are prescribed, the patient should be particularly warned against abusing them, lest he should quickly exhaust their influence. He should have recourse to them only when the attack is violent, and should then smoke two cigarettes during the fit, and not seven, eight, or ten, as many are tempted to do. When the patient cannot smoke, or does not know how, stramonium may be burnt in his room, and thus the air in it charged with the fumes of the drug.

With this plan of treatment, as with all those which are used against nervous disorders, idiosyncrasies should be taken into account. One asthmatic subject, for instance, will be relieved by stramonium, another by belladonna, a third by hyoscyamus, and a fourth by tobacco or a mixture of these various plants. In some cases, as in that of the man whose history I related to you in detail, solanaceous plants cannot be borne, so that other methods of treatment have to be had recourse to, some of which have been justly lauded, as, for example, *arsenical cigarettes*, and the fumes of burning nitre-paper. To prepare arsenical cigarettes *twenty grains of arsenite of potass* are dissolved in *half an ounce of distilled water*, and a sheet of bibulous paper is soaked in this solution until it is entirely taken up. The paper is next dried and divided into twenty equal pieces, which therefore contain about one grain of arsenite each. Each piece of paper is then rolled up into the shape of a cigarette, which the patient smokes after lighting, and, by inspiring slowly, draws in the smoke inside the bronchi: he should take five or six puffs only, once a day.

The nitre cigarettes are prepared in the same way, by soaking some blotting-paper in a nearly saturated solution of nitrate of potass.

If the patient does not know how to smoke, the arsenical or nitre-paper is rolled up into a ball and ignited, and the patient inhales the fumes through a funnel-shaped tube of paper held over the ball.

I have sometimes combined nitre and stramonium, or belladonna fumes, either by rolling leaves of these plants within the paper impregnated with the solution of nitre, or by directly dipping the leaves in a solution of nitre. Of the measures directed against a fit of asthma, there is one which has been, in turn, lauded and rejected too absolutely; for when properly employed it has been of real good service; I allude to the topical application of ammonia to the posterior part of the pharynx.

Ducros, of Sixt, was the first to propose this plan of treatment. He treated all asthmatics by brushing the back of the pharynx with a mixture of equal parts of water and liquid ammonia.

Ducros was a curious individual, and held the queerest medical theories, and he was led to adopt the above plan on account of his singular idea that the back of the pharynx was the centre from which emanated the nervous power, the influence of which he tried to modify. Strange as his starting-point was, he was in some cases successful, and his success in the case of Madame Adélaide d'Orléans, sister of the king Louis Philippe, gave him for a time a very great reputation in Paris. Trials made by other medical men—by Dr. Rayer and by myself—proved the efficacy of the remedy in certain cases: but it occasionally gave rise also to formidable accidents. I must, therefore, while admitting the good effects of this plan of treatment, warn you against the risks attending it.

Two cases among others made a lasting impression on me.

A man, of colossal stature, consulted me one day on account of asthma; he had been sent to me by my friend Dr. A. Lebreton. I tried Ducros's method, but on my first attempt, just as I had passed down to the back of the fauces a brush dipped in a mixture of equal parts of water and liquid ammonia the patient had an awful paroxysm of orthopnœa. He started up on his feet, as if pushed by springs, and rushed to the window in a truly fearful state of suffocation. He thought that he was going to die, and I confess that I thought so too. He grew quieter, however; but neither of us cared to repeat the experiment.

Some time afterwards a lady, whom I have had occasion to see since, consulted me for the same complaint. I again tried the ammonia treatment, but this time I took the greatest possible precautions. Notwithstanding these, I had scarcely touched the pharynx with the brush, than a fit of choking supervened. The result was successful, nevertheless, for the patient was free from asthma for the next two months, which was very unusual with her.

Lastly, the patient in St. Agnes Ward has stated in your presence that he had been treated in that way once, but that he had been seized with such oppression at the chest that he thought he was going to die. From this time, besides, he had fits of asthma every four days, which recurred at the same hour that the topical application of ammonia had been done, whilst, previous to this, his attacks returned every three months only.

Ducros's method, therefore, serves only a few patients, although a great many bear it without any inconvenience. Ducros himself had recourse to it every day, and never, he said, with unpleasant results. The cases which I have just related show, however, that you cannot be too prudent, for one may understand that death might take place in one of these fearful paroxysms. When I have recourse to this method of treatment, I therefore take a precaution which I recommend you to adopt. I first make the patient inhale some ammonia from a bottle, and after this, I apply to the back of the throat, on the first occasion, a solution of one part of liquor ammoniæ to nine of water. The next day I use eight parts of water to one of ammonia; and I diminish the quantity of water by degrees to one-third, until the patient has grown accustomed to it, when I use equal parts of water and liquor ammoniæ.

Another method of applying this treatment consists in placing in the room where the patient is, dishes containing liquor ammoniæ, the vapors of which are diffused in the atmosphere of the room. Dr. Faure's method is in some respects different from the one which I have just described, and you saw me have recourse to it in a patient at No. 22, in St. Agnes Ward.

This man was subject four years ago to fits of asthma which returned every night at the same time, and lasted about two hours. For nearly three years and a half, he had had no attacks, when in the course of the spring of the year 1860, these returned again. He was seized during the night as previously, but the dyspnœa continued during the day, and on examining his chest, I recognized pulmonary emphysema. He never brought up pearly sputa, however, and his expectoration resembled lightly boiled white of egg.

I prescribed for him ammoniacal inhalations, according to Dr. Faure's method, which consists in holding one's mouth at a distance of about a foot over a vase containing a tablespoonful of liquor ammoniæ; the inhalation to be continued for a quarter of an hour, and to be repeated four times in the twenty-four hours. The patient's nostrils had to be stopped up with some cotton-wool, because he could not otherwise bear the smell of the ammonia.

You saw that from the first day the nocturnal paroxysms disappeared, the diurnal dyspnœa ceased almost entirely, and the patient seemed perfectly well after four days of this simple treatment.

It is probably to these vapors, that some asthmatic subjects owe the relief which they experience by merely staying for some time in places where there is a disengagement of ammoniacal gas. I have already mentioned the instance of that merchant sea-captain who was free from asthma so long as he was on board his ship with a cargo of guano, or when staying at the Guano Islands.

Antispasmodics, such as ether, either given with simple syrup or in capsules, are also indicated during the fit.

In some cases, which I have already defined, I have obtained good results from the timely administration of an *emetic*, and ipecacuanha is the remedy to which I then give the preference.

Now that I have described some of the methods of treatment which are useful during a fit of asthma, it remains for me to tell you how to prevent the recurrence of the attacks. Medical interference is unfortunately very often powerless in that regard, for we can better moderate the symptoms of the complaint than cure it radically. The following, however, is a method of treatment which seemed beneficial to me. It consists of a certain number of series:

1. For ten successive days, every month, the patient takes at bedtime first one, then, after three days, two, and for the last four days, four pills like the following:

℞. Extracti Belladonnæ,
Pulveris Radicis Belladonnæ, } gr. $\frac{1}{5}$ āā.
fiat pilula una.

or from one to two, up to four granules of atropine, containing one-fiftieth of a grain each of the drug.

2. During the next ten days, the syrup of turpentine, in doses of a tablespoonful three times a day, or better, three capsules of spirits of turpentine are substituted for the preparations of belladonna.

3. For the last ten days of the month, the patient is made to smoke arsenical cigarettes.

Lastly, to complete the treatment, the patient takes every tenth day, in the morning fasting, a drachm of powdered yellow cinchona bark in a cup of coffee.

The essential part of the treatment consists, I believe, in the internal and prolonged administration of belladonna or atropia, according to the formulæ and to the rules which I have just laid down. It is not necessary that the patient should feel, to a marked degree, the physiological effects of these powerful drugs, but his system should for a long time be kept under their influence. Bark, turpentine, the inhalation of arsenical vapors, are, however, very useful adjuncts.

This method of treatment is far from being infallible, although it has given me, and gives me every day, excellent results. It is successful in some cases, but fails entirely in others, so that other measures have to be adopted.

Dr. Duclos, of Tours, asserts that *flowers of sulphur* are a remedy "of prodigious efficacy" for the prevention of asthma. He prescribes it "in daily doses of from ten to twenty grains, according to the age of the patient, to be taken in one dose in the morning early or just before breakfast. This dose is repeated for five or six months, during twenty days every month, then for a year, eighteen months or two years, for ten days every month." This treatment is, he considers, the most simple and the most easy to carry out that can be imagined. The disease was improved in every case in which he had recourse to it, and he cured a great many individuals.*

Dr. Duclos insists on the necessity of giving flowers of sulphur instead of any other preparation of sulphur. It is not an indifferent matter, in his opinion, as to the choice made; for while he has obtained good results from the flowers of sulphur, he had constantly failed when he used to prescribe sulphuretted waters, like those of Barèges, of Bonnes, and of Cauterets. He remarks, *apropos* of this, and justly too, that among the substances used in materia medica, there are analogues and not succedanea; that sulphuretted waters are not flowers of sulphur, no more than cinchona bark is the sulphate of quinine, or opium, morphia.

I have myself treated asthmas successfully with sulphur; but I cannot share the illusions of my learned colleague of Tours with regard to the infallibility of this remedy in all given cases. Sulphur seems to me to be clearly indicated, and to be of unquestionable service, when the asthma is due to an herpetic diathesis; but if we exaggerate the importance of facts, and draw general conclusions from particular cases, and fancy that the treatment should be always exactly the same in every case, we run the risk of being sorely disappointed.

In those cases in which sulphur is of real benefit, namely, when the neurosis of the respiratory apparatus is dependent on an herpetic diathesis, *arsenic given internally* has been in my hands, and is still, of very great service. No surprise need be felt at this, if the marvellous effects of arsenical preparations in the treatment of herpetic disorders in general, and skin diseases in particular, be taken into account.

Arsenic is not only very efficacious in such cases, but it is also an excellent remedy against asthma in a good many other instances, which have nothing to do with the herpetic diathesis.

The remedy is not a new one besides. I need only mention that Dioscorides administered it in asthma, either mixed with honey or in a mixture containing resin. He used what was in his day called *sandarach*, but is now known as realgar or red sulphuret of arsenic. At a less remote period from us, at the end of the sixteenth century, George Weitth lauded an electuary which contained orpiment (the yellow sulphuret of arsenic), of which he gave a large dose every day to patients suffering from the gravest forms of asthma. The use of arsenic, however, was obstinately opposed by the majority of medical men; the mineral was absolutely rejected from materia medica, and it was thoroughly discredited, when, in our own time, Harles attempted to bring it into favor again. It enjoys now full favor, and deservedly holds an important rank as a therapeutic agent.

In reference to its use in asthma, you have doubtless heard of the arsenic-eaters, or toxicophagi, of various parts of Germany, Lower Austria, and Styria. In those countries the peasants, and even the townspeople, are in the habit of taking several times a week, in the morning fasting, at first a small quantity—about half a grain—of arsenic and by degrees larger doses. By so doing, they hope to make their complexion look fresh, and to grow moderately stout, and also, to become, to use their own expression, more *volatile*, that is to say, to increase their breathing capabilites when walking up hill. When they have to walk a long distance in the mountains, they hold in their mouth a piece of arsenic of about the size of a small lentil, and let it melt by degrees. The results of this practice are really surprising; they can by this means ascend with facility heights which otherwise cause them great fatigue. The men not only eat arsenic themselves, but also habitually mix it with the food of their horses, especially those which work their carts in mountainous districts.

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* Recherches nouvelles sur la nature et le traitement de l'asthme (Bulletin Général de Thérapeutique, Avril 15, 1866, t. lx, p. 290.

Profiting by the example of these people, some medical men asked themselves whether this singular and quite special influence of arsenic on the function of respiration, might not be utilized in the treatment of certain respiratory disorders. Dr. Koepl was one of the first who accordingly, thought of trying the liquor Fowlerii, or arsenicalis, in cases of asthma, and he obtained the good results which he expected in a pretty good number of cases. Others tried the same remedy, and with similar success; and for several years I have myself given arsenic internally in spasmodic asthma with unquestionable success.

I commonly prescribe the *arseniate of soda* in a mixture like the following:

℞. Sodæ arseniatis, gr. j.
Aquæ destillatæ, ℥iij.
Tincturæ cocci, q. s (ad colorandum).

The patient is made to take every day, at the commencement of his two principal meals (a useful precaution in order that the remedy be well borne by some irritable stomachs) a *teaspoonful* of the solution, containing the twenty-fourth part of a grain of arseniate of soda.

I also prescribe *arsenious acid* in pills:

℞. Acidi arseniosi, gr. v
Amyli, gr. c.
Syrupi acaciæ, q. s.
pro pil. centum.

Each pill will contain one-twentieth of a grain of arsenious acid, and is to be taken before each of the two principal meals in the day. When I have to deal with timid individuals, who may dread the idea of taking arsenic internally, I call these *Dioscorides pills.*

According as it is tolerated by the patient, I increase or diminish the dose of the medicine, and I continue it for several months in succession, generally intermitting it for eight or ten days every month.

There is another treatment for the cure of asthma, about which I should say a few words. You have seen me for some time give *iodide of potassium* to two men in St. Agnes Ward who presented perfect types of spasmodic asthma. The history of this method of treatment is rather curious.

Two or three years ago, a French medical paper published an extract from a foreign journal,* in which it was stated that a secret remedy for asthma, of which iodide of potassium was the principal constituent, was being sold at Boston.

The author of the statement, Dr. Horace Green, added that he used the remedy with the greatest success, especially in cases of asthma complicated with bronchitis, and gave the following formula:

℞. Potassii iodidi, gr. xl.
Decocti polygalæ, ℥iij.
Tinct. lobeliæ,
" camphoræ cum opio, } ʒiv āā.

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* Favorite Formulæ of American Practitioners, by Horace Green, of New York, translated into French by M. Noirot (1860) (Schmidt's Jahrbücher der gesammten medicin), Band 114, No. 112.

Two or three tablespoonfuls a day.

About the same time, Dr. Aubrée, now practicing as a pharmaceutist at Burie (Charente-Inférieure), wrote a letter to the Academy claiming the priority of the discovery, and addressed to me another letter, giving me the credit of the method of treatment which he had used for the last fifteen years, or at least part of the credit. I have preserved his letter, of which the following is an extract:

"About fifteen years ago, when I was residing in a small town in the Hérault department (Pézenas), I was consulted by a man from the neighboring village of Velleros, on account of a well-marked neurosis of the respiratory organs. He showed me a prescription by you for tincture of iodine, which was to be rubbed into the axillæ, the pit of the stomach, and the back, and for a mixture of iodide of potassium (one hundred grains to eight ounces of distilled water, sweetened with simple syrup). I repeated the same treatment, and he felt better from the second day. He came back to me rejoicing after a few days, but he was not perfectly well yet. I told him to cease the frictions, and doubled the quantity of iodide of potassium in the mixture. The wheezing in his chest disappeared completely, his breathing recovered its usual rhythm, and from that time he never had another attack of the same complaint."

Encouraged by this case, Dr. Aubrée tried the same remedy in a great many other instances, and gave an *anti-asthmatic elixir*, prepared according to the following formula:

℞. Polygala root,	gr. xl.
Boil in	
Water,	℥iv.
Reduce by boiling to	℥ij.
Filter and add,	
Iodide of potassium,	ʒiv.
Syrup of opium,	℥iv.
Brandy,	℥ij.
Color with,	
Tincture of cochineal,	q. s.
Filter.	

The patient should take three tablespoonfuls of this elixir in the morning fasting, at noon, and in the evening, until the asthma disappears. As some individuals do not bear the remedy well (and this is not surprising, because each dose contains about forty-five grains of iodide of potassium and four-fifths of a grain of extract of opium), Dr. Aubrée makes it an indispensable condition to suck after each tablespoonful a chocolate pastille, which neutralizes the irritating action of the iodide of potassium. He states that he has by this means cured, in the short space of three days, twelve persons.

Since this communication from Dr. Aubrée, and since the secret remedy of certain quack medicines has been made known, I have very frequently given iodide of potassium, but have modified the formula as follows:

The patient takes every day, immediately before dinner, a teaspoonful of a solution of

Iodide of potassium,	℥iij.
Distilled water,	℥vj.

I must say that I have obtained with this remedy successful results, in a great many cases where other methods of treatment had failed. On the other hand, we must not be wilfully blind, for I have known the iodide not only fail but also increase the disease markedly, as it did in the case of the two men it St. Agnes Ward.

One of them was, on the contrary, immediately relieved by *chloroform inhalations*. He was driven to try them through the obstinacy of his fits; the relief he experienced induced him to try it again, and he had come to abuse it so as to consume in one day sixteen ounces. He thus spent all his money, while his health became also deeply impaired. His liver got out of order; he had several attacks of severe jaundice while he was under my care, although he only used then a comparatively small quantity of chloroform, about five ounces at the most, in the twenty-four hours. At the time when he was using such large quantities of the drug, he had fallen into a state of acute mania like *delirium tremens*, and had been compelled to interrupt such dangerous inhalations. They had a marvellously rapid influence on the fits of asthma, calming them completely in less than a minute, although they returned in a short time, so that chloroform had to be administered again.

In some cases, when the paroxysms were much less violent than in this man, I have sometimes seen slight inhalations of chloroform suffice to calm the fit entirely, in the same way as in other instances a few puffs from a stramonium cigarette produce rapid and complete results.

I have dwelt so much on the treatment of asthma, gentlemen, because one plan of treatment cannot be madé to include all cases. Strange differences exist on this point; and whilst one man gets well almost at once, another who is apparently in a similar condition, derives no benefit and even feels worse from the use of the same remedy.

There is no inconvenience, however, and it is often advantageous, to combine these various remedies as I do now. And I will give you an instance of my practice.

On December 2, 1862, I was consulted by a young married lady, twenty-seven years of age, who had been subject to asthma since she was seven or eight years old, and whose fits were so frequent as to scarcely leave her free for a fortnight in the space of three months. I prescribed arseniate of soda at breakfast-time, iodide of potassium at dinner-time, belladonna in the evening, and every week, in the morning fasting, a dose of two drachms of pulvis cinchonæ flavæ. On July 1, 1863, this lady called on me again, and informed me that she had not had a single attack for a long time.

Let me say a few words more before I conclude as to the choice of localities, about which you will certainly be consulted.

When I spoke of the exciting causes of asthma, I told you of the influence of climates and localities on various individuals, and I related cases of persons who had fits of asthma only in certain countries, while others were continually subject to the disease. You should turn to advantage the knowledge of this fact; but when you counsel your patient to change his residence, you should appeal to his personal experience, or warn him, if he has not previously tried this plan which is often so efficacious, that he must be solely guided by his experience. There is indeed no absolute rule on this point, as one locality which will agree with one will not with another. Thus, low places generally agree with persons whose respiration is hard, as it is termed, while high grounds disagree with them. Yet I knew a superior officer who had frequent fits of asthma when he resided in Paris, but was free from them for ten months that he spent at Clermont-Ferrand, and had not the slightest oppression at the chest all the time that he stayed in the mountains of Mont Doré, where he made many excursions on foot and on horseback.

It seems that the elevation of the district where the patient resides exerts an influence which one would hardly have suspected. A resident assistant of the Beaujon Hospital, which is situated in the Faubourg Saint-Honoré, had constant attacks of asthma. He obtained Professor Marjolin's consent to exchage with one of his friends who was at the Hôtel-Dieu, a hospital built, as everybody knows, on the banks of the Seine, and in the lowest part of Paris. He never had a fit at the Hôtel-Dieu, but whenever he went to dinner with his old colleagues at Beaujon, he became at once oppressed in his breathing, so that he had to give up a treat which cost him too dear a price.

LECTURE LVI.

HOOPING-COUGH.

§ 1. *Specific Pulmonary Catarrh.—It is Contagious, and affects an Individual only once in his Life.—Incubation Stage.—Period of Invasion.—It begins like a Common Cold, which occasionally presents Special Characters, and may sometimes entirely constitute the Disease.—The fever of the Invasion Stage lasts from Seven to Eight, Ten, Twelve, or Fifteen Days.—Stationary or Convulsive Stage.—Characteristic Inspiration.—Expectoration of Bronchial Mucus.—Vomiting.—The Paroxysms are more frequent at Night than during the Day.—Third Period.—The whole Duration of Hooping-Cough is limited with great difficulty, and is directly proportionate to the Duration of the Prodromata.*

GENTLEMEN: As there are just now in the nurses' ward two children who are suffering from hooping-cough, I wish to give you a history of that complaint, and to describe it to you in such a way as to impress it deeply on your mind, and make the lesson practically useful to you.

You all know that hooping-cough is characterized by paroxysms which recur with greater or less frequency, and are more or less prolonged, consisting in several abrupt and jerking expiratory movements with loud coughing, followed by a long, painful, and whistling inspiration, which is somewhat pathognomonic.

The nature of this complaint has been regarded in various lights. Some considered it to be a neurosis, and others, a catarrh. It is both in reality, for the neurotic and catarrhal elements are always found. In my opinion, and in that of a great many physicians (and in particular of Dr. G. Sée, who has written a remarkable essay on the subject), hooping-cough is a special affection, a specific pulmonary catarrh. I say that it is a catarrh, because, as I told you just now, the catarrhal element is invariably present, and it is therefore a character which should serve to designate the kind of disease. The nervous element which is superadded to it, the nervous phenomena which accompany it, and which belong to hooping-cough exclusively, impart to it the specific character which will strike you in all that I shall say of its causes, its mode of transmission, its progress, its duration,—of its symptoms, in a word.

In the first place, this complaint may not only be epidemic, but it manifestly is also highly *contagious*. The fact is admitted by everybody. Now, as I have stated to you on several occasions, whenever a disease is transmissible from one human individual to another, from the lower animals to man, or from the latter to the lower animals, the fact necessarily implies the idea of specificity. For there can be no contagion without a germ of a special nature, capable of developing itself in a fit soil, and of reproducing itself, and manifesting its influence by constantly identical phenomena. This, then, constitutes already an important character, and this would of itself suffice to rank hooping-cough in the very extensive class of specific diseases.

Like most specific disorders, *it commonly attacks the same individual but once in his life.* There are yet exceptions to this rule, and I have myself known two cases in which children have had hooping-cough twice. Why, after all, should it behave differently from syphilis, or from eruptive fevers, such as variola, measles, scarlatina, typhoid fever, which have been known to attack the same individual several times, although they do not, as a rule, return?

Like specific diseases, again, hooping-cough is *chiefly met with in children;* and when adults, and even old people, suffer from it, it is either because they had escaped the disease at an earlier period of life, or because they are attacked a second time, as in the rare instances to which I alluded just now.

Lastly, this disorder has an *incubation stage,* which cannot, it is true, be defined, but which cannot be called in question when the fact is taken into account that hooping-cough never shows itself immediately after exposure to contagion, and that a certain number of days elapses before symptoms of the disease manifest themselves.

More than five-and-twenty years ago, I was summoned to a hotel in Chaussée d'Antin Street, to see a young lady who had just come from Bordeaux, and who, according to her father's statement, had caught a violent cold on the journey. She was intensely feverish when she reached Paris, and coughed incessantly night and day. Her cough had none of the characters of hysterical cough, of which I have had the opportunity of showing you examples here. It resembled the cough of a very acute catarrh, with this difference, however, that in ordinary bronchitis there are intervals of quiet, however short they may be, whilst in this case the cough was incessant, recurring twenty, thirty, forty times in the minute. There was, as I have said, very high fever. On ausculting the chest, I only heard some sonorous rhonchi. At first, I confess I thought that the case was one of galloping phthisis, and I could not conceal my anxiety from the patient's friends. But as a few days went by, the characters of the cough changed: there were eight or ten very violent shakings in succession, followed by a few minutes' rest. These characters soon became more distinctly marked, and came to resemble those of hooping-cough, so as to settle all doubt on the point in my mind. By questioning the patient's friends, and inquiring into the previous circumstances of the case, I then learned that a younger brother of this young lady, who had been left behind in Bordeaux, had had hooping-cough, which was at that time epidemic there. Had I been told of this circumstance on my first visit, I should have diagnosed the disease much more easily.

Enlightened by this case, I have since had the opportunity of observing several such; and both in private practice and in the children's wards placed under my care at the Necker and the Children's Hospitals, I have been enabled to recognize hooping-cough by the *obstinate coughing.* When I saw a patient with a cold which gave rise to paroxysms of coughing, recurring fifteen, twenty, and thirty times in the minute, and which continued in this manner for four, six, eight, or ten days in succession, attended with high fever, I at once recognized a specific catarrh, and after a certain time, varying between one and two weeks, hooping-cough manifested itself with its distinctly marked characters.

On the contrary, in some cases (which are much rarer than the preceding, for I have only met with two instances of the kind), the nervous element may show itself alone. From the beginning, the child has *spasms in the throat*, a kind of hiccup, which consists in an inspiratory laryngo-tracheal whistling, like the one which is heard at a later period, at each paroxysm of hooping-cough, and is quite pathognomonic of the disease. In one of the cases to which I alluded just now, I noted the curious circumstance that the paroxysms came on exclusively during expiration. Thus, the child had three or four inspiratory coughs, accompanied by a whistling noise, and expiration was not modified in the least; then, a few days later, this whistling was preceded by efforts to cough, which then occurred during expiration, and in a short time hooping-cough assumed its usual course and symptoms.

In the great majority of cases, I repeat, hooping-cough begins like a simple catarrah, both in children and adults. There is this distinction, however, that the cough is somewhat more frequent and obstinate, and that the patient has a more troublesome sensation of tickling in the throat and inside the trachea.

This catarrhal cough lasts from three days to a fortnight, sometimes three weeks, a month, and even longer, before it assumes the specific characters which it will manifest at a later period. I have in some cases known it continue throughout the complaint, and no convulsive cough supervene. Will it be denied that such cases were really instances of hooping-cough? But notwithstanding the absence of the specific cough, the other manifestations of the disease were amply sufficient for making a diagnosis. The catarrh was, in such cases, unusually obstinate. Thus, the patient's former colds had got well after a week or two, whilst this present attack lasted two, three, or four months. This cold had been caught at the same time as his brothers, sisters, or other companions became affected with hooping-cough. Like them, he had shown, in the beginning, *febrile symptoms*, which had lasted for three, four, five, six, eight, or ten days. The characters of the expectoration had been the same in all, and vomiting had come on in all after a fit of coughing. All the symptoms were present in such cases, therefore, except the convulsive character of the cough. Far, then, from calling in question the nature of the disease, such instances afford a new proof of its analogy to specific disorders, and to eruptive fevers in particular. In the latter we occasionally note the absence of an element which seems to be, and is indeed, their most important character, namely, the eruption, in measles and in scarlatina especially. Is the importance of the other symptoms to be called in question, then, when the specific eruption is absent? And why should it be otherwise with hooping-cough? The catarrh of the invasion stage is generally accompanied by *fever*, which is of greater intensity and longer duration than in a common cold. It rarely happens, as you are well aware, that unless the case be one of capillary bronchitis, whether the patient be a child or an adult, the fever which ushers in an attack of simple bronchitis continues longer than forty-eight or seventy-two hours. In hooping-cough, on the contrary, it is very common to find the fever of invasion last seven, eight, ten, twelve, and occasionally even fifteen days. So that, when I told you just now that hooping-cough began like a simple cold, I should have added that the catarrh of the invasion stage has nothing in common with ordinary catarrh except the cough which accompanies it; it differs essentially from it in regard of the attendant phenomena; and this cough itself, as I pointed out, differs in certain respects from simple bronchitis. From its earliest appearance, therefore, the manifestations of hooping-cough indicate its specific character.

The second stage is that of *spasm*, or of *convulsive* cough; or, if you prefer, the period when the disease is fully developed.

Allow me, gentlemen, to make a short digression here about the etymology of the term *coqueluche* (hooping-cough). It has come down to us from the middle ages. According to some, the disease was called after the hood (*cucullio*), which was used as a head-covering for persons suffering from hooping-cough; but according to others (mentioned by Sprengel), the word *coqueluche* is derived from *coquelicot* (red poppy), because the syrup of red poppy was for the first time employed in medicine in the treatment of hooping-cough. Others again say that it is derived from the word cock, from the hoop which occurs at the end of a fit of coughing, resembling the crowing of a young cock. The disease used to be and is still called in Picardy, the *cough which hoops*, whence the English name for the disorder, hooping-cough. Singular as these denominations may be, and however unscientific the term *coqueluche*, they have the immense advantage of being perfectly understood by everybody, and of at once presenting to the mind the idea of a special affection.

During the first stage of hooping-cough, the patient, as I have said before, coughs incessantly night and day. The cough becomes less frequent in the convulsive period; it returns every two minutes, for example, instead of every minute, while true paroxysms supervene with more frequent puffs of coughing; at first there was only one puff, but in proportion as the disease progresses, the number of these increases, so that even about the end of the catarrhal period, five or six might be counted; in the second stage, the patient has ten, twelve, fifteen, twenty, in succession without drawing in his breath. The characters of this cough are so very special, that it cannot be mistaken; there is nothing like it in any other catarrh, and hysterical nervous cough differs from it essentially.

When the patient is old enough to give an account of his sensations, he often complains of a pretty acute pain in front of the chest, of a sensation of tickling or pricking in the larynx and trachea which incite him to cough. His attempts at resisting are vain, and only retard, without ever averting, the paroxysm. The convulsive cough then bursts out as it were; whereas in a common cold or in any other affection of the respiratory passages in which cough exists, the patient can more or less easily draw in his breath after a few expiratory puffs; he is unable to do so in hooping-cough. An inspiratory movement first precedes the fit, and is followed by a series of expiratory movements which at first succeed one another slowly, and recur, as I said just now, a great many times, driving out all the air contained within the chest, and not allowing the patient time for breathing. The veins of the neck and face become turgid, the eyelids swell, and the eyes become injected with blood; tears are secreted in abundance; the cheeks and ears are congested, and this congestion spreads to the whole surface of the trunk, which is bathed in profuse perspiration. The unfortunate patient, whose respiration is so violently interfered with, falls into a fainting state, which is occasionlly pushed as far as complete syncope. At length, the convulsive movements of the respiratory muscles grow calm, an inspiratory effort takes place, accompanied by the characteristic hoop, which is perhaps due to spasmodic contraction of the larynx, the muscles of which have likewise been convulsed. This inspiration announces a moment's rest, but the truce is only of brief duration, and the same series of symptoms as before soon reproduce themselves. This second paroxysm again terminates in the same manner by an inspiration, which is this time longer than the first, and after several paroxysms of this kind, the patient looks exhausted with fatigue. In general, during these fits, which may last several minutes, he brings up a ropy, viscid, colorless liquid, to a considerable amount, and at the close he is sick and casts up mucus and food. There is pretty frequently also no vomiting. Sometimes, as in a recent case which came under my notice, that of the child of one of my pupils, the paroxysms terminate by a fit of sneezing or two.

When the paroxysms are very violent, they frequently cause accidents to which I shall call your particular attention, namely, epistaxis, subcutaneous hemorrhages, bleeding from the mucous membranes, and hæmoptysis; also cerebral congestion, to which are to be in some measure ascribed the convulsions which occasionally carry the children off. But before I speak of these complications, let us inquire into the patient's condition during the paroxysm. A child, for instance, is at play; a few minutes before the fit comes on, he stops: gay just now, he becomes sad; if he happen to be with companions, he goes away from them, and tries to avoid them. The reason of this is, if I may be allowed the expression, that he then *meditates* his fit, and feels it coming, on account of the sensation of pricking, or tickling, of which I spoke just now. At first he tries to avert the paroxysm; instead of breathing naturally and dilating his lungs to the fullest extent as he did just now, he holds his breath; he seems to understand that by entering his larynx in a volume, the external air will excite the fatiguing cough of which he has a sad experience. But whatever he may do, I repeat, he will fail in averting the fit, and will merely succeed in delaying it. Crying, or emotion of any kind which excites his nervous system, quickens the paroxysm. When this has come on, you see the patient seek a fixed object near him of which he may lay hold. He rushes into the arms of his mother or of his nurse, if he be a baby; if he be of a more advanced age, and happen to be standing, he stamps his feet in a state of convulsive agitation. If he were lying down he sits up quickly, and clutches his bed-curtains or bed-posts. After the fit his face is swollen, and this turgidity of the face, which sometimes continues for three weeks, may sometimes suffice of itself to make an experienced practitioner suspect hooping-cough.

These paroxysmal attacks recur a variable number of times in the course of the twenty-four hours. It is a remarkable circumstance that they are commonly more frequent by night than by day, or to speak still more accurately, from six o'clock in the evening to six in the morning, than from six in the morning to six in the evening. Can one attempt to account for this difference by saying that at night the child is no longer, as during the day, under the influence of excitation which occupied his nervous system in another direction? Whether this explanation be true or not, the fact is no less true and deserves to be noticed. In some cases, however, the reverse obtains, and the child has more paroxysms during the day than at night.

Being desirous of ascertaining the number of paroxysms which a child might have in the course of twenty-four hours (as may be easily done), I adopted the following plan in my children's ward in the Necker Hospital. I asked the child's mother to prick, at each fit, a card with a pin, and by adding up the number of holes, I made out the next day how many paroxysms had occurred since my visit of the previous day. I was thus enabled to draw the conclusion, from a pretty good number of cases, that when the disease is of medium intensity, a child may have about twenty fits in the course of twenty-four hours; when it is more violent, he may have from forty to fifty, and in still more severe cases, the number of paroxysms may get up to sixty, eighty, a hundred even. When the number exceeds forty, the prognosis becomes grave, whence this proposition may be laid down that, under the same circumstances, the disease increases in danger in proportion to the number of paroxysms. More than this, it may be almost absolutely affirmed, that when the attacks return more than sixty times in the twenty-four hours, the child will die of some of the complications to which I have already alluded, and of which I am going presently to speak.

In the *third period* of the disease, the paroxysms become more and more rare, less and less prolonged, and less and less severe. The characteristic hoop of the closing inspiration is less and less marked, and then disappears entirely. Yet, when from any cause, from exposure to cold, or from mental emotion, the child, who had not coughed for several days, does so again, the paroxysms are exactly like those of the second stage. At this period of decline, the bronchial mucus which the patient expectorated after each paroxysm, is replaced by thick, opaque greenish, occasionally purulent sputa, having all the characters of genuine catarrhal expectoration.

The *duration of hooping-cough* is a point of the highest importance. Almost every day I call your particular attention to the absolute necessity of fully knowing the natural course of a disease. I have told you many a time here, and I will repeat it again and again, that this grave question is of paramount importance in practical medicine, because it is, indeed an accurate knowledge of the natural course of a disease which can alone enable us to estimate the value of the methods of treatment which we employ. Eruptive fevers, to which, as Dr. Sée judiciously observes, hooping-cough presents such striking analogies, and other diseases, like phlegmonous angina, for example, which develop themselves within a very distinctly-defined period, may prove fatal before the time appointed by nature, either from intercurrent complications, or from inopportune and unskilled interference of the medical attendant, but treatment never shortens their duration. Besides these diseases, with fixed periods, there are others, such as typhoid fever, the duration of which cannot be exactly known beforehand, although the evolution of their symptoms is equally unavoidable. Hooping-cough belongs to the latter class. It is most difficult to limit its duration rigorously. In some cases, it gets well in a week, sometimes in less, and I remember a child, a patient at the Necker Hospital, in which it lasted three days only. Hooping-cough was then epidemic in my wards, and nearly all the children in them had caught it. The child to whom I allude suddenly showed symptoms of a violent catarrh, which was the next day accompanied by repeated and characteristic convulsive paroxysms. These recurred for the next seventy-two hours, and on the fourth day there only remained the signs of an ordinary coryza. This boy remained for some time in my wards, and although, I repeat, hooping-cough was epidemic there, he never presented any other symptom of the disease.

One rarely meets with such fortunate cases in private practice. The evolution of hooping-cough takes most commonly six weeks at least, and the complaint lasts in general from fifty to sixty days. In opposition to the exceptional cases in which it gets well in a week and even less, others occur in which the disease lasts several months and even a whole year. In order to estimate the efficacy of any plan of treatment whatever in hooping-cough, this natural course of the disease must be therefore taken into account. No method of treatment should be regarded as useful until it has been tried in a great many cases, and has been found to cure in less than six weeks, or to diminish at least the frequency and the intensity of the paroxysms.

It is an interesting fact that the general duration of the disorder is directly proportionate to the duration of the prodromata. If these have lasted a short time only, hooping-cough is of short duration also, and the more quickly the convulsive cough has made its appearance, the more quickly also does it recede. So that, although there are pretty numerous exceptions to this rule, the ulterior course of an attack of hooping-cough may to a certain extent be prejudged from the course of the disease at its onset.

§ 2. *Complications.—Capillary Bronchitis.—Peripneumonic Catarrh.—Pleurisy.—Pulmonary Congestion.—Pulmonary Phthisis.—Vesicular and Interlobular Emphysema.—Vomiting.—Diarrhœa.—Hemorrhages.—Rupture of the Tympanum, and Bleeding from the Ear.—Cerebral Congestion.—Convulsions.—Treatment.*

The *complications* which supervene in the course of hooping-cough are of various kinds. Some of them are inherent in the very nature of the two principal elements of the disease, which is, in my opinion, a specific catarrh characterized by the nervous phenomena which I have described. Under certain circumstances, the catarrhal element becomes excessively acute and intense, and an inflammatory condition is set up which modifies the regular course of the disease, and gives rise to complications which may become dangerous.

When hooping-cough runs a regular course, there is only heard on ausculting the chest, when a paroxysm is impending, a weak respiratory murmur, probably on account of the spasm of the bronchi: loud sonorous mucous rhonchi are heard at the same time. When the paroxysm is over, the vesicular murmur is either normal or, as more commonly happens, a few large moist bubbling rhonchi, and sonorous ones, are still heard. When catarrhal complications arise, fever is lighted up; there is considerable oppression at the chest; on auscultation, fine mucous rhonchi, soon passing into the subcrepitant kind, are heard, and then tubular breathing, signs of the *capillary bronchitis*, and of the *peripneumonic catarrh*, which have become developed. Sometimes also, especially in children and in adults, there are dulness, absence of all respiratory murmur, tubular breathing and ægophony, indicating the presence of *pleuritic effusion*. These inflammations of the lung-substance and of the pleuræ are the most frequent causes of the patient's death.

The phenomena, which then manifest themselves, point again to the specific character of the disease. If, as some assert, hooping-cough were merely an intense form of bronchitis, and if the nervous manifestations of the convulsive cough were dependent upon the inflammatory element, these manifestations should be more exaggerated in proportion as the bronchitis became more acute, while, on the other hand, they should diminish or cease simultaneously with the symptoms of inflammation. But it is the reverse which occurs. Hence, when in a child suffering from hooping-cough, who used to have from 50 to 60 paroxysms in the course of twenty-four hours, you find these paroxysms cease suddenly, although the disease is still in the middle of the stationary stage, be on your guard and take care not to augur favorably from this circumstance, because you will have to deal with some inflammatory complication; the convulsive phenomena ceased so suddenly only because they have been silenced by fever; the nervous has been put down by the inflammatory element.

If a febrile affection manifest itself in the course of hooping-cough; if, for instance, the patient get measles, scarlatina, or small-pox; or if a phlegmonous inflammation supervene, accompanied by general reaction and fever, this fever, in the language of Hippocrates, stops the spasm, *spasmos febris accedens solvit*, and the phenomena dependent upon the nervous element cease for a while. If there be no eruptive fever, and no external inflammation to account for the febrile condition and for the cessation of the paroxysms, be on your guard, I repeat; auscult the chest carefully, and you will discover signs of capillary bronchitis, of peripneumonic catarrh, fine subcrepitant rhonchi and tubular breathing; or signs of pleuritic effusion, tubular breathing, and ægophony.

The nervous element depends so little on the inflammatory element that, when you find the paroxysms recurring as frequently as before, you may predict beforehand that stethoscopic examination of the respiratory apparatus will point to retrocession of the inflammation.

The catarrhal and nervous elements of hooping-cough are, therefore, perfectly independent of one another. They run a parallel course when the disease is regular, but they separate when one of them, from some cause or another, becomes exaggerated and runs a different course from its usual one.

Do not think that this occurs in hooping-cough alone; for it is observed in other diseases, made up of compound elements, and I have called your attention to the same thing in spasmodic asthma. If in this latter complaint, bronchitis, pneumonia or pleurisy supervene, the paroxysms of dyspnœa cease, and although the patient's breathing is then more oppressed than that of other individuals, it is yet less so than previously, and in a different manner from what it was when the patient had fits of asthma. I told you to be on your guard when the spasmodic symptoms of hooping-cough disappear suddenly, because the inflammatory complications which silence them are more grave, other circumstances remaining the same, when they occur in the course of this disease than when they supervene in ordinary circumstances.

The efforts at expiration during the paroxysms necessarily producing a certain amount of pulmonary congestion, the capillary bronchitis, the pneumonia or pleurisy, will be all the more serious and will get well all the more slowly, as hooping-cough may last four, five, six months and more, so that the return of the paroxysms will prevent the complete disappearance of a congestive condition which the efforts at coughing tend to keep up and may aggravate.

As the persistence and obstinacy of inflammatory phenomena favor the evolution of diathetic manifestations, you may conceive how it happens that hooping-cough is such a frequent determining cause of the development of *pulmonary phthisis* in children with the tubercular diathesis; but I cannot admit, as a physician of undoubted talent does, that the specific character of hooping-cough has something to do with this. The inflammatory element alone seems to me to be accountable for the development of tubercles.

The air-cells of the lungs may burst in consequence of the violence of the paroxysms of coughing, and air may thus diffuse itself into the interlobular cellular tissue. There is, then, in the interval between the paroxysms an extraordinary sense of oppression at the chest, and it occasionally happens that this *interlobular emphysema* spreads to the subcutaneous cellular tissue. The air diffused between the lobules of the lung makes its way along the roots of the bronchi, and gives rise to a subcutaneous emphysema which spreads more or less rapidly along the trachea, and first manifests itself in the cervical region, which swells considerably and crepitates in a characteristic manner when pressed. This subcutaneous emphysema may affect the whole body by ascending along the trunk. Such an accident is grave, and generally terminates fatally. It is fortunately rare, but it yet deserves to be mentioned, although Rilliet and Barthez do not speak of it in their work on Diseases of Children as one of the complications of hooping-cough.

Vesicular pulmonary emphysema is nearly always found when the body of a child who has died of hooping-cough is examined. It is a necessary result of the violent coughing. After the complaint has got well, the air-cells gradually recover themselves, and all traces of the lesion disappear. The same thing happens in adults, when emphysema has been the consequence of a very obstinate attack of bronchitis, which at last gets well. But when hooping-cough attacks persons of advanced age, as in cases that have come under my observation, it produces irrecoverable vesicular emphysema, and when it is cured, the sense of oppression at the chest continues to the close of the patient's existence.

During the paroxysms, the patient often passes his *urine*, and even has involuntary evacuations from the sphincters of the bladder and large intestine being unable to resist the violent efforts of coughing. *Hernia* is also frequently produced in hooping-cough as a consequence of these efforts.

The same cause (namely, energetic and convulsive contraction during effort) has been ascribed for the vomiting, which, as I have told you already, follows upon each paroxysm.

But we have seen that this accident could be regarded as inherent in hooping-cough. It seems to constitute its natural crisis, so much so, that a paroxysm of hooping-cough, whatever be the number of the fits of coughing, usually terminates only when vomiting has taken place. It is therefore a very common phenomenon, and is occasionally attended with grave consequences. Say, for instance, that a child has a good many fits of coughing in the course of the 24 hours, say 30 or 40, which consequently recur about every half hour; as every one of them is followed by vomiting, the child's nutrition must necessarily suffer as he brings up all the food he takes. When the medical attendant, therefore, does not take care to carry on the treatment according to the plan which I shall presently describe to you, with the view of combating this fearful complication, it is not rare to find this obstinate vomiting carry off the child, who literally starves to death.

The disorders of nutrition, by depriving the blood of its materials for repair, have probably something to do with the production of *hemorrhage*, to which a patient suffering from hooping-cough is liable, although the obstruction to the venous circulation is in some measure sufficient to explain them. The bloodvessels get congested during the efforts at coughing, and this congestion, which is transient at first, becomes at last permanent from being constantly repeated, and may be carried to such a point that the blood itself, or its more liquid parts, may escape from the capillaries.

Epistaxis is the form of hemorrhage which most frequently occurs, and one may pretty often see a child bleed from the nose in a fit of hooping-cough. When this accident does not recur frequently, it presents no gravity, but not so when epistaxis occurs from the outset of the complaint, and is somewhat copious, and returns regularly. In the beginning, as the blood is of normal plasticity, hemorrhage only occurs when there is vascular congestion; when the circulation resumes its normal course, the bleeding ceases; but when, in consequence of the repeated hemorrhage, the blood has become less plastic, epistaxis occurs not only while the face is congested, but also continues for some time afterwards. As the plasticity of the blood goes on diminishing, and the child becomes more and more anæmic, he bleeds more and more abundantly from the nose, and the epistaxis is so prolonged that medical interference is needed to stop it. You may conceive what very serious complications these hemorrhages are, not because I believe that they often cause the patient's death, but because they render him liable to nervous accidents, to convulsions, which are never so common as in children who are exhausted by loss of blood.

Blood may also be *expectorated;* in some cases this blood comes from the mucous membrane of the mouth, from the gums, the pharynx, the posterior nares, but in others, it comes from the surface of the bronchi. *Hæmoptysis* is a pretty common accident, although the reverse has been asserted; some authors have gone so far as to state that, when moderate, it was a favorable symptom. Without concurring in that view, I admit that hæmoptysis is, generally speaking, of no gravity whatever, and need cause no anxiety.

In the rapid sketch which I gave you of a paroxysm of hooping-cough, I stated that, under the influence of the violent efforts at coughing, the face got congested, the eyes injected with blood, and tears were abundantly secreted. I will now add that the bloodvessels of the eye may be congested to such a degree as to give rise to conjunctival hemorrhage, and I have seen a little boy, two years of age, who was suffering from severe hooping-cough, cry *tears of blood.*

In the case of a young woman, small drops of blood came out during each fit of coughing from a *nævus maternus* situated underneath her left eye. This singular form of hemorrhage continued all the time that the hooping-cough lasted, and yet the disease was very mild.

This tendency to hemorrhage often gives rise to *subcutaneous ecchymoses.* In a little girl from nine to ten years old, there occurred during a severe attack of hooping-cough, an extravasation of blood into the *subconjunctival* cellular tissue, and into the cellular tissue of the eyelids, which passed through the ordinary stages of resolution, and successively stained the affected parts of a dark red, a violet red, a brown, and a greenish-yellow color. You will, doubtless, meet with similar cases.

Hemorrhage from the ears is a rarer accident, and two instances of it came under Mr. Triquet's notice at his dispensary, in the winter of 1860. The children's mother had noticed a flow of blood from their ears during a fit of coughing in the course of hooping-cough. On examining the auditory meatus and the membrana tympani, a linear rent of the latter was found a little below the handle of the malleus. In both instances the left membrane was alone torn.

One of the children was six, and the other five years old. In England, Dr. Gibb has met with this accident four times, in children from four to nine years old.*

These four cases occurred in the course of an epidemic of hooping-cough, which had attacked 200 children from six to nine years old.

Now, in these four children, and in the two which came under Mr. Triquet's observation, an examination of the ear always detected a linear rent of the membrana tympani.

In two of Dr. Gibb's patients, the membrani tympani of both ears was ruptured, and in one of the children the rent was triangular or heart-shaped.

In four of the eight cases the seat of the rupture was near the circumference of the membrane, in two it was central, and in one the membrane was torn into three pieces, from one to two millimetres in length. A small coagulum, which intervened between the lips of these small rents, pointed in a positive manner to the source of the hemorrhage, namely, laceration of the mucous or inner layer of the membrana tympani. The rupture in every case, except where the membrane was torn into three pieces, healed up by immediate or primary adhesion in the space of a few days. The triangular rent was the only one which did not get well, and it gave rise to prolonged suppuration and obstinate deafness.

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* British Med. Journal, and London Gazette, November, 1861.

The mechanism of such an accident is easily understood. During the efforts and convulsive coughing of hooping-cough, air is violently driven through the Eustachian tube into the cavity of the tympanum. The pressure exerted by the column of air overcomes the resistance of the membrana tympani, lacerates it in its weakest part, beneath the handle of the malleus, or tears it away at its circumference; and the laceration of the mucous lining of the membrana tympani gives rise to the hemorrhage which comes from the ear, in rare but undoubted cases, as shown by actual examination. From these hemorrhagic accidents, I am naturally led to speak of the *convulsions*, which, as I said just now, are often their indirect consequence, when very copious and frequent loss of blood has thrown the patient into a state of anæmia, which gives rise to a peculiar nervous susceptibility.

Attacks of eclampsia may again be a direct consequence, if not of the hemorrhage itself, at least of the causes under the influence of which the hemorrhage occurred. They are, perhaps, then caused by cerebral congestion, and seem to be dependent upon a peculiar modification of the cerebral centre, brought on by the afflux of blood resulting from the fits of coughing.

You must all be familiar with the sense of vacancy, of astonishment, which comes on after a violent effort kept up for a little while, and which is evidently the consequence of a transient congestion of the brain. This phenomenon of effort recurs in hooping-cough at very short intervals, and at last gives rise to more serious accidents. Thus, when the patient is old enough to describe his sensations, he often complains after violent fits of coughing, of *headache*, which is occasionally so intense as to make him cry. This pain in the head is followed by a state of hebetude, like what is caused by concussion of the brain, and which lasts more or less. In some cases, symptoms of true cerebral congestion show themselves. I once attended a lady who used to fall into the same state of stupor as that which follows epileptic fits; this lady suffered also on several occasions from incipient paralysis, marked weakness of one arm. You understand how such a disturbance of the functions of the brain may, in children, give rise to convulsions.

Convulsions may come on also independently of hemorrhages and of congestion. They are then due to the nervous element which gives to hooping-cough its specific character; the nervous excitability which already manifests itself, as a rule, by the production of convulsive fits of coughing, spreads to the whole system, either in consequence of the weakness of the patient's constitution, or of acquired weakness, his strength having been exhausted by the prolonged duration of the complaint, by disordered nutrition, or any other cause.

These nervous accidents, which sometimes also consist in *delirium*, in *extreme agitation*, are more frequent and more grave in proportion to the tender age of the child. They nearly always terminate fatally when they coincide with the inflammatory complications which I mentioned to you.

The *treatment* of hooping-cough is a matter of considerable difficulty, because it is a complaint which yields with extreme difficulty to the various measures which we can oppose to it. Yet I do not admit that we are entirely powerless against it; and, in opposition to J. Frank, who asserted that we can kill a patient suffering from hooping-cough before the term of his complaint, but that we can never cure him, I believe that in a pretty good number of instances, a well-conceived plan of treatment markedly diminishes its duration.

I will not pass in review the various remedies which have been lauded against it. Every author has his own formula, and I need not enumerate all these pretended specifics. I will merely indicate to you a few of the methods of treatment which appear to be of some utility, and will dwell specially on the one which is, in my opinion, of unquestionable efficacy.

According to Laennec,* the most useful treatment at the outset of hooping-cough, consists in the administration of *emetics*, repeated every day, or every other day, for a week or two. Children, after all, bear vomiting much better than adults. Laennec even preferred, in the case of children, *tartar emetic* to *ipecacuanha*, because of the extreme variability of strength of the different kinds of ipecacuanha met with in commerce, and which are obtained from different plants. Tartar emetic, he added, being soluble, may be much more easily administered in as small doses as may be required by the age and feebleness of the child. Others, instead of tartar emetic and ipecacuanha, prefer *sulphate of zinc* or *sulphate of copper*, on the ground that they have an antispasmodic, in addition to their emetic, action.

I do not admit this twofold action of the salts of zinc and copper. When I wish to bring on vomiting in a child, I, however, give sulphate of copper, because it is the most certain emetic which I know of. I prefer it to ipecacuanha, because, as Laennec remarked, ipecacuanha often fails, and to tartar emetic, because this latter sometimes gives rise to very unpleasant consequences. However prudently it may be administered, according to the individual, and according to his condition at the time, it may act more powerfully than was intended. It has in some cases brought on profuse purging, vomiting, and diarrhœa, and has given rise to choleriform symptoms, and thrown the patient into a truly alarming state of weakness.

For these reasons I prefer using the sulphate of copper, which I administer after the following method: I order from five to nine grains of the salt in the case of a child, and fifteen grains in that of an adult, to be dissolved in three ounces of distilled water, of which a dessertspoonful is taken every ten minutes, until vomiting is produced.

This method of administering emetics in fractional doses is the one which you see me adopt constantly, whatever be the drug I prescribe, and whatever be the indications of the emetic treatment. By this means I need not fear to go beyond the mark which I propose to myself. At the outset of hooping-cough, and when the disease is in its fully developed stage, when the cough is accompanied by symptoms of impending suffocation, emetics are of some utility, and I have known them in several cases very markedly diminish the number of paroxysms.

Antispasmodics must necessarily have held an important position in the treatment of a disease in which the nervous element plays a very marked part. Hence a great many formulæ contain valerian, castoreum, musk, assafœtida, ammoniacum, oxide of zinc, &c.; but the utility of these various remedies, and of antispasmodics in general, has always seemed very questionable to me.

Narcotics and *stupefying* drugs are much more efficacious, and of them, *belladonna*, to which I alluded just now, or its alkaloid, *atropia*, is, in my opinion, the most heroic remedy which can be used in the treatment of hooping-cough.

In order, however, that belladonna should produce its full effects, it should be administered according to a particular method, which is of such importance that if you neglect to observe it, you will not succeed in curing hooping-cough, any more than you will succeed in curing ague, however large the dose of bark you may give, unless you follow certain rules, which I will some day point out to you.

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* Traité d'auscultation médiate, 4th ed., Paris, 1837, t. iv, p. 228.

Before I give you the details of the plan of treatment to adopt, I must first establish the following capital point: The active principle of solanaceous plants influences neuroses only when given in sufficiently large doses, and this influence lasts for some time; but lest the therapeutic effects should be greater than desired, the medicine should first be given in doses which are probably less than those needed for exerting a favorable action on the disease; these doses must be gradually increased until therapeutic effects begin to show themselves. As soon as this result is obtained, it is generally sufficient to continue the same daily dose in order to increase the good effect produced. If the dose which has brought on these good results were increased hastily with the view of accelerating the cure, and especially if it were repeated on the same day, one might at first wonder at the success obtained, but an unpleasant dryness of the fauces, and some disturbance of vision, which increases rapidly, would soon render a diminution of the dose necessary, and the consequence of this would be to allow the disease to reproduce itself, and to escape the influence of the mode of treatment.

Bearing well in mind these general principles, the treatment is to be carried on after the following method:

If the patient be an infant, have pills made containing each one-tenth of a grain of extract of belladonna, and one-tenth of a grain of powdered belladonna leaf. For children above four years old, and for adults, the pills should contain one-fifth of a grain of extract, and one-fifth of a grain of the pounded leaf. The pills should not be silvered; and as there are children who do not know how to swallow pills, even when they are given to them in jam, honey, or panada, they can be dissolved in a little syrup, and thus easily taken when put on the tongue. One pill is given in the morning, fasting, and another the following morning. First take care to ascertain the number of paroxysms by means of the method which I have told you, namely, by pricking a card with a pin, and keep a separate account of the diurnal and nocturnal attacks. You can then easily judge of the effects of the treatment by comparing the number of fits of the preceding day with that of the fits on the previous days. Thus, say that a child who had at first thirty-five paroxysms in the course of the twenty-four hours, has only thirty after taking belladonna, the medicine will evidently have acted on him. Or say, that the sum total of the fits is the same, but that instead of there being four or five paroxysms, there be only two or three; or say again, that the paroxysms and the fits of coughing, of which they consist, are as numerous as before, but that the fits have been less violent: in all these cases there is, after all, real improvement, and the same dose is henceforth to be given. If, on the contrary, the fits have been as numerous and as violent, an additional pill is to be given, and two pills are to be taken at the same time. This is a point of capital importance, gentlemen. Whatever quantity of belladonna you may give, it is an essential point that it be taken in one dose. If you have been obliged to increase the original quantity ten or twelve times, let the patient take it in one dose, in the morning, fasting, at the same hour, and not at distant intervals in the course of the day. But before you increase the quantity, wait two or three days, and according as there has been an improvement or not, keep to the same dose, or increase it by degrees, unless symptoms of poisoning should come on, when of course you must stop.

When the fits of coughing have markedly diminished in number and violence, when, for example, they have fallen down from thirty to ten, go on giving for seven or eight days the dose of belladonna which has apparently brought on this improvement. If this continues, diminish the quantity of the medicine, by a reverse method to the former, namely, by taking off first one, then two, next three pills, and so on. If the fits of coughing increase again, resume the dose which stopped them. Lastly, when these fits have entirely ceased, and the disease may be justly regarded as cured, still go on with the belladonna for six or eight days before entirely stopping all treatment.

Since atropia has been used in medicine, it has been substituted for belladonna, and with this advantage, that while it possesses all the active properties of the plant, it has a fixed composition which the officinal preparations of belladonna do not always possess.

When the child is very young, I prescribe a solution of one-fifth of a grain of the neutral sulphate of atropia in five ounces of water, a teaspoonful of which is therefore equivalent to $\frac{1}{200}$ of a grain of the alkaloid. This is the dose given in the beginning, and it is gradually increased, according to the rules which I laid down just now with respect to belladonna.

You must be on your guard against what I might term pseudo-relapses, for hooping-cough is a complaint which seems to recur after it has been really cured. A child may have, a month after the final cessation of the disease, a fit of coughing as in hooping-cough, when he cries or gets into a passion. More than this, the same thing may happen if he gets a catarrh, six or twelve months afterwards. Do not from this infer that there is a relapse. His cough assumes those characters because his economy—his nervous system—falls into its former bad habit, if I may be allowed the expression.

The treatment of hooping-cough by *revulsives,* by the application of blisters to the chest, frictions with croton oil, with spirits of turpentine, is far from giving the good results which has been ascribed to it. I would say nothing about it, if it were not to raise my voice against the dangers of a remedy about which so much noise was made by the man who proposed it: I allude to frictions with *Autenrieth's ointment.* Autenrieth ordered the chest of children suffering from hooping-cough, especially towards the close of the second stage, when the expectoration is becoming muco-purulent, to be rubbed three times a day with an ointment containing tartar emetic in variable proportions. This was to be continued, until pustules formed, which soon passed into ulcers. The appearance of these pustules, not only over the chest, but over other regions of the body also, especially the inner aspect of the thighs and the genital organs both in boys and girls, was in Autenrieth's opinion a sign that the system was saturated with the drug, a result which he thought one should always endeavor to bring about.

I have often told you my opinion with regard to this pretended saturation with tartar emetic, whether we look at its manifestations about the mouth or about the skin. The characteristic eruption to which it gives rise is said to be the result of the general effects of the drug, and not of the local irritation caused by tartar emetic when in contact with the skin or with mucous membranes. This eruption, however, is shown to be the effect of a local action by the fact that it does not manifest itself when the tartar emetic is given in pills instead of in a mixture, so as to prevent prolonged contact of the drug with the mucous membrane of the mouth and pharynx, and that when it is taken internally in very large doses, as in pneumonia, according to Rasori's method, no cutaneous eruption ever occurs.

Setting aside this pretended saturation of the system with tartar emetic, the treatment of hooping-cough by Autenrieth's ointment offers the greatest dangers and no advantage whatever. It is horribly painful, considerably more so than blisters, and occasionally gives rise to inflammation which, starting from the pustules, affects the cellular tissue, spreads in depth and gives rise to serious accidents. As one of several instances of the kind, I will mention the case recorded by Dr. Blache in the article on hooping-cough in the "Dictionnaire de Médecine." The patient was a little girl six years old, and the use of the tartar emetic ointment produced the most lamentable results. Deep ulcers followed upon the pustules, one of which, at the base of the sternum, was nearly two inches in diameter. It had exposed and completely detached from the bone the extremities of the costal cartilages, which floated in pus that was secreted in extreme abundance, in spite of all attempts at diminishing the suppuration. Symptoms of pyæmia soon showed themselves, and the child died of a colliquative diarrhœa which nothing could stop. Autenrieth was led into error by the circumstance that the convulsive phenomena of hooping-cough grow quieter after the use of the tartar emetic ointment, in consequence of the febrile reaction excited by the inflammation of the skin, but show themselves again as soon as the inflammation has ceased.

Besides these immediate disadvantages, tartar emetic ointment has others which are less serious, but are still worthy of the practitioner's attention. The pustules and the ulcers which form after them, leave behind indelible scars which may simulate the marks of scrofula. Trifling as this circumstance may seem, you will understand its value, and you will one day appreciate the utility of all these small details into which I am not afraid of entering in the course of my clinical teaching, whenever an opportunity presents itself.

Before concluding, I have to speak of the treatment of the complications of hooping-cough.

I have told you that *vomiting* was often a very serious complication of the disease, as it was apt in some cases to cause death by starvation. It is indispensable, then, that you should know how to feed the patient, and the first rule to bear in mind is to give him food so that he may keep it. Experience can alone enlighten you on this point; for some patients are sick only during the day, and you must then tell them to postpone their meals until evening. When vomiting recurs both during the day and at night, the patient should take food immediately after a paroxysm, because the next paroxysm will be some time before coming on. However averse the child may be to taking food, he must be made to do so, and he should have solids instead of liquids, as they are less easily brought up.

When belladonna or atropia is administered, the paroxysms return at longer intervals under the influence of that treatment. Advantage may be taken of these intervals of rest to give the patient nourishment; besides, even though it should not lengthen the intervals between the paroxysms, belladonna stops the vomiting or renders it less violent. In some exceptional cases, in spite of the administration of this remedy, the patient cannot keep down his food. Opiates should then be given in small doses, in combination with preparations of belladonna. The child should take immediately after vomiting, and just before eating, *half a drop, or even one drop of laudanum (of Sydenham)*. To compel the patient to eat immediately after he has brought up his food and to give opium internally, are therapeutic stratagems of the greatest importance.

On account of the consequences which may follow it, *hemorrhage*, especially when from the nose (the gravest form of bleeding in hooping-cough), should be treated at once. Of the means that we may employ there is one the good results of which are real although unaccountable, namely, the plan of raising the arm which is on the same side as the nostril from which the blood issues. I might enumerate a great many hæmostatic remedies: astringent powders and liquids; injections with water acidulated with sulphuric, nitric, and hydrochloric acids; cold applications to the forehead and nucha; and various other measures which you know, and at the head of which I place injections into the nostril of water as hot as the patient can bear. In extreme cases, and when the bleeding resists every other measure, the nostrils may be plugged, as a last resource, either with Gariel's india-rubber bladders, or by means of Belloc's sound. Plugging of the nostrils presents no inconvenience in adults, but it may in children bring on excessive agitation which increases the violence and number of the paroxysms of hooping-cough. It should therefore be employed only as a very last resource. While acting directly on the seat of the hemorrhage, internal remedies may be administered at the same time, such as acidulated drinks, the sulphuric lemonade, mixtures containing the *eau de Rabel*, ratanhia, matico, kino, and best of all, powdered bark.

As to the grave inflammatory complications about the chest, such as capillary bronchitis, pneumonia, pleurisy, they demand special treatment, of which I need not speak here.

LECTURE LVII.

ANGINA PECTORIS.

Angina Pectoris symptomatic of an Organic Affection of the Heart or of the Great Vessels.—In such cases, the Organic Lesions merely favor the Development of the Neurosis.—Idiopathic Angina Pectoris, due to a Rheumatic or Gouty Diathesis.—It may be a Manifestation of Epilepsy, and may then constitute either a Variety of Epileptiform Neuralgia, as is most frequently the case, or a Variety of Aura Epileptica.—Angina Pectoris dependent on Graves's Disease.—Its Invasion is Sudden, its Symptoms Variable.—It may cause Sudden Death.—Treatment.

GENTLEMEN: In spite of the numerous publications which treat of angina pectoris, the history of that complaint is not very satisfactorily, known; and the various opinions which have been expressed as to its nature have thrown so little light on the subject that I wish, in my turn, to communicate to you my views concerning this singular neuralgia.

A woman, who died some time ago in St. Bernard Ward, of aneurism of the aorta, furnished us with a remarkable instance of this complaint. Her attacks, which at first occurred at pretty distant intervals, recurred very frequently towards the last, and few among you have not had an opportunity of witnessing one of those awful paroxysms.

She was suddenly seized with an excruciating pain, without any appreciable determining cause, either while sitting motionless on her bed (the only posture which she could retain) or while moving. This pain started from the precordial region, and radiated from it to the base of the chest, producing there a sensation of constriction which the patient compared to that which might be caused by an iron girdle tightened with force. It then spread to the loins, and, ascending towards the cervical region, attacked the left arm, and extended into the very tips of the fingers. The skin of the hand and forearm could be then seen to become excessively pale, and almost immediately afterwards to turn of a markedly bluish or livid tint. After the pain had ceased, the arm and hand felt numb for a few minutes. The pain was such as to make the patient cry out; her features were contracted, she sat in an upright position, as if dreading to be choked, although she breathed pretty freely. The paroxysm lasted a few seconds, and returned at intervals, which grew proportionately shorter as the disease drew to a fatal termination.

In this instance, the angina pectoris was symptomatic of an organic lesion, and such was also the case in a patient about whom I was lately consulted by Dr. Périer.

He was a military superintendent, fifty-five years old. His attacks, which dated seven years back according to his statement, were chiefly characterized by a sensation of numbness and tingling in the skin of the left axilla, and spreading from there to the whole corresponding side of the chest. He often felt shooting pain, like that of neuralgia, but which was quieted by his squeezing his back against a resisting surface, as a piece of furniture, for example.

For the last six or eight months, he had become subject to some oppression at the chest. A somewhat rapid walk, the least active exercise, brought this back, and he was troubled with pain, even if he had been engaged in merely signing many papers, in the discharge of the duties of his post.

On examining his chest, all the physical signs of aneurism of the aorta were made out. The action of the heart was violent without abnormal bruit; higher up and in front, a distant double bellows-sound was heard, and was audible also in the back all over the left side of the chest, but in the greatest intensity along the vertebral column, on a level with the spine of the scapula. Deep percussion also over the plessimeter made out dulness over the same spot. Vesicular breathing was perfectly normal all over the chest.

These two cases would seem to confirm an opinion held by some physicians, namely, that *angina pectoris* depends on the presence of appreciable organic lesions of the heart, of the great vessels, or of neighboring organs. You are aware that Heberden (who was the first to give to this complaint the name by which it is now known, and who has left us a pretty good description of it), and, after him, Parry, Kreysing, Burns, J. Frank, &c., ascribed angina pectoris to ossification of the coronary arteries. Others, on the contrary, have referred it to hypertrophy with dilatation of the heart, ossification of the auriculo-ventricular or aortic valves, pericarditis, accumulation of fat on this membrane in the mediastinum or on the heart itself, displacement of this organ, compression of it by a tumor or through abnormal development of some one of the abdominal viscera, aneurismal dilatation of the aorta, inflammation of this vessel, mediastinal abscess, ossification of the costal cartilages, &c.

I do not deny that angina pectoris may coexist with one or other of these various lesions, and that it often (most often, perhaps) is symptomatic, as has been said, of organic diseases of the heart or of the great vessels. But while, on the one hand, the variety of these lesions makes one suspect their etiological value, on the other, the numerous cases in which such lesions exist without the patient suffering from anything like paroxysms of angina pectoris, and, per contra, authentic instances of individuals who during life presented all the characteristic symptoms of angina pectoris, while, after death, dissection disclosed no anatomical lesion by which these symptoms could be accounted for, prove that this complaint is not essentially due to the presence of organic diseases.

From the absence of appreciable structural changes, and from the extreme variability of the phenomena, which I shall endeavor to describe to you, we must conclude that angina pectoris is a neurosis, or, to use a more precise term, a neuralgia. As to its seat, which some have placed in the diaphragm, others in the respiratory muscles, and most in the heart, this neuralgia generally affects the cardiac nerves given off by the pneumogastric, and radiates to the nerves of the cervical and brachial plexuses.

One of my oldest and most intimate patients, a lady, forty-seven years old, suffered in her youth from very obstinate chlorosis, accompanied by very acute neuralgic pain, which varied very much in its seat. For some years past, she has had very mobile rheumatoid pains, attacking sometimes the limbs, and at other times the viscera, and curious nervous disorders, which might be called hypochondriasis, if this lady were not a person of very great sense. I may add that her health is excellent, so far as the functions of organic life are concerned. For the last two years, she has noticed that, when she goes up a staircase pretty quickly, she is suddenly seized with an acute pain behind the sternum, rapidly extending to the left shoulder and arm, and causing trifling numbness. On her stopping, the sensations disappear in less than a minute. I have examined her heart and her lungs with the greatest care, I might say, with the most devoted solicitude, on several occasions, immediately after she had just had one of these seizures, and never at any time have I discovered in the heart's rhythm, in the valvular sounds in the region of the aorta, or in the lungs, the least sign, the least phenomenon, different from what is found in health, with the exception of some marked acceleration of the heart's action.

Quite recently, when I intended to speak to you of angina pectoris, I was consulted by a gentleman, aged forty-five, who had all the appearances of the most flourishing health. He took more than ten minutes to come up to my door, and, when in the ante-room, he dropped on a bench, looking pale, and in a condition which frightened my servant. A few minutes sufficed to make him right again.

When, half an hour afterwards, he came into my consulting-room, I could never have suspected from his blooming appearance what had so lately occurred. He then told me that fifteen years ago he had had a very bad attack of syphilis, of which he had not been well cured. Three years afterwards, he had a very violent and obstinate attack of sciatica, and, subsequently, pains in the limbs, of which he was cured, after many unsuccessful treatments, by iodide of potassium. Later, again, he had had an attack of gout in the big toe. He had never passed any gravel, and there was no history of gout in his family antecedents.

The angina pectoris had begun a year previously. The attack was very slight, and only recurred when he took any violent exercise, at rare intervals; in a short time, less active causes sufficed to bring on a paroxysm, which recurred at more frequent intervals. For some months, for the last month particularly, his life had become unbearable. If he happened to walk up the least ascent, he was instantly seized with pain, and was compelled to stop. He had just come from Lyons on the day when he consulted me. He had travelled all night, and as he came out of the railway carriage, he had to walk a few steps about the station to get a cab. Although he walked quietly, he was seized so violently that he had a kind of fainting fit, and was obliged to sit down in the mud. His travelling companions put him upon his legs again. The pain which he felt was excruciating; it began behind the sternum, nearly on a level with the fourth and fifth ribs, somewhat about the region of the heart, which beat violently during the attack. It extended from there to the root of the neck, and to both arms equally, causing a painful sensation of numbness as far as the tips of his fingers. He fancied that his hands swelled a little at such times. He was then obliged to stop short, and to keep his chest motionless, dreading to draw in his breath lest he should increase the fearful constriction which crushed his chest. When the pain was more intense, he was seized with vertigo, and fell into a state almost like syncope.

The emotion which my examination caused him, and the movements which he made to take off, and afterwards put on his clothes, sufficed to bring on a slight paroxysm.

It would certainly be difficult to meet with a more marked case; and I confess that I felt sure I would find some grave lesion of the heart, or of the great vessels. But on the most searching examination, I detected no abnormal condition of the intrathoracic organs. And as I have already met with a good many cases of this kind in the course of my career, and as I have seen persons as gravely affected as this gentleman was get perfectly well, I must of necessity admit that angina pectoris, even when most intense, need not be a symptom of an organic lesion. Presently, when I come to speak of treatment, I will relate to you two cases of cure, one in a patient of Dr. Duchenne (of Boulogne), and the other in a patient of Dr. Aran. These will show you still more conclusively that angina pectoris may be only an idiopathic neuralgia, in the sense usually meant by this term.

A case, however, recently came under my notice and that of my esteemed friend, Dr. Marx, which shows that one should be very careful before affirming that no organic lesions exist. An ex-bill-broker on the Paris Bourse, who had been formerly subject to very severe hepatic colic, which had left him for several years, began to complain of choking sensations, which came on suddenly whenever he took a little more active exercise than usual. The sensation of choking was accompanied by an acute pain behind the sternum, radiating to the left shoulder and arm. There was no habitual dyspnœa, and nothing could excite the suspicion that the angina pectoris was a symptom of an organic lesion. But auscultation afterwards detected the presence of an aneurism of the arch of the aorta, which increased rapidly, and from that time there came on habitual orthopnœa, and paroxysms of angina pectoris recurred on the patient making the slightest movement. Dr. Marx had one day spent a few moments with him, encouraging and consoling him, and had been accompanied by him on his going away as far as the bedroom door; but the doctor had no sooner got to the bottom of the stairs than he was hastily summoned by the patient's servant. On going up again in all haste, he found a corpse. The aneurism had suddenly burst into the trachea, and had caused fatal hæmoptysis.

In the month of September, 1865, I was consulted by a patient sent to me by Dr. Lefebvre, of Roubaix, and suffering from angina pectoris. The complaint had set in suddenly about the middle of the preceding year, during an after-dinner walk, and the paroxysms had recurred several days in succession. They disappeared for some time, and then returned with greater intensity than ever, *at the same hour* invariably. They soon ceased from being periodic, and recurred under the influence of the slightest effort, or during sleep, on the patient starting up. At last, symptoms of a serious hypertrophy of the heart, with lesions of the ventricles, showed themselves. I will, therefore, willingly admit that, in some cases, even though the most careful examination will not be able to detect anything in the aorta or in the mediastinum, there are lesions present which become manifest at a later period. The same thing occurs in the case of angina pectoris as in that of some obstinate intercostal neuralgias, the organic cause of which has been long overlooked, although it does not follow from this that even the most obstinate intercostal neuralgias are always symptomatic.

When I first began practice, I attended for several years a gentleman whose complaint I did not for a long time recognize, and whose case taught me a lesson which I have never forgotten. He was sixty years of age, and enjoyed excellent health. Two of his brothers had died a sudden death, and in one of them the cause was found to be rupture of an aneurism.

For some years past, this gentleman complained of a violent pain about the base of his chest, in the course of the intercostal nerves; the pain was most intense in front, and where it was so, the skin was also slightly benumbed. It sometimes left the chest, and spread to the sides of the neck and head, where it simulated a neuralgia.

The symptoms were not constant, but returned at uncertain intervals. All the medical men whom the patient had consulted, and I among the rest, thought that the case was one of rheumatic neuralgia. After a few years, the pain became almost continuous, although it was very bearable. When the patient tried to walk, however, it became so fearfully intensified that he was compelled to remain almost motionless. Rest made everything right, as is the case in angina pectoris; but he often could find no relief except by lying flat on his stomach on a couch. He tried I know not how many plans of treatment. His great wealth allowed him to consult the most eminent practitioners, and to spend two or three months every year at various mineral springs. At last, he complained to me one day of a queer throbbing sensation in the back, on a level with the seventh and eighth ribs on the left side. On laying my hand over that part, I felt an impulse isochronous with the heart's beat. From that time, percussion and auscultation settled all doubt about the existence of aneurism of the aorta. The disease made rapid progress; four ribs became eroded after a time, and a tumor of the size of a child's head showed itself under the skin. I need not add that the case terminated as such cases always do; the aneurism destroyed the skin, and burst suddenly outside.

A few years ago, I saw with my colleague, Mr. Richet, a merchant who was exactly in the same state. He complained of pain in the base of the chest, which recurred in paroxysms, and if, instead of following the course of the intercostal nerves, this pain had been seated in the nerves which are usually affected in angina pectoris, it would have been confounded with this affection. It was for a long time ascribed to rheumatism, and the most varied and the most energetic treatment was vainly tried. At last, after several years had elapsed, stethoscopic examinations, which until then had revealed nothing, enabled us to recognize the existence of an aneurism of the thoracic aorta. I at once foresaw the issue of the case, and indeed, death occurred suddenly a few months afterwards, during the night. The patient was residing at the time at Saint-Germain-en-Laye, and my excellent friend, Dr. Lepiez, who made the autopsy, ascertained that the aneurism had burst into the pleural cavity.

The close relation between these symptomatic neuralgias, the history of which I have just related, and angina pectoris, is sufficiently evident.

Besides, if we study neuralgias of other regions, we shall find that they pretty frequently take on this paroxysmal course of angina pectoris.

The frequently perfect periodicity presented by neuralgias due to some grave organic lesion is something very remarkable. I have already related to you the cases of two ladies suffering from carcinoma of the uterus whom I saw with Récamier and with my excellent friend, Dr. Lasègue. In 1862, I saw a third case of the kind, that of a lady with a uterine polypus, whom I attended with Professor Nélaton. In all three the most fearful neuralgic pain recurred every day at the same time, with the regularity of the most typical ague.

Some of you may also remember a man who was at No. 10, in St. Agnes Ward, and who suffered from pains returning every day at the same time, with unspeakable violence, sometimes accompanied with an attack of unilateral eclampsia, after which there remained some hemiplegia. After death, we found cancer of the brain.

I lay so much stress on the perfectly periodic character of neuralgias, due to the gravest organic lesions, because some pathologists have asserted that periodicity, when well marked, was a character distinguishing pure neuroses from neuralgias depending on a grave organic visceral lesion.

In the case of angina pectoris, the periodic recurrence of the attacks by no means, therefore, excludes the idea of an organic affection of the heart, or its valves, or of the great vessels. I admit, and the majority of practitioners do so, that this singular neurosis may be symptomatic; but I admit it merely in this sense, namely, that there is a mere coincidence, and that the organic lesions, whatever they may be, only afford an opportunity for the development of the neurosis which is superadded to them.

I merely advert now to the fact that neurosis may be ingrafted on organic lesions, and be independent of them, since those lesions are persistent, and cannot, therefore, be regarded as the essential condition and the true cause of nervous disturbances which are of a transient character. At some other time, I mean to go more deeply into that question.

What, then, are the causes of angina pectoris? I mean, of course, the *predisposing causes*, the exciting or determining causes being set aside for the time.

Fothergill relates the following case, which has been quoted by Desportes:*

"A man, about thirty years old, of rather small stature, with a short neck, and of a robust constitution, and used to taking moderate and regular exercise, was subject to a complaint of such marked characters that it could not be mistaken for any other. Whenever he walked up a hill, or even whenever he walked a little faster than usual, or if, when riding, he made his horse gallop, he was obliged to stop suddenly, on account of a constricting sensation which he had in his chest, and which, he said, made him fear that he should die if he were obliged to move on. This sensation was felt across the chest, and extended along the arms as far as the elbows; it lasted a pretty long time. Moderate exercise of any kind did not give rise to the sensation. The patient had noticed that he suffered less when he moved about with an empty stomach than when he had taken food. The lungs did not seem to be affected; there had been no cough, no symptoms of inflammation, no bronchitis, no signs of hydrothorax, no transient fit of anger to account for the production of such sensations.

"Fothergill recommended light diet, the bowels to be kept properly open, moderate horse exercise, and the avoidance of long and fatiguing walks. He prescribed a few soap pills, native cinnabar made up into pills with some gum, and a weak bitter tonic with iron for a few months. The patient afterwards took the Bath waters for several seasons. He got perfectly well, and was enjoying good health twenty years afterwards, or at all events, Fothergill had not then heard that he had been attacked with the same pain again."

This case, gentlemen, is quoted as an instance of idiopathic angina pectoris. It would be difficult, indeed, to find another cause for the complaint than a strange predisposition on the part of the patient. Similar cases are not perhaps as rare as might be supposed, and you will doubtless have occasion to meet in your career with individuals who will tell you that they have had similar sensations in various degrees. These neuralgic pains which, starting from the precordial region, and attended with a sensation of constriction of the chest, radiated to the throat, and extended to the arm, have either shown themselves once only or have been transient, and never returned, so that they never felt sufficiently alarmed to apply to a medical man, and it is only by chance that they ever mention them. Yet, as the case which I have just quoted shows, idiopathic angina may, as regards the frequency and intensity of its paroxysms, be exactly like that which is due to more palpable causes.

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* Traité de l'Angine de Poitrine, Paris, 1811.

Among such causes, *rheumatism* and *gout* must be ranked.

Some authors have thought that *angina pectoris* was merely a manifestation of the rheumatic or the gouty diathesis, which settles upon the heart, according to the majority, but, according to others, on the lungs and even on the stomach, the cardiac symptoms occurring then only from sympathy with the gastric disturbance. Without adopting such an exclusive opinion, I think that angina pectoris is in some instances indeed a rheumatic or gouty affection. A retrocession of gout or of rheumatism need not be appealed to, for it is conceivable that this neuralgia may develop itself in the same manner as the other neuralgias from which gouty and rheumatic subjects generally suffer.

The following cases are instances in point:

On February 2, 1861, I was consulted by M. B. de R., a patient of Dr. Maugeret (of Tours). He was sixty years of age; his father had suffered from asthma, and he had himself all the appearance of a gouty subject, and, for the last six years, had had saccharine diabetes. He told me that shortly after he had begun to pass sugar in his urine, he had been seized with angina pectoris, which presented somewhat unusual characters. The paroxysms recurred about one o'clock in the morning, independently of difficult digestion, or, as sometimes happens, of bad dreams. It began with an acute pain in the muscles of the left arm, and radiated from there towards the chest, a little above the heart; it went on increasing progressively for an hour or two, then diminished slowly, and ceased about morning.

These attacks returned several nights in succession, and after disappearing for a few days or a few weeks, they recurred again with the same characters. Although the pain was very acute, it did not prevent him from drawing in a deep breath at will, and he had never had a sensation of imminent suffocation.

During the day he could walk easily on even ground, but if the ground had the slightest ascent, if he went up a staircase pretty quickly, he was obliged to stop, under pain of being compelled to sit down or of falling. These symptoms came on if he took the least exercise after dinner, and increased a little by degrees every year; they were relieved by rubbing in belladonna ointment into the left armpit.

I examined his heart and great vessels with the greatest care, but detected nothing abnormal.

Now, do not these nocturnal attacks of angina pectoris remind you of fits of asthma, at least as regards the evolution of the phenomena?

Two days after I had seen the above patient, on February 4, Mr. T., a former pharmaceutical chemist, sent for me. He was suffering from capillary bronchitis, accompanied with strange pain in the chest, something like angina pectoris, which dated six months back. Thus, from time to time, for several days in succession, he could not take the least exercise without feeling a violent and sudden pain behind the middle of the sternum, with extreme difficulty of breathing. This pain extended immediately to both arms, but was more intense in the left. He felt a little relief only by stopping short, and raising both his hands to his head, when his arms immediately felt benumbed. All was over in about a minute; but the paroxysm lasted longer if the patient did not at once obey an irresistible call to pass water; and if he had four attacks in an hour, he was obliged to pass water four times. He added also that, when the paroxysm was drawing to its close, as when his arms were getting numb, he felt the mucous membrane of his nose get congested.

These very frequent and almost irresistible calls to pass water, which are likewise present in some cases of asthma, do, in my opinion, establish an analogy between this case and angina pectoris.

On July 24, of the same year, I was consulted by a Sicilian gentleman, aged forty-eight, tall and robust, whose father was deaf and dumb, and rather gouty, and whose maternal grandfather had been the subject of the most acute gout. He was habitually dyspeptic, had for many years suffered from cutaneous eruptions, and was subject to headaches. In 1858, he had had a violent attack of gout in the big toe, which he treated by leeches and colchicum, and which disappeared suddenly. In the following year, his dyspepsia became worse, and he soon afterwards had paroxysms of angina pectoris, beginning in the left arm, and extending with rapidity to the heart. The pain and sensation of thoracic constriction were so fearful that he thought he was going to die. These attacks recurred chiefly during the night, and came on during the day if he took the least exercise, rarely lasting more than three minutes. His intellect was unimpaired. After an indifferent treatment, he improved, and was well when he came to Paris; he could walk fast and go briskly up a staircase without feeling anything. I advised him not to interfere with his gout if it ever appeared again, and I enjoined, as I do to all gouty subjects, great regularity and sobriety in his manner of living, and exercise. I meant to try an appropriate treatment when the attacks returned. The heart and great vessels seemed to me to be perfectly healthy.

I saw this patient a month afterwards, and his health was still excellent. He had not had angina pectoris. I examined again his heart and great vessels with the greatest care, but found nothing wrong with them.

Ten days before, on July 14, I had been consulted by a lady, aged fifty-five, who for the last seven or eight years had had several attacks of gout. In the beginning of the year 1862, she had had her first attacks of angina pectoris. The pain began at first in both shoulders, spread rapidly to the tongue, the neck, and then to the arms and chest. It came on if the patient made the slightest movement, or was in the least moved, and it was not accompanied by numbness. The attack seldom lasted one, two, or three minutes, and terminated more quickly when perspiration set in.

On the most careful examination, I found no sign of organic lesion of the heart or of the great vessels.

If we take such cases into account, then, in which gout, as may be at least supposed, had something to do with the occurrence of angina pectoris, it would appear that angina pectoris, like asthma and other neuroses, may be a manifestation of the gouty or the rheumatic diathesis. But there is a predisposing cause, which cannot, I think, be called in question, and which I have already mentioned, although no one has indicated it, namely, *epilepsy*. In some cases, and perhaps in a pretty good number of instances, according to my experience, angina pectoris is an expression of this cruel and fearful complaint, and is a variety of the vertiginous form of the disease, in other words, it is an epileptiform neuralgia. Its invasion is as sudden, its progress as rapid, and its disappearance as sudden, and, as I have already told you, it is not of very uncommon occurrence to find persons who have in former years suffered from angina pectoris become subject afterwards to epileptic fits, just as in other instances angina pectoris has been preceded by well-marked epileptiform seizures. A case of the kind lately again came under my notice.

A gentleman, aged forty-five, who was subject to epileptic fits, had for the last six months suffered from symptoms of which he gave me the following description. Whenever he took exercise of a somewhat violent character, or walked a long distance, he suddenly felt a painful oppression at the chest. For the last month this symptom had recurred three times a day, even when he was quiet, and it had become very severe. He had an acute pain, seated at first in the right half of the chest in front, giving rise to the sensation as if he wore a padded plastron; and after a minute it spread to the corresponding arm, which felt very numb and painful, and was of a higher temperature than the left. These symptoms lasted for a quarter of an hour nearly, and then disappeared entirely.

At the outset, there was an abundant secretion of intestinal gases.

The patient's general health seemed to be excellent. His appetite was good, his digestion regular. He complained of nothing besides the symptoms for which he had come to consult me, and I found no symptom, no sign of organic lesions of the lungs or of the heart and great vessels. I recommended a treatment by belladonna and bicarbonate of soda, of which I shall speak by and by.

In addition to the suddenness of its access, the rapidity of its progress, and its abrupt cessation, angina pectoris presents, in other respects, many points of resemblance to epileptiform neuralgia. When I spoke to you of this latter complaint, I told you that the pain, which constitutes its chief element, is accompanied with congestion of the affected region. The same thing occurs in angina pectoris, as in two cases which I have related to you, and as I shall take care to point out when I come to the description of the symptoms.

I do not think that it has been proved that males are more subject than females to this singular affection. It is certain, however, that angina pectoris almost exclusively attacks individuals above forty or fifty years of age, although it has been met with in young persons. Fothergill's patient was about thirty years old, and Desportes has related the history of an individual, aged twenty-five, in whom, I may add in passing, "the lungs, heart, coronary arteries, great thoracic vessels, or the valves at their cardiac ends, disclosed on examination after death no change whatever, no induration, no ossification." Heberden had also stated that angina pectoris could come on in youth; and Robert Hamilton, that it did not even spare childhood.

Since this neuralgic affection may be the expression of a diathesis, we need not be surprised that the fact has been admitted that it may be *hereditary.* Hamilton relates that a soldier, who had angina pectoris, assured him that it was an hereditary complaint in his family, and that his father, his two brothers, and his sister had suffered from it.

As is the case with all neuroses, the *exciting causes* of angina pectoris are exceedingly numerous and variable. The patient is often seized with a more or less violent paroxysm, without being able to know the reason why; he may be even seized during sleep. This especially occurs when the angina pectoris is merely an epileptic aura.

Some patients state that sudden atmospheric changes bring on the paroxysms, or that they cannot walk, run, or ride, against the wind without being compelled to stop from an attack of the complaint.

The most frequent causes, especially when the angina pectoris is due to an organic lesion of the heart or of the great vessels, are sudden movements, unusually active exercise, as brisk walking, or the act of going up a staircase, or, again, fits of coughing, prolonged speaking, straining at stool. These efforts or these muscular movements need not even be very violent, since, as in the case of the military superintendent whose history I related to you in the beginning of this lecture, the pain came on after the patient had been engaged in putting his signature to many papers.

In some instances, the first seizures come on after some excess in eating or drinking; in many cases, the paroxysms are always more violent after a meal, even when moderate, whether the individual moves about or remains quiet. Jurine has, however, recorded the case of a man whose attacks were most violent and prolonged when he was fasting.

Deep mental emotions, especially fits of anger, are frequent exciting causes of angina pectoris, and they not only bring on a paroxysm, but they increase the intensity of the disease to such a degree as even to cause death.

Such are the circumstances in which the singular affection which has engaged our attention to-day comes on in the majority of instances, although no rule can be laid down concerning it. Their multiplicity shows the essentially nervous nature of the complaint, and this fact will become still more evident from the changeableness of the symptoms.

It almost never happens that angina pectoris is ushered in by premonitory symptoms: its access is sudden. Pain is suddenly felt behind the sternum, accompanied with a sense of constriction and anxiety, generally seated in the left side of the chest, but occasionally in the right side, and it is so intense as to make the patient dread suffocation and syncope, and to deprive him of the power of speech.

It is rarely confined to that part, for in nearly every instance it spreads simultaneously, sometimes along the neck as far as the articulation of the lower jaw, the movements of which are impeded, but more frequently along the pectorales muscles to the shoulder-joint, from which it descends along the inner aspect of the arm as far as the elbow, and down the forearm to the fingers.

The left side is the one generally attacked, as I have told you, but in some cases the right is the side affected, as in the epileptic patient whose history I have related to you. In other cases, instead of ascending to the neck or arm, the pain descends to the epigastrium as far as the groin; in others, again, but very rarely, it is felt in all those regions at the same time.

Its extension to the upper extremity is so constant a phenomenon that some authors, particularly Wall, who described angina pectoris nearly simultaneously with Heberden, have given it as an essential character of the disease.

It has occasionally been known to follow an opposite course, beginning in the arm, and thence quickly spreading to the chest. Do you not find, gentlemen, great analogy between this and what occurs in the *aura epileptica*, and is not this in contradiction to the view that angina pectoris is of necessity caused by a material lesion of the organs contained in the thoracic cavity.

Sometimes, again, this pain is felt in the hand alone, without starting from the chest, and without passing along the nerves of the arm, or taking an ascending course.

On March 29, 1863, I saw, in consultation with Drs. Gruby and Maître, a Russian nobleman suffering from hypertrophy of the heart with systolic bellows-murmur at the apex. He felt from time to time an acute pain in the cardiac region, which disappeared after having been strictly local; then all of a sudden, without any manifestation about the heart, he had in the left hand a pain which he compared to that of cramp, and which was accompanied with numbness. There was no muscular spasm. The pain lasted about a minute, and disappeared without leaving any traces.

Lastly, in some cases, angina pectoris consists in violent palpitation, with numbness of the left arm, without pain. This was the case in a young married lady, aged twenty-two, who consulted me on November 22, 1862. Her grandfather had been gouty, her mother suffered from violent neuralgias, and she herself had been subject to angina pectoris since she was sixteen years old. For the space of four years, she had only had excessively violent palpitation, without any sensation in the arm, but for the last four years the palpitation was accompanied by painless numbness of the left arm, which compelled her to drop whatever she might be holding in her hand. These symptoms recurred whenever she took a little more active exercise than usual. I found no signs of cardiac or valvular lesions.

When pain is present, as unquestionably happens in the great majority of cases, it is not generally increased by making pressure over the affected parts, or by moving the arm into which it extends. Nay, mere pressure may relieve it, and I may again remind you on this point of the patient, whose history I have already related to you, and who used to relieve his throbbing pain by squeezing his back against a piece of furniture.

Although patients suffering from angina pectoris think they are going to be suffocated during a paroxysm, the chest is normally resonant on percussion, and if it be auscülted as they draw in breath again, vesicular breathing is heard everywhere. This is far from being the case in fits of dyspnœa.

Should the patient assume any peculiar attitude, it is on account of the pain, and not from distress of breathing. Very varied attitudes are assumed: one patient will lie motionless on his back; another will incline backwards on the back of his chair, or on his pillows; a third will place himself on all fours, resting on his knees and elbows; while a fourth may stoop so much as to bend in two.

During a paroxysm, the face turns pale, and soon afterwards becomes more or less red; this congestion, which I have compared to what occurs in epileptic fits, occurs also in other parts which are painful. Thus, in the woman in St. Bernard Ward, I noted that the skin of the left hand, which was very painful, became at first extremely pale, and then of a livid bluish hue. Sometimes, also, the face and limbs are bedewed with perspiration.

The intellect is, in general, unimpaired all the time, although some exceptional instances have been recorded of individuals who had a wandering look, and who muttered unintelligible words, as if in a state of ecstasy. Lastly, there is sometimes loss of consciousness, as the pain goes off, especially if the angina pectoris be an aura epileptica, although the intensity of the pain, and perhaps some great disturbance in the heart's action, may bring on syncope of a very different character from the loss of consciousness which accompanies the epileptic form of angina pectoris.

When the disease manifests itself for the first time, the paroxysms are transient, and last scarcely a minute or two; but when it is of old date, the attacks may last several hours, and even several days, with exacerbations.

The attack terminates as suddenly as it began, but the patient retains for some little time longer a sensation of numbness in the regions which have been the seat of pain. If the paroxysms have been frequent and violent from the disease having reached a great degree of intensity, there remains for a longer or shorter period trembling and weakness either of the whole body or of the affected limb only, which may persist until another paroxysm takes place. An individual may have only one attack of angina pectoris, and be rid of it forever. Such cases are very rare, and it is doubtful whether the diagnosis was then very accurate. In the majority of instances, several paroxysms follow one another, at more or less distant intervals, after years, twelve, six, or three months, or weeks, the intervals becoming shorter in proportion as the lesion which gives rise to this complaint makes progress. We have seen that the paroxysms may return periodically. In the intervals, the person apparently enjoys perfect health, unless, of course, the angina pectoris be due to the presence of an organic affection, as some disease of the heart or great vessels, to which the general condition is subordinate. From what I have told you of its coexisting with organic lesions, in perhaps the majority of instances, and of its being one of the manifestations of the vertiginous form of epilepsy, it evidently follows that angina pectoris is a most serious complaint, as being a symptom of diseases which sooner or later terminate in death. Although, from its nature, idiopathic, rheumatic, or gouty angina pectoris admits of a less severe *prognosis*, this should in all cases be extremely reserved. For there are occasional though rare cases on record of individuals who have died during a paroxysm, and I may mention, among other instances, that of John Hunter, who died suddenly after a fit of anger which caused the recurrence of angina pectoris to which he had been subject for eight years.

The disease may terminate fatally shortly after the manifestation of the first paroxysms, or the patient may live for many years, whether the attacks recur at nearer intervals and increase in intensity, as is generally the case when the angina pectoris is symptomatic of a cardiac affection, or is the expression of epilepsy, or whether they recur at distant intervals only, decreasing in intensity or persisting to a less degree.

The disease is curable when it is not under the dependence of an appreciable cause, or when it is due to a rheumatic or gouty diathesis; and this happy result may be especially expected when the patient is young, and still more when the seizures have been of moderate intensity. The complaint is almost unavoidably fatal when it is hereditary.

The violence of the seizures, the facility with which they recur under the influence of exciting causes, necessarily increase the gravity of the prognosis; and hence may be inferred an important rule for treatment, namely, that these causes should be avoided, and, above all, mental emotions, which are perhaps the most powerful of all.

The extreme variability of the phenomena which characterize angina pectoris often render its *diagnosis* very uncertain, and it is not surprising that very different conditions have been confounded with it. As Wichmann remarked, twenty-five years after Heberden, an individual need only complain of anxiety, and of a sense of constriction about the chest, even of impeded breathing, for its being immediately ascribed to angina pectoris.

Thus, pleurodynia of the precordial region, which set in suddenly, and temporarily impeded respiration, disappearing rapidly, has been mistaken for angina pectoris. The pain, in such cases, is more superficial than that of angina pectoris, and does not, like it, shoot beyond the part which it attacked in the first instance. It is seated in the pectorales muscles, and is relieved and even removed by taking in a deep breath, prolonged for awhile, and by making pressure on the affected part. Lastly, it is not lancinating, and is not accompanied by a sense of anxiety, and is not followed by a feeling of numbness like angina pectoris.

Thoracic and cervico-brachial neuralgias are distinguishable from angina pectoris by the pain being felt only in the course of the diseased nerves, and by its being continuous, although it recurs in paroxysms, and by its obstinate persistence for a more or less prolonged period. Its accession and disappearance have not, therefore, the suddenness which characterizes angina pectoris.

The diagnosis is made with difficulty when an individual afflicted with aneurism of the aorta suffers from sternal pains, shooting towards the shoulder, and accompanied by a sense of choking which, from its growing worse at times, might lead one into error. But, even then, these pains do not recur in very distinct paroxysms; they are continuous, or, at least, never cease spontaneously. The same remark applies to the pungent, lancinating, and excruciating pain, attended with oppression at the chest, which occasionally supervenes in pericarditis.

In conclusion, in spite of their extreme diversity, the characters of angina pectoris are such that it seems to me difficult to mistake them.

As a rule, I know nothing so difficult as the treatment of nervous disorders. Neuroses are not only capricious in respect of their etiological conditions and of their symptomatic manifestations, but of their amenability to treatment also. Some patients get well after the use of remedies which fail in others, and a treatment which has proved unsuccessful in one case is sometimes followed by the best results in instances apparently perfectly similar. The very variability of their manifestations, the suddenness of their invasion without any appreciable cause, and their oft-unexpected abrupt cessation, frequently throw doubt on the real utility of our interference. This is especially the case in angina pectoris.

The paroxysms are often of such short duration, and generally terminate so suddenly, that their disappearance can hardly be ascribed to the influence of treatment. If they have been brought on by somewhat active exercise, as a brisk walk, or by running, the patient need only stop still to cause the phenomena to pass off, although cases have been recorded in which individuals have, in defiance of the pain they felt, continued to walk, and have got rid of it. Some persons have been able to stop the paroxysm by forcibly holding their breath. I have already alluded to Jurine's patient, who had most violent seizures when fasting, which recurred in great frequency unless he immediately took some food.

Although we can with difficulty appreciate in a just measure the utility of a particular remedy, we can judge of the opportunity of certain modes of treatment by taking into consideration the nature of the symptoms which we are anxious to remove. Now, to begin with, there is a measure which I must distinctly reject as inapplicable to a complaint like angina pectoris, in which there is such an imminent risk of syncope, I mean *bloodletting*. Although it has been recommended by eminent physicians, and even by Laennec, it seems to me to be, to say the least, irrational, whether the blood be removed by opening a vein at the elbow, or by applying leeches to the epigastrium or to the precordial region.

Emetics, antimony, in particular, have been vaunted in the violent seizures, but they seem to me to be contraindicated, on account of their lowering influence on the system.

Diffusible stimulants, ethereal preparations, ammonia in small doses, alcoholic infusions of mint, are much more indicated during a seizure than opium and other narcotics which have been lauded as so beneficial.

When the paroxysm is prolonged, and there is a marked tendency to syncope, the effects of these remedies taken internally may be aided by the use of stimulating alcoholic or ammoniacal liniments, and by dipping the hands or feet in hot water in which mustard has been dissolved.

But we should particularly endeavor to avert the paroxysms, and not combat them when they recur.

A great many methods of treatment have been in turn adopted and laid aside, some of which were altogether empirical, and others founded on the various opinions held with regard to the nature of angina pectoris. Narcotics, *opium*, or its active principles, solanaceous preparations, lactuca virosa (the latter vaunted by Schelinger, of Frankfort), headed the list.

I recollect a patient afflicted with very severe angina pectoris, recurring in paroxysms several times a day with alarming violence, who improved rapidly, and obtained what he termed *a cure* by the use of frictions, made several times a day, over the sternum, with a liniment of stramonium. Hypodermic injections of atropia, about the starting-point of the pain, and in the neck and armpit, in some cases retard the seizures, diminish their violence, and ultimately cure, especially if the neuralgia be not dependent upon an organic affection of the heart or aorta.

On the hypothesis, admitted by some, as I have told you, that angina pectoris is due to ossification of the coronary arteries, *phosphoric acid* has been recommended with the view of preventing and even of removing these ossifications. I need not add that this absurd idea could only occur to a chemist, who should have studied physiology and medicine before dabbling in therapeutics.

Bretonneau, whose practical sense had not been stultified by the most extensive chemical knowledge, and who in proportion as he grew older in practice openly confessed the deplorable errors which chemistry had led him to commit, and the little assistance he had derived from it in therapeutics, Bretonneau was yet led by a chemical theory to adopt a useful treatment in angina pectoris.

Although he was successful, I have often heard him laugh at the theory which he had imagined, and express surprise that, for once in his life, chemistry, the favorite science of his youth, had helped him to do some good in therapeutics.

This is the manner in which the illustrious Tours physician was led, as he stated himself, to adopt his peculiar treatment of angina pectoris. He, of course, thought that the complaint was due to calcareous concretions of the first part of the aorta. "On being consulted by a person afflicted with angina pectoris, I asked myself whether he would not derive some benefit from the prolonged use of *bicarbonate of soda*, from which such marvellous results are often obtained in the calculous diathesis. There was such a great difference, however, between the concretions proper to angina pectoris and urinary calculi, that it was very doubtful whether any good results would be obtained, even by persevering with the greatest docility and patience in a treatment based on such vague reasons. I felt, therefore, more pained than surprised when, after this treatment had been tried for two months, I ascertained that no favorable change had followed.

"Yet from that time it became evident that, if the complaint had not yielded, it had not been aggravated; then it manifestly improved, and after the patient had made use, for six months, of the artificial Vichy water, he got rid entirely of all his symptoms of angina pectoris."

Thus, happily helped by chance, Bretonneau often repeated the experiment after that time, and treated several cases successfully. The bicarbonate of soda is given after a peculiar method. First, in doses of two scruples; one scruple before each of the two principal meals; and this quantity is to be gradually increased, if it be well borne by the patient, to eight and even ten scruples a day, the patient taking from two to two and a half scruples half an hour before and immediately after each of the two principal meals in the day. Bretonneau recommended to increase the dose gradually for ten days, and then by degrees to diminish it for the next ten days. The treatment is then suspended for the space of fifteen or twenty days, after which it is resumed, and is continued in the same manner for more than a year, to be again followed after an interruption of several months.

Bretonneau combined belladonna with the carbonate of soda, and administered it according to certain rules also. He prescribed pills, consisting of one-tenth of a grain each of extract and of powdered root of belladonna.

The patient takes in the beginning one of these pills in the morning, a quarter of an hour before his first meal, and does so for three days running. For the next ten days, he takes two at the same time in the day, and at once. For twenty days, three, always at once. If no improvement follows, the dose is increased to four pills, and should the paroxysms recur with the same violence and frequency, the dose of the medicine is increased by one-fifth of a grain every ten days, unless there supervene unpleasant dryness of the mouth, marked disturbance of vision, accompanied by a very striking dilatation of the pupils, showing that the too rapid increase of the dose has produced effects which should be guarded against. Whenever, therefore, a progressive amelioration has been obtained before this rapid increase in the dose of the medicine has commenced, it should not be increased again; and it is only when the improvement obtained seems to diminish that the daily quantity administered should be raised by one-fifth of a grain.

Belladonna should be persevered in during the intervals when the bicarbonate of soda is stopped.

This treatment is, of course, beneficial in those cases only when there is no vascular lesion, precisely in cases the very opposite of those suggested to Bretonneau by his chemical theory.

This treatment of angina pectoris by belladonna is, as you may see, exactly the same as the one which I recommend in epilepsy. And you need not be surprised at this, for, as I have told you, angina pectoris, in many cases, is only an epileptiform neuralgia, or a kind of aura epileptica.

For the same reason, you will understand how cases have been recorded in which angina pectoris had been successfully treated by *nitrate of silver*, which has also been lauded in epilepsy.

I shall not enumerate to you all the remedies that have been vaunted in this complaint, but will merely mention that Alexander, quoted by Harles in his monograph on the use of *arsenic* in medecine, has related the history of a man, aged fifty-seven, who got rid of a very severe angina pectoris by taking six drops three times a day of Fowler's solution.

I do not dwell on the hygienic rules to follow, because it is above all things clear that the patient must avoid all causes capable of bringing on a paroxysm. That he should take exercise in moderation, should observe perfect rest of mind, and avoid all deep mental emotion, are precepts the necessity of which is self-evident.

I will not bring this lecture to a close without speaking of the use of electricity, which holds a very important place in the treatment of angina pectoris. We are indebted to Dr. Duchenne (of Boulogne) for the methodical employment of this therapeutic agent, which is sometimes so powerful.*

I will, with your permission, read to you a case published by Dr. Duchenne. It affords another proof that the most violent angina pectoris may not be due to an organic lesion of the heart or the great vessels, because, had such a lesion been present, electricity might have relieved the pain, but could never have cured the patient, particularly in such a short time.

"Pérone, aged fifty, a currier, residing at Belleville, 25 Tourtil Street, of a stout build and sanguine temperament, rather fat and with a short neck. He has never had any serious illness. Two years ago, he had some rheumatic pains in the right shoulder, which compelled him to interrupt his work for a month, although he had no fever. He is not generally afflicted with short breath, and is not subject to palpitation. He lives in a healthy place, and his apartments are not damp.

"On November 29, 1852, at nine o'clock in the morning, before breakfast, he suddenly felt, without any known cause, a deep burning sensation in the upper and middle regions of the chest, and a pain which extended into the left upper extremity.

"He had at the same time a sensation of tingling, which went on increasing from the elbow as far as the tips of the fingers. During this attack, his heart beat with force and rapidity, his head felt heavy, was rather painful, and he spoke with difficulty from insufficient respiration, and the attempt increased the pain. He was compelled to stoop forward, to keep still or to sit down, as his pain was worse when he held himself erect; he felt extreme anxiety, and had a dread of impending death. This first attack began to diminish only eighteen hours after it had set in, after a copious bleeding. Mustard foot-baths, sedatives taken internally, and a warm bath had been tried at first, but without any good results. The improvement obtained was not very great, because the patient was compelled to observe the most complete rest, in the sitting posture, as a fresh paroxysm followed any attempt at lying down. The seizures recurred for the slightest cause; sneezing, yawning, or the least emotion, was sufficient to bring one on. During the day, he was perfectly calm, except when he had a paroxysm, lasting about eight or ten minutes, as severe as the former ones, and brought on by his attempting to move, or by any emotion. He could not sleep. By degrees the seizures became less frequent, although they continued as violent as before, and frightened both the patient and those about him.

"There was no disturbance of the appetite and of digestion; and there never was any fever during the course of the disease. A fortnight after the accession of the complaint, frictions with tartar emetic ointment were made over the front of the chest; a purgative was administered every fourth day, and twenty leeches applied to the arms. In spite of this treatment, the paroxysms returned whenever the patient took the least exercise, so that he was compelled to remain perfectly quiet. Dr. Mongeal, his medical attendant, on seeing this condition continue, decided on sending the patient to me, with the idea that contraction of the diaphragm might be at the bottom of the complaint, which he justly termed *angina pectoris*, in the letter which he wrote to me.

"I noted the following circumstances when Pérone consulted me on April 28, 1853.

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* De l'électrisation localisée et de son application à la pathologie et à la thérapeutique; 2d edition, Paris, 1861.

"He rode from Belleville to my house, and when coming up to my apartment, which is on the second floor, he was obliged to stop on every stair, on account of a sense of constriction in his chest and of the other phenomena which I have described above. He became perfectly quiet after resting for a quarter of an hour; on auscultation and percussion, nothing wrong was found with the bronchi, lungs, heart, or great vessels: the pulse was normal. Pressure made over different parts of the chest gave no pain.

"I then asked him to bring on a paroxysm, which he could do by stooping as if he wanted to pick up something. The following phenomena then manifested themselves simultaneously: a very acute deep burning pain, with sensation of constriction, was set up in the upper part of the sternum, shooting into the left upper extremity, running along the posterior aspect of the arm, and the outer of the forearm, and terminating in the index finger; numbness and formication were felt in the whole of the limb. The patient held all the time both his hands folded on the upper part of the chest, compressing it as if to relieve the pain. His head was bent forwards, his shoulders drawn upwards and forwards through the contraction of the pectoralis major and part of the trapezius: the pain became worse whenever he tried to stand erect, or put his shoulders back. On my asking him to walk, he had no sooner taken a couple of steps than he was obliged to stop and sit down, on account of the increased intensity of the pain behind his sternum. His breathing was short and agitated, his heart beat violently, his pulse was frequent, his face red and injected, his eyes opened wide, his body covered with a profuse clammy sweat, and his physiognomy expressive of extreme anxiety. His respiratory sounds were perfectly pure, however, and the valvular clicks well marked. The heart was of normal size, and percussion of the thoracic walls disclosed no abnormal dulness.

"When he tried to speak, his voice was broken and weak, and came out with difficulty, and his pain was increased.

"There was perfect isochronism between the movements of the chest-walls and of the abdomen during respiration; no pain was felt at the base of the chest, and there was no impairment of the voluntary movements; there was only numbness of the left arm and hand, the movements of which were weaker.

"After resting for eight or ten minutes, he became calm again, but it was only by degrees that the pain and constriction of the chest disappeared.

"*Report of the Experiments and their Results.*—I brought on a second paroxysm by making Pérone walk, and I applied to his nipple the extremity of my induction-apparatus graduated to maximum intensity and working with very rapid intermissions. As the nipple was galvanized, he uttered such a loud shriek that I had to interrupt the current. The pain had been excruciating, but merely instantaneous, and to my great surprise, after the artificial pain which I had brought on, the pain of the angina also disappeared completely, as well as the sensations of numbness and formication which accompanied it; respiration had become quiet again; in a word, the patient felt at once in his normal condition.

"The question arose whether this sudden transition was the result of a mere coincidence, or whether it was due to the immense and instantaneous perturbation produced by galvanization of the nipple. In order to determine the point, I had only to repeat the experiment. But it was not so easy as before to bring on a fresh paroxysm, for the patient had to go through various movements for four or five minutes, in order to do this, while, before the galvanization, he had merely to stoop.

"The second experiment was as rapidly successful as the first; but instead of acting on the nipple, I this time galvanized the skin of the painful part (the upper region of the sternum). I took a sort of pleasure in thus arresting a complaint, hitherto held to be beyond relief during a paroxysm, and repeated the experiment several times with the same success, and I noticed that the more I repeated it the greater was the difficulty experienced by the patient in bringing on a paroxysm, so much so that he was only able to effect it on the last occasion by rapidly walking from the bottom of the staircase to my second floor.

"On the next day, he informed me that he had been able to go back to Belleville, without feeling the least uneasiness, or having to stop, and that for the first time since the accession of his complaint, he had been able to sleep. In the morning only, he had had a sensation of constriction, unattended with pain, in the upper part of the chest; he came to me from Belleville on foot, and walked upstairs to my room without stopping or feeling the least uneasiness. In a word, he thought that he was cured.

"I suggested to him to bring back a paroxysm of angina so as to go through the same process as on the previous day. He began at once, but it was only after nearly a quarter of an hour and after making violent efforts, as when he is engaged in currying leather, that he succeeded in bringing on a paroxysm, almost as violent as the former ones. But in two or three seconds, this was again arrested by faradization of the skin.

"From that day the post-sternal pain, the sensations of formication and numbness in the left upper limb, disappeared, and could never be brought on again. A sense of oppression was only brought on at such times, a sort of pressure over that part of the chest which was previously painful. Faradization of the skin, practiced four or five times at pretty distant intervals, removed these last symptoms, and a fortnight after the treatment had been commenced, I could allow Pérone to resume his occupation as a currier.

"A year has now elapsed since he has resumed his laborious occupation, but his angina has not returned."

Another case, which adds value to the above, was communicated to me by Aran, and I will give you the principal features of it:

Mrs. X., aged thirty-two, of middle stature, stated that ten years previously she fell into a kind of lethargic state, which lasted seven days, in consequence of intense grief on losing one of her children. (During that time she was known to continue to breathe by holding a looking-glass before her mouth.)

This condition came to an end on her shedding tears abundantly, but for seven months afterwards, she suffered from palpitation of the heart, with extreme anxiety, difficulty of breathing, and impairment of the intellect. She had got better, in spite of the persistent palpitation, when two years ago (in 1851) deep grief, in consequence of some reverses of fortune, brought on a new series of morbid phenomena, differing from the previous one with regard to their characters, their course, and intensity. Thus, her new complaint came on in more or less frequent paroxysms, in the intervals of which she was free from it.

These were the chief symptoms of the paroxysm: acute precordial pain, compared by the patient to burning heat; very marked post-sternal constriction, with pain shooting into the left arm, and attended with numbness, which lasted for some time after the attack, and with complete paralysis of the limb; extreme anxiety and a terrified aspect. Contraction of the pectorales and of the muscles which flex the head forwards; exaggeration of the pain on making the least attempt to hold up the head and draw back the shoulders, no dyspnœa, as during a fit of asthma, but short and frequent breathing. The attacks were not accompanied by hysterical symptoms; thus, there was no sense of constriction of the throat, no tears, although these could be easily brought on by talking to her of her dead child, and then she loses her reason. Lastly, on auscultation and percussion, no lesion can be made out in the lungs, the bronchi, the heart, or the great bloodvessels.

Dr. Aran had been for a long time trying to cure this condition, but without success, when Dr. Duchenne told him of the important case which I have related to you. As you may imagine, so distinguished a physician as Aran did not allow the opportunity to pass of testing a method of treatment which had answered so well in an analogous case, especially as his patient was in danger of her life. Faradization of the skin was had recourse to during the paroxysms, and the result was as happy and immediate as in Dr. Duchenne's case, so much so indeed that she was almost completely cured of her angina pectoris, and was enabled to resume her ordinary occupation.

LECTURE LVIII.

EXOPHTHALMIC GOITRE, OR GRAVES'S DISEASE.

The Chief Symptoms of the Disease are Three in Number: Hypertrophy of the Thyroid Gland, Exophthalmos, and Palpitation.—The Disease may be Incompletely Developed.—Nervous Phenomena frequent.—Nature of the Affection.—It is probably a Neurosis of the Sympathetic.—Cases and Arguments in favor of this View.—Good Results of Hydropathy.

GENTLEMEN: You may have noticed at No. 34, in St. Bernard Ward, a young woman who has a somewhat strange physiognomy. Her face has a savage expression, her eyeballs are prominent, and her complexion pale. She complains of palpitation of the heart; her pulse at the wrist is frequent, regular, and of normal volume and resistance. Her breathing seems to be impeded, and you could see that her thyroid gland was considerably hypertrophied. The coexistence of these three pathological phenomena—palpitation, hypertrophy of the thyroid gland, and prominence of the eyeballs—constitutes a morbid entity of which you will find numerous instances on record, and which has been designated under the names exophthalmic goitre, exophthalmic cachexia, cachectic exophthalmus, Basedow's disease, &c.

Although ophthalmologists, like Demours, Mackenzie, Sichel, and Desmarres, had already mentioned this complaint, which is so remarkable from its three prominent symptoms, Graves was the one who called attention to it, and afterwards, from Basedow giving a fuller description of it, it was known after his name. Hence, Dr. Hirsch, who perhaps did not know Graves's researches on the subject, has of late years claimed for this complaint the designation of *Basedow's disease*.

In some clinical lectures which I delivered in November, 1860, I mentioned to you, on the authority of Stokes, that the credit of priority belonged to Graves in a great measure; and those of you who may wish for more proofs need only refer to the "Lectures on Clinical Medicine," published by the illustrious Dublin professor, and the chapter on Exophthalmic Goitre in Dr. Stokes's work on "Diseases of the Heart." I leave to the professor of systematic medicine the task of giving you an historical notice of exophthalmic goitre, and of doing justice to the authors who were the first to investigate the subject. As to myself, I mean in this lecture to give you the clinical history of this singular and interesting complaint, to relate a few instances of it, so as to show it to you under different aspects, and to discuss its nature by the light of cases of the disease, which have now been published in pretty large number, and the value of which may be easily tested by fresh observations. I mean particularly to draw your attention to the treatment which has seemed to me the best, which is indicated by the very nature of the complaint, the only serious basis of all treatment, when we have not specifics at command, or cannot have recourse to methods approved by empiricism.

Many individuals suffering from exophthalmic goitre will come and consult you on account of palpitation, but you will be at once struck with their strange look and their prominent eyes. The prominence of both eyeballs should immediately point to your diagnosis. On inquiry, you will find that the exophthalmos is of old date, that it increased by degrees, but that it sometimes does so to such a degree that the patient is afraid lest his eyes should fall out; he has a sensation as if these organs were going to drop out of their sockets, and experiences a difficulty in closing his eyelids completely, and his eyeballs are often partially uncovered during sleep. In a young woman at Clermont, whose case was published by Dr. Pain, the eyeballs were pushed forward so much that one of them actually came out of the orbit, and had to be put back with the fingers.

The exophthalmos is most marked under the influence of mental emotion, and at the menstrual periods. The coats of the eye generally present no alteration, and I have never noticed ulceration of the cornea.

When there is considerable prominence of the eyeballs, the anterior insertions of the recti muscles can be easily seen, and the spot is remarkable for its great vascularity.

There is generally no disturbance of vision, although the patient may become either long or shortsighted; frequently, however, the eye retains to a great degree the power of adapting itself to distances. I knew a man who could read at very variable distances, while the eyeball and the pupil underwent certain modifications; thus, he had convergent strabismus and dilatation of the pupils when the object was held near his eyes, while the eyes recovered their normal position and the pupils contracted when the object was at a distance. It could be seen that, according to the difference in the position of the object, adaptation cost an effort, for there was a greater secretion of tears, which first increased the brilliancy of the eyes, and then dropped on the lower lid. Two patients complained of weakness of sight, and of occasional muscæ volitantes. I have never met with diplopia.

The double exophthalmos and the temporary disturbances of vision naturally attracted the attention of oculists, and interesting ophthalmoscopic observations have therefore been published. A very good summary was published in the Danish language by Dr. Withuisen, of the appearances found on ophthalmoscopic examination of the eye in a case of exophthalmic cachexia: "The ocular media were very transparent, and the retina was of a marked red color from a bright injection of its vessels.

The point of entry of the optic nerve was of a yellowish-red tint, perfectly different from its normal one; the branches of the arteria centralis retinæ were more largely developed than usual, but did not pulsate. On each side of the optic papilla pigment was deposited in semilunar masses almost black in hue. These masses had a concave and distinct edge on the side turned to the papilla, but their other margin was convex and toothed. In both eyes, these masses were of larger size on the outer side."

This case is of considerable interest, and this is increased by the fact that an ophthalmoscopic examination was made during convalescence. It was then found that the hyperæmia of the fundus oculi was less, in fact, that the congestive appearances previously noted had nearly disappeared, but the masses of pigment had undergone no change. Other observers have also noted congestion of the retinal vessels without alteration of the ocular media.

To give this case of Dr. Withuisen its full value, I wish to state positively that it was observed with great care, and that it was an undoubted instance of Graves's disease, for "it had been ascertained, on making an external examination of the eye, that there were double exophthalmos and varicose dilatation of the vessels of the conjunctiva about the insertion of the recti muscles. The cornea was of normal convexity, but the pupil was dilated and the iris slow to contract. The anterior chamber was flatter than natural, probably in consequence of the prolapse of the iris. The patient had a somewhat strange look, expressive of surprise. She complained of being shortsighted, and of having a difficulty in looking steadfastly at the same object for some little time. There were occasional vertigo, and frequent pain in the eyes, with headache, and when she closed her eyes, she sometimes saw circles of fire."

I have quoted this case almost in full, because the eyes were examined with great care. It was thus made out that the membranes of the eye may be the seat of great injection, and of a modification of nutrition, giving rise to deposits of pigment and of a yellowish material on the retina. When I come to speak of the pathological anatomy of the disease, I will tell you the other alterations or modifications which dissection has shown, and we shall then possess all necessary elements for discussing the mechanical cause of exophthalmos and the nature of the functional modifications of the organ of vision.

You must not think that there always is very considerable prolapsus of the eyeball; in some cases, you must admit it on the authority of the people about the patient, unless you have known him yourself previously. Even when the eyes are not strikingly prominent, they have always a special look in them, and there is some transient or lasting disturbance of vision which indicates an abnormal condition of the eyes.

But if the prominence of the eyeballs may escape notice there are two other pathological facts which attract attention forcibly in nearly every case, namely, hypertrophy of the thyroid gland and palpitation of the heart.

The thyroid gland is sometimes very considerably enlarged; its two lobes may hypertrophy to an equal degree, but in the majority of cases, according to Graves, Stokes, and others, and according to my own experience also, the right lobe is the one chiefly affected. The transverse portion of the gland may be involved, so that a goitre of considerable size is thus produced. The hypertrophy usually occurs gradually; it begins insidiously, as it were, and a casual circumstance alone reveals this condition to the patient. In a short time, the swelling of the thyroid enlarges more and more, simultaneously with an increase in the symptoms of the general disease. Occasionally there are periods of arrest, but the tumor has in some cases become sufficiently voluminous by this time to give rise to very appreciable modifications of the voice and of breathing. There is marked difficulty of breathing, especially when the patient lies on his back, either from the trachea being compressed by the weight of the tumor, or from its being enveloped in the hypertrophied portion of the gland which forms like a constricting band round it, as in cases of suffocating goitre. In some instances, as I will show you presently, the hypertrophy takes place almost suddenly. Lastly, I will relate to you a case in which very striking hypertrophy was succeeded by atrophy, the gland becoming affected with true cirrhosis.

The changes in the voice which occur in exophthalmic goitre may be due to the modifications of respiration, or to pressure on one of or both the recurrent laryngeal nerves. The voice may then be weak or hoarse. I need not observe that by modifying the contractility of the muscles of the glottis, pressure on the recurrent laryngeal nerves may have a share in causing difficulty of breathing.

Pathological anatomy clearly demonstrates in such cases that there is glandular hypertrophy proper, that is to say, hypertrophy of the glandular elements, of the *acini;* while, in addition to functional changes which are the result of this condition, great development of the bloodvesels of the gland may be made out clinically. For on applying the hand over the swelling, an expansive movement may be felt, which indicates that there is something more than dilatation of the superficial vessels only. This expansive movement is sometimes considerably marked over the right lobe, and in a case mentioned by Graves, the swelling formed by the thyroid was mistaken for an aneurism. Dr. Vidal, of the Paris hospitals, has told me of another case in which a similar mistake was made. In both these instances, the mistake was found out before any surgical interference. But this might be decided on, unless the general symptoms of the disease be taken into account, for in exophthalmic goitre, simple or double bellows-murmurs, with accentuation of the diastole, may be heard with the stethoscope, as in simple or cirsoid aneurism.

Every one is agreed on these two facts: double exophthalmos and hypertrophy of the thyroid gland. When I come to discuss the nature of the disease, I will investigate their course and the paroxysms which they present, and will tell you how to interpret them. Meanwhile I pass on to the other great clinical fact, which, with the two preceding, constitutes the symptomatic trio or tripod characterizing Graves's disease: I mean, the state of the heart.

The patients complain of palpitation long before the exophthalmos and goitre have attracted their notice, or that of their friends. There is violent beating of the heart, which, by pushing forward with force the generally emaciated chest-wall, soon produces prominence of the precordial region, while the heart's impulse against the chest is so powerful that it can sometimes be heard from a distance. These pulsations increase in frequency and strength under the influence of mental emotion or of exertion of any kind, so that the patient cannot take any continued exercise. The valvular sounds are exaggerated, and are generally accompanied by a soft systolic bellows-murmur, audible in the large arteries also. The carotids pulsate more forcibly than natural, and they, as well as the jugular veins, have a share in the production of the sounds heard over the enlarged thyroid.

When we look at the group of symptoms made out by examining the heart, namely, violent but regular impulse and bellows-murmur at the base, we can easily understand how Stokes has been led to describe a variety of exophthalmic goitre with hypertrophy of the heart. I must add that valvular disease was present in some of Stokes's cases, although he already saw that this was not the rule, and he therefore described separately exophthalmic cachexia complicated with organic disease of the heart when treating of the disease. This clinical division should, I think, be retained, because, although exophthalmic goitre is not, in my opinion, necessarily attended with dilatation of the cavities or alteration of the valves of the heart, yet such lesions may coexist with it, and may perhaps have been instrumental in bringing it on. But there is a wide difference between this interpretation and the theory broached by Stokes, namely, that the disease is merely a cardiac neurosis, to which all the morbid phenomena are due. Nor can I concur with Dr. Aran,* who has stated that the heart was always enlarged in cases of exophthalmic goitre.

It might be asked with regard to Dr. Aran's cases, be it said in passing, whether there was merely dilatation of the cavities or true hypertrophy of the walls of the organ. For he simply stated that there was increased cardiac dulness. The majority of the cases which have come under my own observation, and those which have been published by Dr. L. Legros and Professor Teissier,† do not tell in favor of Dr. Aran's views, but as assertions made by such a sagacious observer should be taken into consideration, I will inquire how the discrepancy has arisen.

Aran, after making out that there was extensive cardiac dulness, tried to support his opinion by arguments drawn from the character of the heart's impulse and the pulsation of the carotids. But I will observe that the heart may beat and the large vessels pulsate with violence without there being necessarily hypertrophy of the heart, and these symptoms may exist in chlorosis and hysteria. In such cases, these transient phenomena are merely the consequences of perfectly determinate nervous states.

But it is important to ascertain whether the increase in size of the heart be a constant fact. It is not so, I think, and it may therefore be asked how my opinion is to be reconciled with that of the observers who believe that there is such an increase in size.

It has been stated by some that, in addition to palpitation and violent impulse of the heart, the area of cardiac dulness is increased. From this it might be inferred that the organ had increased in size. But it is a remarkable circumstance that the pulse at the wrist was, in the majority of instances, found to be normal, while, if there had been cardiac hypertrophy, the radial arteries would have been full and vibrating like the carotids. Had the heart been, on the contrary, passively dilated, the radial arteries would have been weak and perhaps irregular, and, in some cases, jugular pulsation might have been noted. But no such symptoms were ever found to be present, and the exaggerated beating of the heart and the increased area of cardiac dulness were the only ones in favor of the idea of cardiac hypertrophy. I have already suggested an explanation of the exaggerated impulse; and as to the other pnenomenon, increased area of cardiac dulness, it is often present, but is merely apparent. For there are two kinds of precordial dulness: one of which is absolute, and measures normally from 4 to 5 square centimetres (about 2 square inches), and the other relative, that is to say, dulness extending beyond the limits of the preceding, which may vary indefinitely, according to the relations of the heart to the lungs, according to the amount of obstruction to the circulation, and to the greater or less degree of thickness and density of the thoracic parietes. The relative dulness may measure from 10 to 12, 13, 14 centimetres transversely

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* Bulletin de l'Académie Impériale de Médecine, Paris, 1860, t. xxvi, p. 122, and following

† Du Goître exophthalmique, 1863.

(from 4 to nearly 6 inches), and from 8 to 10 or 12 centimetres (3⅕ to 4 or 4⅘ inches) vertically. This dulness may be readily made out in Graves's disease, because it frequently happens that the patient's chest-walls are very thin.

I lay great stress on this cause of error, because in a case of exophthalmic goitre, in St. Bernard Ward, several observers differed as to the presence or absence of cardiac hypertrophy, some believing that there was hypertrophy, on account of the increase in the area of cardiac dulness. Professor Bouillaud kindly examined the patient, and this eminent practitioner, whose experience in the diagnosis of heart-disease is so great, stated positively that there was no increase in the real absolute dulness, and that there was no hypertrophy of the heart.

The greatest care should be taken to determine the limits of the real or absolute, and of the relative, dulness, as the former alone is to be regarded as indicating hypertrophy of the heart.

Although there may not be active hypertrophy of the central organ of circulation, there may sometimes be dilatation of its cavities, for one may conceive that during the paroxysms of Graves's disease, when the heart gets fatigued after acting tumultuously, its walls, especially those of the right auricle, should get distended. At such times, percussion will show an increase in the area of dulness, but only temporarily.

The rule, therefore, is that Graves's disease is not necessarily attended with disease of the heart, but it may attack individuals who become subsequently subject to cardiac affections. The patient's previous history and the presence of signs indicating organic lesions will enable the practitioner to ascribe to each disease its proper share in the production of the cardiac condition.

From the cases of Graves's disease which I have examined, I have come to the conclusion that this complaint does not necessarily bring on hypertrophy of the heart, but I can conceive that it may give rise to a lesion analogous to the cardiac hypertrophy which occurs during pregnancy, a condition which may be transient only, disappearing a few weeks or months after the primary complaint has been cured, or may remain permanently, as in some instances, which have as yet been rare. It seems, indeed, to be a well-established fact now that the heart and other muscular organs may increase in size pretty considerably without being diseased on that account. Cases published by Dr. Larcher, the subjects of which were pregnant women, and the results of dissections made by Dr. Blot of the bodies of women who had died shortly after delivery, leave no doubt as to the physiological hypertrophy of the heart during pregnancy. Dr. Beau is himself of opinion that, if there be cardiac hypertrophy in Graves's disease, the lesion is curable.

There can be no question as to the enlargement of the bloodvessels in the neck. The carotid and thyroid arteries undergo important modifications; the latter especially increase markedly in calibre, both the trunks and their ramifications; the thyroid veins dilate in the same manner; and the bloodvessels are so largely developed that it would be an act of rashness to cut with a knife into the swelling formed by the thyroid gland. There is an increase in the amount of bloodvessels, and dissection proves that the vascular as well as the glandular elements of the thyroid are hypertrophied. The thyroid may be seen to expand, and on auscultation, a bellows-sound may be heard over it. Similar murmurs and pulsations may be observed over the region of the cœliac axis. These phenomena do not extend into the lower portion of the abdominal aorta, nor into the iliac and femoral arteries, and it is a remarkable circumstance that, while the heart and the cervical bloodvessels seem to indicate exaggerated activity and force of the circulation, the pulse at the wrist is not abnormally full. This clinical fact did not escape Graves, Stokes, Hirsch, and all those who have studied exophthalmic goitre. It seems to indicate that the pulsations of the heart and of the cervical bloodvessels are due to some special cause limiting its action to the walls of those organs.

We shall see by and by what inference can be drawn from this localization of the morbid action, with regard to the nature of the disease, by putting together pathological facts and the physiological phenomena which Professor Claude Bernard has so well studied experimentally.

As yet I have only spoken of the three principal symptoms which together constitute Graves's disease, but there are other secondary symptoms which should be taken into account. In some cases, there is diminished or capricious appetite; in others, on the contrary, the patient is not easily satisfied, digestion is good, and yet there is progressive emaciation, and the patient's color goes. Sometimes diarrhœa comes on, which increases the tendency to emaciation. When the complaint seems to improve, however, the diarrhœa diminishes and stops: the patient takes advantage of her voracious appetite, and recovers the appearances of good health. If she has not yet attained her full growth, she is then noticed to grow with prodigious rapidity, and to gain strength in a sustained manner. As fresh paroxysms return, these advantageous results of a good nutrition may disappear, but, as a rule, the paroxysms become less and less frequent from the time when nutrition begins to improve. Most of the women who have Graves's disease suffer from amenorrhœa also. In the beginning, menstruation is only disturbed, but it is after a time completely suppressed, and hopes of a favorable issue are not to be entertained until this function is perfectly re-established. This is an important prognostic sign. The amenorrhœa is accompanied by leucorrhœa, which is sometimes very profuse, and thus increases the tendency to debility.

The patient often also presents all the characters of anæmia, and, in some cases, even of well-marked chlorosis. The capricious appetite, the development of flatus in the intestines, the alternation of diarrhœa and constipation, the palpitation of the heart and the bellows-murmur heard over the bloodvessels, the extreme pallor of the face and of the mucous membranes, the œdema of the lower limbs, the disordered menstruation, and, in some cases, the marked changes in the patient's temper, might mislead one as to the nature of the affection, if all these phenomena were not secondary to, or concomitant with, the three great symptoms of Graves's disease. Several practitioners are inclined to ascribe to anæmia a share in the production of this complaint, but I am glad to be able to refer you to an essay, rich in cases, lately published by Professor Teissier, in which he has shown that exophthalmic goitre may exist without anæmia. In four of his cases, there was not a trace of anæmia: the patients had, on the contrary, all the appearances of a fine and sanguine temperament: they were stout, had powerful muscles, and, moreover, had a sense of their strength.

The patient's temper is so altered that the persons about her can scarcely put up with her irritability, her want of grateful feelings, and her exacting ways. I have known a young lady, who was usually of a sweet disposition, become disrespectful and quick-tempered, almost violent. Besides this change in the temper, there is sleeplessness, an unpleasant complication, which, if it lasts, throws the patient into a state of perfect despair; she cannot find an easy posture, but keeps shifting about in bed, and longs for the return of day; she feels perfectly weary, and yet cannot rest for a moment.

Now, what is the mode of invasion, and what the course, of this strange complaint? in what order do the symptoms succeed one another?

Without any very determinate cause to account for it, generally in persons of a nervous temperament, a certain degree of irritability becomes noticeable; the temper is not so even as it was. Within a short time, the expression of the face, and that of the eyes in particular, is in keeping with the sudden burst of displeasure, and the transient fit of anger. It is a remarkable circumstance, however, that the eyes permanently retain a strange look; they are unusually lustrous, and look larger. The exophthalmos soon becomes manifest, and then presents the characters which I have already mentioned. The patient is conscious of the mobility of her temper, and says that she often tries, but in vain, to repress it. She feels sad, and cannot account for her painful sensations; she complains of a sense of beating in the head, inside the eyeballs and along the neck, and expresses alarm at her palpitation, on account of its frequency and violence.

A medical man is consulted on account of the capricious temper, the strange look about the eyes and the palpitation of the heart; and, until the time when Graves's disease came to be described as a morbid entity, serious mistakes were frequently made. Practitioners who did not know this complaint thought that the peculiar mental condition of the patient and her palpitation were merely curious nervous symptoms due to anæmia or chlorosis, or to painful or irregular menstruation.

The patient, however, especially if a woman, drew the practitioner's attention to the swelling in her throat, and mentioned that for some time past she had had a sensation of fulness and of pulsation in that region; her statement was noted down, but was not taken into account, as at the outset the thyroid gland is not enlarged to a considerable extent. By degrees, however, as the throat swelled, the practitioner's attention was forcibly drawn to the coincidence of those three symptoms, palpitation of the heart, exophthalmos, and hypertrophy of the thyroid gland. This coincidence was looked upon as curious, and instances supposed to be analogous, in which it had been noticed, were recalled to mind, especially when the patient happened to be chlorotic. But as after all these three symptoms were rarely met with in combination, and were not detected at the onset of the complaint, the cases in which they occurred were regarded as curiosities, and the facts observed, remaining uninterpreted, were as a sealed letter.

Nowadays a mistake is less easily committed, and the disease will be recognized whenever the simultaneous or closely successive development of the various phenomena which characterize it is observed. Do not think, however, that the diagnosis is always easy. A certain degree of care is necessary, in order to recognize these phenomena in the beginning, and the form which they assume, when they are in an incipient stage, should be well borne in mind. You should thus suspect this complaint when you find a lustrous appearance of the eyes coexisting with palpitation of a violence out of proportion with the organic condition of the heart, and you should banish all doubt when you find, in addition to the above symptoms, marked increase in the pulsation of the vessels of the neck, and slight hypertrophy of the thyroid gland. Bear in mind, however, that the latter symptom may be late in showing itself, especially if the patient be a male. This is not the case in women: hypertrophy of the thyroid is well marked in them when they consult a medical man, and it keeps pace with the increase in violence of the palpitation of the heart and the prominence of the eyeballs. In several cases, however, which have come under my notice, the goitre was late in its appearance, although the patients were women.

Quite recently Dr. Cazalis, a distinguished physician of the Paris hospitals, did me the honor of consulting me about an engineer, thirty-five years of age, who presented all the symptoms of exophthalmic cachexia, and who complained of such violent palpitation that Dr. Cazalis and I paid particular attention to the organic condition of the heart. The most careful examination enabled us to say that there was no hypertrophy of that organ, for the transverse dulness measured two inches only, and no abnormal bruit was audible over the aortic and mitral orifices. The thyroid gland did not seem to be hypertrophied, but on carefully examining the anterior aspect of the throat while the patient was placed in a favorable posture, we made out that the throat was slightly larger on the right side. The increase of size was scarcely appreciable, but it was sufficient, when complemented by the other symptoms, to enable us to state positively that the patient was suffering from Graves's disease. I saw the patient four months after this, and then found slight swelling of the right lobe of the thyroid gland, with some aortic blowing murmur. He was, on the whole, considerably better.

Graves's disease is pretty common in women, but is relatively rare in men. Of fifty cases of this complaint collected by Withuisen, only eight occurred in males. For that reason, then, I will now relate to you the history of the patient whom I saw with Dr. Cazalis.

He consulted Dr. Cazalis, for the first time, on September 2, 1861. He had enjoyed pretty good health in his youth. Six months previously, while in Russia, he fell ill of a fever unpreceded by premonitory symptoms, or by any change in the use of his faculties or the performance of his functions, or by febrile malaise, or the least general perturbation. His pulse became extremely frequent, and almost persistently so, from 120 to 130.

His appetite was better than it had been, and yet he did not gain flesh; he lost flesh, on the contrary, although his digestion was good, and he had no intestinal disorder. He would not have thought himself ill if it had not been for the constant frequency of his pulse (120). He tried quinine at Wilna, without any good results; and in Germany he was recommended the Kreuznach waters, in order to bring back to his skin herpetic eruptions, which he had had in former years, but which had long since disappeared.

He took the Kreuznach waters for six weeks. They produced some increased excitement, and while he was going on with his treatment, in August, 1861, the prominence of his eyeballs was noticed, together with some injection of his conjunctivæ, especially the right one. On his return to Chartres, he consulted Dr. Roque, who diagnosed exophthalmic cachexia, and advised him to go to Paris.

Dr. Cazalis introduced him to me, and drew up the following account of our consultation:

"M. X. is of medium size, very thin, dark, and without cachectic hue. On looking at him, one is at first struck with the prominence of his eyeballs, which, together with the marked dilatation of his pupils, gives to his face a strange undescribable expression. He related to us his previous history, as given above, and we then noted the following facts:

"1. His pulse is from 120 to 125, equal, regular, but *very frequent, very small, and contracted.* The walls of the artery seem scarcely to yield to the heart's impulse, and resist expansion.

"2. The heart is ascertained to be of *normal* dimensions by percussing the precordial region.

"3. The heart's impulse is very powerful; its rhythm is good, but it beats in a rapid hurried manner, almost convulsively. The powerful thump of the heart against the chest-walls contrasts with the smallness of the pulse.

"4. Both sounds of the heart are free from murmur, but we think we can occasionally detect some hesitation in the second sound.

"5. Although no friction-sound and no blowing murmur are heard over the heart itself there is friction-sound heard over the ascending aorta and the arch, and behind also, over the thoracic aorta. The same sound may be detected about the origin of the cervical arteries.

"6. Auscultation of the chest discloses unexpected signs. The patient has neither cough nor dyspnœa; he has never had asthma, and yet all over both lungs, although in a varying degree, the respiratory murmur is found to be loud, almost sibilant, expiration loud and prolonged: in a word, sibilant rhonchi are heard as in the most marked instances of humid asthma. Now, is this pulmonary condition to be ascribed to the present complaint? We believe not, and we are inclined to think that the state of the lungs is a consequence of iodism. We base our opinion on the fact that the patient's system has become saturated with iodine from the use of the Kreuznach waters, and that similar symptoms have been noted in persons that were not asthmatic, but were under the full influence of iodine.

"7. The eyeballs project considerably from the orbits, and the patient is himself aware of the change in his appearance. The right eye is more prominent than the left. The pupils are largely dilated. There is great alteration of sight; it is less distinct, the images are obscured, badly defined, and surrounded by a mist.

"The eyes are full of tears; the right conjunctiva especially is very much injected: there is true ophthalmia. We ascribed these phenomena to iodism.

"8. The thyroid gland is markedly hypertrophied, especially in the lower part and in the right lobe. The enlargement does not yet interfere with the neighboring organs.

"9. M. X. has always had a good appetite, but has a still better one since his present illness set in. Digestion is excellent, but yet there is considerable and increasing emaciation.

"10. There is pretty marked thirst; the amount of urine passed is proportionate to that of the liquids taken; the secretion is of normal tint, and contains neither sugar nor albumen.

"11. The patient sleeps badly now, but used to sleep very well. He wakes three or four times in the night, which is a very unusual thing with him; and he is some time before he drops off to sleep again. Since he has been to Kreuznach, he has been worse in that respect.

"12. The patient says that he feels in a peculiar excited state which he cannot define, and his friends attest the same thing. Since he drank the Kreuznach waters, his speech has become jerking.

"13. The patient's sexual power had failed a little, but shows a tendency to recover itself."

I need scarcely call your attention to the completeness of this case. The three great symptoms of Graves's disease are there present: there is impaired nutrition, in spite of a perfect digestion; there is irrritability, a jerking speech, an habitual febrile condition, and there are differences of resistance and fulness between the carotid and the radial pulse. The case is a perfect type of this complaint, and presents a complete picture of Graves's disease in its developed state.

I mentioned that, in this case, the enlargement of the thyroid gland was not very marked. When this gland does not increase in size, or when the eyeballs do not become prominent, the disease may be said to be incomplete, as one of the chief symptoms is absent. This happened in two cases, which I will relate to you, one of which occurred in my private practice, and the other in a patient under my care at the Hôtel-Dieu. One of the chief symptoms is absent in each case; the disease is incomplete, but its existence is unquestionable, as you will be able yourselves to judge from the combination of the other symptoms.

Mrs. X. (from Jura), aged 38, and married for the last seven years only, consulted me on October 23, 1861. She enjoyed pretty good health up to the time of her marriage. In December, 1857, she was attacked with a continued fever, with daily remissions, so much so that intermittent fever, recurring at the same hour every night, and lasting till the next morning, was diagnosed, and that for a whole year. She complained at the same time of violent pains in the head, of distressing and persistent want of sleep, and of a difficulty of breathing which compelled her to spend a part of the night in an arm-chair, with an open window. She coughed but did not expectorate anything. Subsequently, the fever still keeping on, she had violent palpitation of the heart, which scarcely ever ceased day or night. She never had a sensation of throbbing in the neck or head. Her eyes had begun to swell a few months after the fever set in, and before she had felt any palpitation. At the end of five months, the exophthalmos was at its maximum, and persisted to the same marked degree until the autumn of 1860. It is stated that the exophthalmos has been paroxysmal, that the eyeballs diminished in size for a little time, and then quickly increased to a very large size again. There was at such times pain in the eyeballs; the patient was unable to work on account of slight mists before the eyes, and the right eye was more prominent, but less affected, than the left. These paroxysms were very marked on several occasions, but the patient cannot say whether there was simultaneous increase of the other symptoms. The exophthalmos has gradually diminished for the last year, and the prominence of the eyeballs is not at present unpleasantly marked. The palpitation and the fever ceased before the prominence of the eyeballs became less.

The catamenia went on diminishing by degrees from the beginning of the complaint, and stopped for eight or ten months, but are now regular.

There was loss of appetite, diarrhœa, and extreme emaciation during the continuance of the latter.

Such was the patient's previous history. When I saw her, her complexion was fresh and her face full; this contrasted curiously with the extreme emaciation of the rest of the body, and with the complete atrophy of the breasts. The eyeballs were prominent, not painful, and there was slight double external strabismus.

No trace of goitre; the throat was, on the contrary, markedly thin.

On careful examination of the heart, its size and the rhythm of its pulsations were found to be normal; no bruit was heard over it; the pulse at the wrist was normal, but somewhat frequent, 88.

Every summer the patient is seized with fever again, and has a persistent cough; but auscultation detects no organic lesion of the lungs.

There could be no doubt that this lady had been suffering from Graves's disease for some years past. Her feverish condition, her continued want of sleep, her irregular menstruation, disordered digestive and nutritive functions, her palpitation, unexplained by an organic lesion of the heart, and the prominence of her eyeballs, set all doubt at rest, although a chief symptom was absent, namely, hypertrophy of the thyroid gland. The disease, in such a case, may be said to be incomplete, but it exists nevertheless. The following case will give you another illustration that one of the principal symptoms, the exophthalmos, may be absent, and yet the diagnosis not be doubtful.

A woman, aged 29, was admitted into St. Bernard Ward, on October 18, 1861. She was born at Dijon, and has lived there for a long time. For three years previously, she got out of breath whenever she went up a staircase or made an effort. She has been five years married, generally menstruates regularly, and is the mother of two children. She enjoyed good health during pregnancy, and states that her breathing improved very markedly while she was in that condition, but became still worse than before a few weeks after delivery. She had at such times palpitation of the heart, although she could continue to attend to her household work. A month previous to her admission into hospital, her breathing became very bad, and her palpitation increased; she noticed also that her throat swelled, and she slept badly. Her eyeballs did not become prominent, but her eyes had a strange restless look, and were constantly on the move.

She continued to nurse her child, who was now four months and a half old, although she had not much appetite, slept very little, and was obliged to spend whole nights in the sitting posture, as she was seized with dyspnœa whenever she lay down. She did not lose flesh sensibly; and her child looked in good health.

Six weeks ago, she felt a violent pain and throbbing inside her head, which made her shriek; she became very impatient also.

On admission, she was in the following state:

The thyroid gland was very markedly prominent, especially its right lobe; it pulsated and seemed as if it were thrust forward at each systole of the heart; a slight bellows-murmur could be heard over it, but not over the arteries in the neck. The patient felt her goitre pulsate, and stated that her throat began to swell six weeks ago, that is, three months after delivery.

There was no exophthalmos, but the eyes were shining and very mobile. The same sensation of throbbing was complained of in the eyes as in the goitre.

The heart's impulse was strong and frequent, and the patient had a pain in her back. The relative cardiac dulness measured 9 square centimetres (3½ inches); there was no cardiac bruit; the valvular click of both sounds was normal, perhaps a little accentuated and drier.

The pulse at the wrist was frequent, not full.

The patient coughed, and had occasionally some fever, and expectorated as in a mild attack of bronchitis. There were only a few scattered moist rhonchi, without any relative dulness at the apex; there has never been hæmoptysis.

The patient was extremely agitated, and very irritable.

The baby was sent out to be nursed, but after that the patient's symptoms seemed to increase, and her eyelids to get more widely separated.

The diarrhœa has stopped under treatment, but the appetite continued bad.

For the first case which I related to you, that of the gentleman under the care of Dr. Cazalis, there was scarcely any hypertrophy of the thyroid gland, although the complaint dated several months back. In the second case, goitre did not show itself, although the disease had lasted several years. Lastly, in the third case, the patient had no marked prominence of the eyeballs, but merely a strange look about the eyes; in a few weeks from this time, perhaps, this symptom will show itself.

I have placed these three cases together, because they prove how irregularly goitre and exophthalmos occur in Graves's disease, and because they show that one of the principal symptoms of the complaint may be absent, and yet all the others be present, so as to characterize it.

I must add, gentlemen, that I believe that exophthalmic goitre may exist in a still more undeveloped condition, and that the disease may be foreseen, and does really exist, in a great number of instances, without there being exophthalmos, bronchocele, or extreme frequency of the pulse. I am glad to find that my excellent friend and colleague, Dr. Teissier, Professor of Clinical Medicine in the Lyons School of Medicine, shares my views on this point completely, from his having seen incomplete cases of the disease. Dr. Teissier has noted four times absence of exophthalmos in patients who had palpitation of the heart, with swelling and enlargement of the thyroid gland, acceleration of the pulse, nervous restlessness, sleeplessness, and shining strange-looking eyes; in a word, in persons who exhibited most of the symptoms of Graves's disease. In these four patients, as well as in others, Dr. Teissier noted also a symptom well worthy of attention, namely, a rise in temperature of which the patient complained, and which could be measured by the thermometer. The increase has often been of one or two degrees Centigrade, the normal temperature being 35° or 36° C. (96° to 98° F.). This symptom had not escaped Basedow, and you need not be surprised at its being present, if you recall to mind that certain lesions of the sympathetic nerve are followed by a rise in temperature. You are also aware that in other neurotic affections, as in diabetes mellitus, for example, the patient often complains of a sensation of great heat, especially at night, and sleeps lightly covered only.

There is just now at No. 2, in St. Bernard Ward, a woman suffering from Graves's disease, who presents all these symptoms: her pulse is usually 120, and her skin always warm and dry. During the periods of exacerbation, the pulse rises rapidly to 140 and 150 pulsations; the sensation of heat becomes unbearable, and the patient throws off all her coverings from her bed.

I have noted besides, in this woman, a symptom to which attention has not yet been called, and which I should like observers to look for, namely, the *cerebral macula*. If the epidermis be slightly irritated, after two seconds at most, a beautiful red stain is seen, which lasts nearly a minute. I can hardly believe that there is not in this case very marked *asthenia* of the *vaso-motor nerves*, in consequence of which the capillaries dilate rapidly, easily, and persistently, under the influence of the slightest irritation, just as happens in cerebral fever and in some ataxic cases of typhoid fever. Now, my opinion is that these three phenomena, acceleration of the pulse, rise in temperature, and cerebral macula, are of the same kind, and are traceable to the same cause, namely, some deep modification affecting the sympathetic and vaso-motor nerves in particular, which gives rise to this artificial febrile condition and its usual concomitants, frequency of pulse and rise in temperature. As to the change in the sympathetic nerve, I shall presently discuss its nature with the aid of the experiments that have been made by Claude Bernard and by Schiff, but I will at once state now that I believe it to consist in asthenia, if not a momentary paralysis, of the vaso-motor nerves.

Graves's disease begins with an extraordinary nervous irritability, marked changes of temper, frequent flushing of the face, a sensation of fulness in the head, the eyes, and throat, and with violent throbbing of the heart. These symptoms come on in paroxysms which last from a few minutes to a few hours, and even days. Menstruation, in women, becomes disordered; the menses generally diminish in quantity, flow at long intervals, and even stop entirely.

The digestive system soon exhibits symptoms of disturbance; anorexia replaces bulimia; the patient complains of violent throbbings in the pit of the stomach, and has vomiting. In the majority of cases, the patient grows thin, even when the appetite is very good; in some rare instances, as in the case of a lady whom I saw in consultation, certain organs get developed, as the mammæ; while no indication is to be derived from the thyroid gland or from the eyes, and the emaciation of the rest of the body presents a strange contrast to the increase in size of certain parts. There must be, in such cases, partial hypertrophic congestion, probably due to a functional disturbance of the vaso-motor nerves. These premonitory symptoms may extend over several months and years, and sooner or later, the three prominent symptoms of Graves's disease show themselves, setting all previous doubts at rest as to the nature of the symptoms.

I will now describe the order in which the chief symptoms of this complaint develop themselves. The order is more apparent than real. I believe that all phenomena which are due to the same cause should appear simultaneously, and have the same period of invasion. This evidently happened in the case of a patient whose history I shall relate to you further on, who felt for the first time and on the same night, after violent mental emotion, strong palpitation, swelling of the thyroid gland, and exophthalmos, with copious epistaxis. It merely happens that the patient or the practitioner cannot always detect the phenomena. Palpitation first attracts attention, because the least disturbance of the heart's action cannot escape notice, and in addition to the throbbing at the heart, a marked sense of oppression comes on. The patient can no longer attend to his work, and is prevented from doing so by his palpitation; mental emotion also increases it. He complains of this, then, before the strange look about his eyes, the protrusion of his eyeballs, and his capricious temper have yet attracted notice.

The exophthalmos is slowly developed, but when it has once set in, it continues and nearly always makes very remarkable progress.

Since, as I have told you, the hypertrophy of the thyroid gland is sometimes but slightly marked, it is very natural that it should be noticed at a late period. It has been forming for a long time already, when the patient first begins to speak of the increase in size of his throat, which he often remarks only when he has some difficulty in buttoning his collar. But if you carefully examine with the hand, and compare the size of the two sides of the throat, you will often be able from the very commencement of the disease to recognize in the right lateral lobe of the thyroid a marked difference which the patient has not yet observed.

Palpitation is to the patient the first symptom of the disease, and it is only at a later period that the exophthalmos and goitrous swelling show themselves.

There are two very distinct forms of the complaint, a rapid or acute form, and a slow or chronic one. They both have paroxysms, when the patient suddenly feels great oppression; the palpitation increases in violence, the eyeballs protrude more, the goitre is more prominent, and the dyspnœa is so great as to threaten suffocation. These paroxysms are peculiarly grave in the acute form of the disease, and may put the patient in great peril of his life. The following case, of which Dr. Labarraque took careful notes, will show you to what extreme measures a practitioner may be obliged to have recourse, in order to save life in the acute paroxysms of Graves's disease.

"T., aged fourteen years and a half, of a good constitution, but of a somewhat lymphatic temperament. He went to school when he was twelve years old, and joined in games usual among boys of his age, and could swim without ever feeling the least difficulty of breathing. More than two years ago, however, it was noticed that he could not bear sea-bathing, except for a very short time: river-bathing had not the same effect on him.

"Some time afterwards, about eighteen months ago, he complained of some marked alterations of sight: he became more and more short-sighted, and this myopia, which has lasted up to this time, came on within a few weeks. He could not see what was going on at the board, in the class-room, and had to wear No. 9 glasses for short sight.

"In the beginning of August, 1860, he went away for his holidays, feeling nothing particular at that time, and merely looking a little less well than usual.

"About a week after his arrival at Vierville, his friends noticed that his throat was rather full, especially at the base, but there seemed to be nothing serious about this.

"A few days afterwards, the fulness, having markedly increased, attracted greater notice.

"From that time, he could not bathe in the sea, as he used to be so fond of doing. The first time that he attempted it, as he plunged into the water, he felt violent dyspnœa as if he were going to choke, and he could scarcely manage to get away. He took a very hot foot-bath, had sinapisms applied, and the paroxysm ceased.

"But even at that time, although the swelling of the throat was getting gradually larger and larger, there was no marked difficulty of breathing.

"Dr. Lebâtard, of Trouville, who was consulted, prescribed syrup of iodide of iron internally, and iodide of lead ointment to be rubbed on the swelling.

"This treatment was carefully followed, but without any good results. The disease seemed to get worse, on the contrary; the swelling went on increasing, and respiration became impeded. The face altered, and assumed the dead white hue of commencing asphyxia.

"Towards the end of the holidays, the treatment was suspended, and after a few days the boy began to improve markedly; indeed, he was so much better that he was preparing to go back to school.

"At the end of eight days, the disease began to progress again, at a fearful rate: the anterior aspect of the throat continued to swell, almost visibly; the respiration became more obstructed and whistling, and the boy could neither run nor go up a staircase, and could scarcely walk at all.

"Dr. Blache, who was then consulted, expressed anxiety about the case. He stopped the treatment by iodine, enjoined rest, and ordered a weak ammoniacal ointment to be rubbed into the swelling, and frequent immersions into hot water of the hands and feet.

"Three days after this, there were such threatenings of suffocation that Dr. Blache was sent for in the middle of the night, and on his arrival desired to have a consultation with Dr. Trousseau. A few hours afterwards a consultation was held by us three."

The disease, as you see, gentlemen, had set in three months previously in this case, in August, and it was in the beginning of November that an acute paroxysm came on, without any appreciable determining cause, and threatening the boy's life. The face turned blue, the vessels of the neck swelled out, there was intense dyspnœa and imminent asphyxia. Tracheotomy being indicated, Dr. Demarquay was asked to hold himself in readiness to perform it; but it was decided that, before having recourse to this extreme measure, the boy should be bled at once, and ice be applied over the front of the throat, and digitalis administered every hour. This plan answered all the indications, namely, of unloading the general venous system, of diminishing the fulness of the thyroid gland, and of quieting the extreme agitation of the heart.

The dyspnœa continued to be great during the day, but the symptoms of asphyxia disappeared, and the boy, who had not slept for the last eight days, slept on the following night for eight hours without waking. All present danger being removed, we waited for the good results of a continued sedative treatment, but we did not feel perfectly free from anxiety, and we asked the surgeon still to hold himself ready to act. When the boy woke the next morning, he was still agitated, as is so often the case when respiration is not free in consequence of some obstruction to the entrance of air. The agitation soon grew less, and three days after our first consultation, the boy could be said to have come to life again. He had no dyspnœa, and could in our presence, go up or down stairs without experiencing any difficulty of breathing. The amelioration became persistent from the third day, and went on increasing; three weeks afterwards, T. could walk three miles to come and thank me for having attended him. By this time, his goitre has almost entirely disappeared, as well as the exophthalmos and he has no palpitation.

I will now, gentlemen, relate to you the history of a case of exophthalmic goitre, running a chronic course, and of eleven years' duration.

Seven years ago, Dr. Labarraque was consulted at the Dispensary of the Philanthropic Society by a woman, thirty-nine years of age. She complained of difficulty of breathing, and palpitation of the heart; she had prominent eyes, and a very enlarged thyroid gland. She was treated successfully by bleeding, digitalis, and repeated doses of drastic purgatives. The exophthalmos and goitre became less, and the palpitation ceased.

Dr. Labarraque had only a vague recollection of that case, as, when it came under his care, Graves's disease was not known as a morbid entity, when a few days ago, he was consulted by the same woman about one of her children. She was not perfectly well, and still bore undoubted marks of her complaint. Dr. Labarraque kindly sent her to me, and on Sunday, November 18, I took down her history myself, of which the following are the chief points:

She is forty-six years old. (Note, in passing, that exophthalmia is rare at that age, and generally occurs from twenty to twenty-five.) In 1849, after a great fright (Stokes and Graves have mentioned fright as a cause of this complaint), she became subject from the day on which she was frightened to palpitation, which has continued ever since. Five or six months afterwards, her thyroid gland increased in size, her eyeballs became prominent, and she complained of short-sightedness. Her sight, although short, was at first excellent, and allowed her to work at lace-making, but amblyopia came on; she was troubled with muscæ volitantes, and saw large black spots on white grounds, so that she was compelled to give up her occupation as a lace-worker. At that period, that is to say, five or six months after, she became subject to palpitation; the catamenia stopped completely; her appetite was voracious, and she had diarrhœa at the same time.

The amenorrhœa lasted four months, and she then became pregnant. She was delivered on October 21, 1851, and recovered her health; she even got rid of her palpitation. She remained in this satisfactory condition until 1855, when she had an attack of pleurisy, after which the symptoms of her former complaint again showed themselves. Her appetite again became voracious, while diarrhœa returned, and she lost flesh and strength considerably. Dr. Labarraque, who saw her at that time, found that she had exophthalmos, an enlarged thyroid gland, and palpitation of the heart. She was treated by bleeding, drastic purgatives, and digitalis, and was enabled to resume her occupation at the end of eight or ten months.

In August, 1856, her daughter got married, but she could not sign the marriage-contract, because of the great tremulousness of her hand, resulting from her extreme nervous excitability. At the present date, she still has prominent eyes, suffers from palpitation, and has an hypertrophied thyroid gland, of the *right lateral lobe* in particular.

Her pulse varies from 140 to 120 and 108; the carotids beat with considerable force, but the radial artery is merely frequent and of normal volume. This difference has been already pointed out by Graves and other observers.

I will now relate to you the history of a third patient, of a woman who was under my care in 1861, and whom I readmitted in 1862.

She is twenty-five years of age. She was born in the vicinity of Paris, and menstruated for the first time when thirteen years old. Her health has been pretty good up to the beginning of this year; but after the birth of her last child (her throat, I may observe, did not swell during pregnancy), she was violently moved on finding out that she had been right in suspecting that her husband was unfaithful to her. From that time, she had violent palpitation of the heart; her eyes, according to the statement of her friends, assumed a strange aspect; they had been previously deep-sunken, but were now unusually prominent, and had a lustrous savage appearance. Soon afterwards, she noticed herself that her throat became gradually larger, whilst her appetite increased extraordinarily.

On her admission, I noted the strange look and the prominence of her eyes; she had a very developed goitre; her heart beat with violence, and the impulse was visibly propagated into the carotids and into the enlarged thyroid. A continuous bellows-murmur was heard over the swelling, which was raised en masse by the arteries at each contraction of the heart, while the hand applied over it had the same sensation of expansion as over an aneurism. The heart's impulse was violent; there was a soft bellows-murmur audible at the base, and prolonged into the aorta. The radial artery was of normal strength, and its pulsations were from 110 to 130. The patient had been taking iron, but it had to be stopped, because it increased the symptoms. Digitalis was given by itself, and ice applied to the swelling.

The eyes had the same savage look as before; the swelling of the thyroid was as marked, and the gland was still the seat of vascular throbbing and murmur, although the palpitation had diminished, and the cardiac bruit was less loud; when the patient was suddenly seized with vomiting, precordial anxiety, and marked increase in all the symptoms of her complaint. The catamenia showed themselves on that day, but for a few hours only. I regretted afterwards that I was not informed in time of this last circumstance, because I would have tried by bleeding the patient at the elbow, or by applying leeches to her lower extremities, to increase the quantity and duration of the menstrual flow. Amenorrhœa had, perhaps, in this case, a large share in the production of the disease, for the patient had not menstruated since her last labor, although she did not nurse her child, and the first morbid phenomena showed themselves eight days after her confinement.

The paroxysm was perhaps a consequence only of the menorrhagic fever, that is, of the natural effort needed for the re-establishment of such an important function. Nearly complete aphonia set in after the paroxysm; but was it a mechanical complication, it may be asked; a result of increased congestion of the thyroid gland, or a purely nervous phenomenon, dependent on the general neuralgic condition of the patient, of which exophthalmic goitre itself was only a consequence?

In 1863, I saw a young woman who had been formerly under my care for Graves's disease; she had suddenly lost her voice completely two days before, in consequence of some deep grief. This aphonia lasted six days, without any anatomical alteration of the larynx being detected by means of the laryngoscope, and it disappeared suddenly, without any transition, after the pharynx had been slightly cauterized with nitrate of silver.

But to return to our patient: six days after the paroxysm which I mentioned just now, her eyes had a less savage look in them, the swelling of the thyroid was less voluminous, the bellows-murmurs were less loud, and the heart seemed to have become comparatively quiet. A short time afterwards, she was discharged in a satisfactory state compared with her former condition.

We have just seen the acute and the chronic form of exophthalmic goitre. Each of them presents paroxysms which may return at long intervals only, of several months or several years, and may vary indefinitely as to their duration and gravity, or which may recur every month or several times a month. Perhaps when these paroxysms come to be better known, they will be found to have a certain relation to the hemorrhagic molimen which takes place in the uterus every month; and if, on the one hand, it should be noted that in several cases amenorrhœa existed in the beginning, and, on the other hand, it be found that the symptoms abate, and the general disease improves, from the time that menstruation is re-established, or when the woman becomes pregnant, the practitioner will perhaps be able to deduce precious indications for treatment from these relations or those fortunate coincidences.

When the disease is fully developed, congestion of the eyes and of the thyroid gland may occur several times a day, concurrently with an increase of the cardiac pulsations.

The disease may go on increasing for several months, and then remain stationary for one or two years; paroxysms no longer show themselves, and the stage of decline then begins. The heart beats with diminished frequency and less intensity; the eyes become less prominent, and lose their savage expression, the bronchocele diminishes in size, is less elastic, contracts, and becomes harder; its erectile tissue, as Graves expresses it, grows less and less apt to be distended by the flow of blood. It rarely happens that the disease disappears completely; it merely recedes, and there always remains swelling and induration of the thyroid gland, with unusual prominence of the eyeballs.

The various blowing murmurs heard over the gland and the bloodvessels of the neck may disappear completely, as well as those of the heart. The improvement in the local is preceded by the disappearance of the general symptoms: the functions of the stomach and of the intestines become normal again, the temper ceases to be capricious, and the patient is able to resume his or her usual occupation. The disease, in females, is occasionally brought to an end by the re-establishment of the menstrual flow or by the supervention of pregnancy. Some cases, then, do terminate favorably, but not all. The anæmia, which results from the disturbance of digestion, is sometimes so considerable that hectic fever supervenes, or the patient is so weakened that he becomes susceptible of any morbid influence, and dies of some intercurrent affection, which, in the majority of instances, has its seat in the respiratory organs.

Some, like Hirsch and Praël, have recorded cases, which have terminated fatally in consequence of pulmonary, intestinal, or meningeal hemorrhages. I have myself known an instance in which cerebral hemorrhage proved the cause of death.

What is to be especially dreaded during the paroxysms is the danger which the patient runs of being choked. Under certain conditions, tracheotomy offers the only means of staying the progress of asphyxia, but the surgeon should not forget the extreme vascularity of exophthalmic goitre, and should be on his guard against hemorrhage which may prove *fatal* in a few minutes.

Before I speak of the pathological anatomy of exophthalmic goitre, I will first relate to you the particulars of two cases which are of great importance, as showing the nature of this complaint. Stokes has perhaps laid too much stress on the existence of a cardiac neurosis, and he places in too subordinate a position the other phenomena of the disease compared with the functional lesion of the heart; hence the too great facility with which he is led to admit the existence of an organic lesion, namely, dilatation of the heart. Dilatation of the heart, when present, is accompanied by hypertrophy in exceptional cases only. I believe that in Graves's disease there may be a temporary hypertrophy of the heart, analogous to that met with during pregnancy. The first of the two following cases proves that although the complaint lasted two whole years, no persistent organic lesion occurred. The second establishes that, even when the disease is at its maximum, there need not be, in every case, dilatation, even though merely passive; and from this it may be inferred that the practitioner should not look upon a condition which is so often absent as one of primary importance.

The first of these two cases was communicated to me by one of my esteemed colleagues in the Faculty, whose daughter became affected, when eighteen years old, with exophthalmic goitre and palpitation. She suffered from amenorrhœa also, and a disordered digestion, marked by violent hunger alternating with want of appetite and a dislike for food. This young lady's appetite is now regular; her exophthalmos and thyroid swelling have disappeared, and it is a fact worthy of notice that she recovered at a distance from Paris, in a mountainous country where goitre is endemic. She took very little iron, and her recovery is to be mainly ascribed to change of air, as it has been observed in other cases. She can take exercise with ease, and no longer has palpitation; in fact, she is perfectly cured, and her heart presents no appreciable organic change.

The second case was observed at Clermont (Oise), by Drs. Labitte and Pain. The patient was sent to me by Dr. Pain with a very detailed report of the chief symptoms which she had exhibited for two years, and of which the following is a summary:

Miss X. has enjoyed very good health up to the age of fourteen. She showed great aptitude for learning, and, when a little girl joined in her companions' games without ever feeling any difficulty of breathing. She menstruated for the first time at the age of twelve, without her health getting impaired in any way, and afterwards every twenty-eight days regularly. When she was fourteen years old, she seems to have menstruated with less regularity, about the months of December, 1858, and January, 1859, and to have had a very painful temporal neuralgia, which only yielded to cold affusions on the head. In April, 1859, she had epistaxis, which continued for six weeks, and her catamenia stopped completely. From that time, Miss X.'s friends noticed that her eyes were getting larger and more prominent, and that her thyroid swelled in a remarkable manner. In September, 1859, she went to Normandy, where she was born; her health improved a little, and there was a slight menstrual flow. Palpitation of the heart had come on simultaneously with the exophthalmos and the goitre.

From October, 1852, to June, 1860, the three chief symptoms of the complaint continued to increase in severity, and according to the patient's mother, all the symptoms were markedly exaggerated towards the end of every month, from the 20th to the 30th. In June, she went again to Normandy; there was a temporary improvement, and the menses, which had been again suppressed since the month of October, 1859, reappeared.

At the end of June, an alarming paroxysm occurred, with threatenings of suffocation, excessive throbbing of the thyroid gland, which had considerably increased in size, especially on the right side, extraordinary prominence of the eyeballs, violent palpitation, and blowing murmurs over the heart and the thyroid gland.

At the end of July, acute symptoms showed themselves, with fever and delirium.

During all this time, there was excessive hunger alternating with complete loss of appetite, and occasional vomiting.

The patient's voice changed from the time the exophthalmic goitre first showed itself; sleep was disturbed by nightmares, and the patient often started out of sleep, on account of a choking sensation. The eyes were incompletely closed during sleep, and there was a great flow of tears. The patient has never had ophthalmia. Her temper has grown very irritable; she perspired profusely, especially at the end of every month. The disturbance in the digestion, the want of rest at night, and her nervous excitement soon produced very great loss of flesh and extreme debility, which was still more increased by frequent diarrhœa.

Miss X. had frequent attacks of epistaxis, and her mother had noticed that the paroxysm came to an end after one of these attacks, which were sometimes attended with a copious loss of blood, and that all the symptoms improved. Steel was given at first, but without any good results, while digitalis was substituted with better effect. The determining cause of the disease was perhaps some deep emotion. No member of the patient's family was afflicted with goitre, and there was no case of goitre in that part of the country where she lived.

When I saw her myself, she was in the following condition:

The exophthalmos was the most marked I had ever seen. The eyeball protruded so much that, by asking her to move her eye about, I could see through the transparent conjunctiva the insertions of the internal, the external, and the superior recti muscles to the sclerotic. The eyes looked brilliant and savage; the ocular media were perfectly transparent; the pupil was still very contractile; there was no impairment of sight, and Miss X. could read at any distances large and small type, and her eyes could so remarkably adapt themselves to distances that she could be short or long-sighted at will. When she held the book which she read at the usual distance, or at a distance greater than the average one, the axis of her eyes did not alter, but when the book was held very near, double convergent strabismus was immediately produced, the pupils contracted, and she could read with ease.

The goitre was of very large size, especially on the right side; very large veins were visible under the skin; the swelling was elastic, and arterial pulsations could be detected in it. There was very slight expansion of the swelling, but it was lifted en masse at each arterial diastole. Over it, continuous blowing murmurs were heard with rasping and sawing sounds.

There was violent throbbing, but no bulging, of the precordial region; the heart's apex beat in the fourth intercostal space; the absolute dulness on percussion measured four square centimetres only.

No blowing murmur was heard; and there were only very dry and sonorous valvular clicks, like those which may be heard in young people who have just been running, and whose hearts' sounds can be heard very distinctly through their thin chest-walls. The pulse ranged from 110 to 120; it had some strength, but was not in the least full.

Menstruation had been suppressed since the month of June, and there was profuse leucorrhœa. The appetite had become more regular for the last few days, and there was less diarrhœa. The emaciation was excessive; the skin, which was formerly transparent, was now brown and freckled in some parts; the complexion was pretty good. When Miss X. was attacked with epistaxis, her blood was of a pale rose color, and stained linen yellow, showing her to be anæmic.

The treatment consisted in the administration of digitalis, in hydropathy, and the application of ice to the precordial region. For several months, no improvement followed, but when I saw the young lady a year afterwards, she was stout and fresh-looking and in a considerably better state, although her eyes were as prominent and her goitre as large as before.

Dr. Pain kindly supplied me with further details of this interesting case in June, 1862.

Twice in the course of a year, there came on such a paroxysmal increase of the exophthalmos that one of the eyeballs became dislocated, as it were. The eyelids got behind the greater half of the circumference of the eyeball, which had to be pushed back with a certain amount of force in order to get the lids to come forward again.

On several occasions (and this is a curious phenomenon, which makes congestion of the vesseels of the thyroid gland and of the orbits resemble that of cavernous bodies), the goitre disappeared suddenly and returned after a little while. This only occurred in the morning.

There was at times such a degree of nervous excitement that insanity was dreaded.

The patient's temper has now become gentle again, and there are no longer any congestive paroxysms; her complexion is good, and she is moderately stout; the pulsations of the heart have diminished, and I have been informed by Dr. Pain himself that the young lady has completely recovered for some months past.

I will now proceed with the general description of the disease.

I have told you already that in exophthalmic goitre, the heart did not necessarily present organic lesions, but that some careful observers had noted hypertrophy of that organ, with or without valvular lesion (Praël, of Berlin, 1857), passive dilatation (Graves), either temporary, and noted only during the paroxysms, or permanent, when the disease had extended over a long period, and I have myself met with hypertrophy without valvular lesion. Lastly, exophthalmic goitre may affect individuals already suffering from heart disease.

The pathological anatomy of this complaint is treated of in Dr. Withuisen's memoir. After expressing regret that Mackenzie, who has seen the only case of this disease which terminated fatally at the outset, does not give any details as to the anatomical condition of the organs, which would have had an important bearing on the nature of the complaint, the Danish physician gives, in his memoir, a summary of seven autopsies. As this question is one of present interest, you will, I hope, excuse my entering into it at great length.

Withuisen remarks that the seven individuals whose bodies were thus examined had been ill for several years, and that the changes found may to a certain extent be regarded as consecutive. I quote his own words:

"The best anatomical description is that given by Neumann. The patient, in that case, had died with symptoms of organic heart disease and of a cerebral affection. The left ventricle of the heart was hypertrophied, without being dilated; the sigmoid valves were rigid, with thickened uneven edges; there was dilatation of the right heart without hypertrophy of its walls; the heart was fatty at the base. There were numerous atheromatous deposits in the aorta and the branches given off by the arch; the cerebral, ophthalmic, and ciliary arteries were likewise the seats of atheromatous deposits; the arteries at the base of the brain presented aneurismal dilatations here and there. The alterations in the ophthalmic and ciliary arteries had very probably a great deal to do with the loss of vision; which had occurred several months before death.

"The thyroid gland was of very voluminous size; it had a fibrous structure, and through it were scattered coagula of variable date; there were no cysts. The arteries supplying the gland were very much developed, especially the inferior thyroid, the coats of which were hard and fragile, and presented several aneurismal dilatations.

"The veins in the gland showed many traces of inflammation; they were partly obliterated and reduced to the condition of fibrous cords.

"The eyeballs were pushed out of their sockets by a considerable quantity of fat, but their size was also greater than usual, for the antero-posterior diameter of the left eye was 11½ French lines in length, and that of the right eye, 11 lines; the transverse diameters of both eyes were 11 lines long." Now, as Withuisen remarks, these diameters were greater than the normal ones, for, according to Mr. Sappey, the average length of the antero-posterior diameter is 10.6 lines, and that of the transverse, 10.1.

"There were numerous small extravasations of blood on the retina, and the choroid was of a uniformly red color.

"Some time afterwards, Dr. Praël found in one case atrophy of the eyeballs, without increase of the cellular and adipose tissue in the socket. The thyroid gland was considerably hypertrophied; the left heart was dilated and hypertrophied; there were a good many atheromatous patches on the endocardium and along the arch of the aorta, and there was also aortic constriction and regurgitation. The patient's death was preceded by symptoms of softening of the brain, and this condition was found on examining the body afterwards.

"A patient died, while under the care of Dr. Henry Marsh, from gangrene of the lower extremities; and disease of the tricuspid and mitral orifices was found." It is not stated in Withuisen's memoir whether Dr. Marsh examined the eyeballs and the thyroid gland.

"Another case, observed by Dr. Smith (of Dublin), need be merely mentioned: the patient died of apoplexy; there were hypertrophy of the left heart and disease of the aorta. Basedow has recorded also the case of a man who, after having for ten years presented the signs of exophthalmic goitre, died suddenly from cardiac disease. Dissection disclosed incompetency of the aortic valves. The thyroid gland was hypertrophied and full of cysts and of varicose veins. The eyeballs were atrophied, but were pushed forwards by a large quantity of cellular and adipose tissue. The same condition was found by Keusinger to explain the prominence of the eyeballs. He also found considerable hypertrophy of the thyroid gland in a man who had for years exhibited symptoms of Graves's disease, and died of a heart affection.

"The seventh autopsy was made by Dr. Kœben, who found dilatation of the heart without valvular lesion in a similar case, and increase in size of the thyroid gland, a great many cells of which were filled by a gelatinous matter. The eyeballs, which had been very prominent during life, had sunken into the orbit after death, although the left orbit contained a sufficiently considerable amount of fat to prevent retrocession of the eye."

These reports of post-mortem examinations, which Withuisen has brought together in his memoir, give you a great many of the elements of the question. Although some organic heart disease was found in every one of these cases, I think that it would be exaggerating the importance of these lesions if too great a share in the production of exophthalmic goitre were to be ascribed to them. In this disease, I repeat, I believe that the heart may, in the majority of instances, present variable and temporary alterations, analogous to those which are met with during pregnancy, and it is in rarer cases that the cardiac lesion is permanent, only when the neurosis has been of prolonged duration.

The thyroid gland is very remarkably altered in structure.

It normally is so very vascular that Graves regarded the facility with which the gland becomes congested under the influence of the heart's action as a sufficient reason for comparing it to erectile tissue. The gland is, indeed, supplied by four large arteries, and occasionally by a fifth additional vessel which comes directly from the aorta. The veins of the gland are also very developed, and we have proofs of this excessive arterial and venous development whenever a surgeon carries his knife through the thickness of the gland.

Now, in cases of exophthalmic goitre, the thyroid arteries increase in diameter and become flexuous; their extremities and branches enlarge considerably, and their anastomoses seem to multiply (Basedow, Stokes, and Hirsch). This exaggerated development of the arteries resembles the vascular dilatation known under the name of cirsoid aneurism, and accounts for the blowing-murmurs heard over the swelling, and for the expansion detected by applying the hand over the thyroid gland. I mentioned when describing the swelling, that it was lifted up en masse during the diastole of the carotids, and that it expanded outwards in consequence of the dilatation of the divisions and branches of the thyroid arteries. The venous system of the swelling is also very developed (Marsh); large veins ramify on its surface and throughout its thickness (Henoch). When the disease is to terminate favorably, the swelling, as I have told you already, becomes less elastic and harder; in such cases, dissection has shown that the vascular system decreased, while there was an increase in the connective tissue, which had become fibrous; small sanguineous cysts have also been seen which have undergone various metamorphoses. In the case recorded by Dr. Kœben, the thyroid cells were filled with some gelatinous matter.

All observers must have been struck with the exophthalmos, and hence attempts have been made to find out the anatomical cause which gave rise to this prominence of the eyeballs. With that view, the eyeball, the vessels of the eye, and the intra-orbital cellular tissue have been particularly examined. Mr. Follin could scarcely find, during life, by means of the ophthalmoscope, an increase in the vascularity of the choroid; nothing in the structures in the eye could account for the exorbitis. Mr. Broca was not more successful. The only lesions noted in the eye by Withuisen and

Neumann have been deposits of pigment round the optic papilla, but there was nothing which could explain the exophthalmos. Stokes thinks that it may be due to dropsy of the eyeball, but does not give a single proof in support of his view. Romberg found elongation and dilatation of the ophthalmic artery. Mr. Fano noted in one case considerable increase in the size of the veins of the orbit, so great, indeed, that, when the eyeball was pressed back, voluminous veins were seen to raise the upper lid en masse. With regard to the cellular tissue of the orbit, Mr. Richet has seen in an individual suffering from anasarca the general œdema disappear, but the cellular and fatty tissue in the orbit continue to be œdematous. It is not stated whether there was not, in this instance, a local obstacle to the venous circulation. Basedow, Hastinger and Kœben have found an increase in the amount of cellular tissue within the orbit. In a case which I am going presently to relate to you, I found myself enormous hypertrophy of the cellular and adipose tissue inside the orbit, which pushed the eye out of its socket.

When we find, however, that the prominence of the eyeball may, in a great many cases, show itself rapidly in a paroxysm and then disappear, we are led to ascribe that condition to a violent and active congestion. Thus might be explained the easy production as well as disappearance of the exophthalmos. But if the repeated congestions become hypertrophous, that is to say, if frequent congestions increase the nutrition of the cellular and adipose tissue within the orbit, these tissues gradually increase in quantity, and, by pushing the eyeball forwards, give rise to permanent exophthalmos. When I described the symptoms of the disease, I mentioned that serious disorders of the stomach and intestines had been noted, and pathological anatomy has shown that in some cases death had been brought on by hemorrhage into the stomach, the intestines, or the lungs. Lastly, the liver and spleen may be gorged with blood, increased in size, and I have myself found hypertrophic cirrhosis in two cases of exophthalmic goitre. The kidneys themselves have been the seats of grave alterations, fatty and amyloid degeneration, and have presented all the characters of Bright's disease. I need not add that we must make allowance for complications, and that, when there is an organic heart affection, in a case of exophthalmic goitre, the cardiac lesion is the cause of most of the passive congestions of parenchymatous organs.

I now pass on to the *differential diagnosis.*

There is no other complaint, gentlemen, which can be mistaken for exophthalmic goitre, for there is no other in which the three great symptoms pointed out by Graves exist. The insidious and sudden invasion of the malady, its distant or frequently recurring paroxysms, its variable duration, its course and nature, are as many distinctive characters, and if each of the principal phenomena of this morbid entity be examined one by one, you will find that they again facilitate the diagnosis of this complaint.

The exophthalmos is double, equal on both sides, and unaccompanied by strabismus, characters which distinguish it from all exophthalmos of orbital or cranial origin; the eyeballs are extremely mobile, and the eyes are lustrous, while this is not the case when the prominence of the eyeballs is due to an organic alteration of the heart, when the eyes are often dull and merely prominent. The eyes of shortsighted individuals have a peculiar appearance, which can be hardly described, but the date and course of the myopia will not admit of a doubt long.

Can hydrophthalmia be mistaken for exophthalmos? The former affection (supposing it to occur on both sides, which is an exceptional occurrence) may be recognized by the dilatation of the pupils, distension of the sclerotic by the effused liquid; the cornea bulges markedly in front of the sclerotic; the prominence of the eyes is due to the dropsy of the chambers of the eye, whilst in Graves's disease the prominence of the eyes is owing to their protrusion.

I need not dwell much on the differences in the origin, shape, and mode of increase, of exophthalmic goitre, of goitre proper, and goitre occurring during pregnancy. The first of these may develop itself, apart from all the conditions which give rise to endemic goitre; it reaches its greatest point of development in the right lateral lobe of the thyroid gland; it increases very rapidly, enlarges during the paroxysms, and is entirely made up of vessels; whilst, in endemic goitre, the throat-swelling consists in an hypertrophy of all the elements of the thyroid gland. Lastly, iodine, which often cures endemic goitre, frequently causes an enlargement of exophthalmic goitre. Goitre occurring in pregnant women is apparently due to pregnancy, while that of exophthalmos is seemingly cured by pregnancy, or reduced in size by the re-establishment of menstruation. I have perhaps dwelt too much already on these differential characters, but before I leave off this subject, I must remind you of the palpitation, the intensity of which constantly increases the prominence of the eyeballs and the size of the thyroid gland.

Exophthalmic goitre has been termed a cachectic affection. But we must first know what is meant by cachexia in general, for it is a term which has been often used, and its meaning has varied much at different periods. Nowadays, cachexia is understood to mean a deep alteration of the system consequent on morbid causes long inherent in the individual's constitution. This profound alteration is accompanied with important modifications in the proportion of the elements of the blood. There is diminution of the red globules, and increase of serum and fibrin; this modification constitutes anæmia or hydræmia. There are numerous cachectic conditions, produced by any morbid cause powerful enough, by acting on the system for a prolonged period, to bring on general debility and anæmia, such, for instance, as the scrofulous, the cancerous, or the syphilitic diathesis, or abundant hemorrhages from constitutional causes, &c.

If this is what is meant by cachexia, can we admit the existence of an exophthalmic cachexia? We cannot when the disease is temporary and curable, but we can when it resists all our attempts at curing it. For it is very evident that the blood crasis becomes deeply modified under the influence of great and prolonged disturbances of circulation. Oxygenation of the blood is imperfectly carried on in the systemic capillaries of exophthalmic individuals, whose pulse ranges from 120 to 160 beats in a minute. And the disturbance in hæmatosis necessarily gives rise to anæmia which shall be all the more marked in proportion to the duration of the complaint.

The dyscrasia depends, to a great degree also, on the impairment of the digestive functions. I need merely remind you of the strange bulimia which coexists with progressive emaciation. Thus, disorders of circulation and digestion give rise to anæmia, and prolonged anæmia is followed by cachexia. But this cachexia is only the last term of a morbid series which begins with multiple congestions, while these are themselves due to a peculiar modification of the sympathetic nerve, as I hope to be able to prove to you presently. To sum up, exophthalmic goitre is, in my opinion, a neurosis of the sympathetic, if not a complaint attended with a material lesion of the ganglionic nervous system.

This neurosis gives rise to local congestions, the proximate cause of which is a modification of the vaso-motor apparatus. This view is supported by physiological and pathological facts showing that there are local congestions of nervous origin. Thus, in chlorosis, a condition in which the nervous system and the blood-crasis are so deeply modified, we note flushings of the face and congestions of the uterus followed by hemorrhages, constituting what I have described under the name of *menorrhagic chlorosis.*

In hysteria, an essentially neurotic affection, we find delirium, coma, and protracted convulsions, followed by such a degree of congestion of the brain as to justify the practitioner in having recourse to bleeding. Can the profuse sweating and the copious flow of urine which occur in this complaint be understood except as the consequence of great congestion of the perspiratory glands and the kidneys? Lastly, Graves questions whether the sensation of choking which hysterical women have, and which has been compared to a ball rising in the throat, or to claws constricting the root of the neck, is not caused by sudden congestion of the thyroid gland. He states that several practitioners, whose scientific knowledge he valued, had been frequently struck with the swelling of the thyroid gland during hysterical seizures.

The congestion of the thyroid gland, in hysteria as in exophthalmic goitre, might be due to nervous paroxysms influencing the central organ of circulation, or some peripheral portion of the vascular system. The heart's action has been occasionally found to be accelerated and tumultuous during hysterical fits.

In one of the cases which I related to you at the beginning of this lecture, that of a young lady, from Clermont-sur-Oise, the swelling of the thyroid gland occasionally disappeared of a sudden to return a short time afterwards.

Other instances of local congestions dependent on the nervous system might be cited. Thus, an acute pain is frequently attended with redness and perspiration of the face; mental emotions cause blushing; modesty, anger, and love impart to the physiognomy a peculiar expression which is due to congestion of the face and eyes.

Now the existence of congestion in exophthalmic goitre cannot for a moment be called in question. The swelling of the thyroid, which increases or diminishes according as the heart's action is accelerated or slackened, the prominence of the eyes, their shining appearance especially during the paroxysms, the heat and moisture of the skin, the mental disturbances, are all phenomena clearly indicating the existence of a congestion. By a process of reasoning, one is led to admit partial congestions in other diseases. In nervous asthma, the oppression at the chest and the dyspnœa are accompanied by pulmonary congestions, and the rhonchi heard in the lung-cells and bronchi, and the critical expectoration which terminates the paroxysm, are proofs of their existence. The least mental emotion, a bright artificial light, can cause this local congestion to disappear, which is dependent on the nervous element that gives rise to the asthma.

I will relate to you in illustration of this the following case which came under Dr. Gubler's observation: An in-patient at the Beaujon Hospital, a young man of medium size, whose health was generally good, and who had no goitre, no emphysema, no nervous asthma of the ordinary kind, was from time to time seized with a fit of difficulty of breathing, during which he sat up in bed, clutching at the bars in order to assist his breathing, and had the aspect of a man threatened with asphyxia. His face became of a violet hue; his bluish nails and lips showed that his blood was venous, and his eyes, which were considerably injected, and opened very wide, protruded as in exophthalmic cachexia. These paroxysms of dyspnœa lasted a few hours, and the attack did not extend over more than a day or two. After that, he recovered his usual health, and his breathing was then so little impeded that he one day carried on his shoulders, from the bath-rooms on the ground-floor up to the third floor, a patient who could not walk, and did not pant for breath more than the most healthy individual would have done under the circumstances. Dr. Gubler could never make out, by the most careful physical examination, the existence of an organic lesion whatever, either of the heart or great bloodvessels, or of the organs of hæmatosis. During the paroxysms of dyspnœa, the respiratory murmur became feebler, and the chest resonance less clear; here and there a few rhonchi were heard, but those symptoms were very natural, if we admit that there was an internal congestion like that which could be seen externally.

This, gentlemen, is a good example of temporary congestion, very probably due to a nervous cause. But in inflammations also we find local congestions. Thus, in whitlow, the congestion is confined to the phalanx, or to the inflamed finger, and in general, unless there be general reaction, the arterial throbbings are restricted to the affected part: the individual's finger is feverish, if I may use the expression. Articulations attacked with rheumatism afford instances also of limited arterial and venous congestion. Those are inflammatory congestions, it is true; but you can every day observe physiological congestions, which are frequently directly brought on by nervous agency. Certain descriptions, lascivious sights cause the rapid, immediate, and temporary congestion of the sexual organs. In a physiological condition, this congestion constitutes erection; in a pathological state, it is priapism. Does not the structure of the organs which serve for the function of generation indicate that they are intended for congestion? The vascular tissue is then disposed in a special manner, and is called by anatomists erectile or cavernous tissue, or plexus susceptible of erection, as in the case of the ovarian plexuses, as shown by Professor Rouget's beautiful researches. It is a temporary congestion which causes catamenia in women, and the phenomenon of rut in the lower animals, and when a woman has reached the critical age, how can we account for the successive hemorrhages which occur just when the function is on the point of ceasing, unless we ascribe them to congestion of the vascular system? Now, all these hemorrhagic congestions depend more or less on the nervous system, for fright will be sufficient to arrest the menstrual flow, or to interrupt the local congestion necessary for accomplishing the act of generation.

There are, then, temporary physiological local congestions of nervous origin. But if you will review with me certain phenomena of natural history, you will find among them proofs of analogous congestions. In plants which reproduce themselves by buds, the sap, at a particular time, flows abundantly towards those parts which are to give off the buds, and gives rise, therefore, to a local congestion. If you place two cuttings from the same vine, one in an atmosphere of 4° or 5° C. below zero, and the other in a conservatory heated to 20° C. above zero, you will see the latter produce buds, while the former will give no signs of vegetative activity. Is not a local congestion produced by such a process?

In the lowest classes of animals, as the fresh-water polyp, the hydra is produced by gemmation. This mode of reproduction is accompanied by a local congestion which manifests itself by the growth of fresh polyps, of fresh hydræ, which in their turn give origin, by the same process, to new beings, and on the same trunk you will find several living generations.

The same thing happens in animals of a higher order; nature has disposed everything for the continuance of the species, and the phenomena of puberty, especially in the breeding season, manifest themselves by local congestions of the membranous combs and the webbed feet of salamanders, in the caruncles of the turkey-cock, the goitres and cutaneous combs of basilisks and of dragons, and even in that kind of bladder which is seen in a camel's mouth at the season of rut, and which has been shown by Savi to be due to the projection forward of the distended soft palate. These singular productions are, no doubt, proofs of the general expansion brought on by puberty, and which determines a state of true erection in the caruncles of the turkeycock and the comb of the cock, &c.

These temporary congestive phenomena may be observed in the female also. During the twenty-four or twenty-six days that she is laying eggs, a hen's crest is red, and her collar of a deep blue, but her crest gets wrinkled when she begins to sit over her eggs. Need I add, that during the period of rut, in most female animals, the congestion of the genital organs manifests itself by a flow of blood, and by an increase in the secretion of the glands which are annexed to those organs?

Since we find that in animals rapid congestions of a variable duration return regularly under the influence of a physiological nervous cause, can we not suppose that a morbid condition, which is characterized by rapid congestions, also varying in their duration, and recurring in paroxysms, may be due proximately to a modification of nervous influence, and should be consequently classed with neuroses? Besides, may not the congestion of the thyroid gland and of the eyeballs be regarded as a kind of pathological erection of those organs, and are we not justified by Mr. Claude Bernard's beautiful experiments on the sympathetic nerve, in comparing the morbid congestions of exophthalmic goitre to those abnormal congestions which that learned physiologist produces at will in different regions of the body by irritating or by cutting branches of the sympathetic?

Exophthalmic goitre is, in my opinion, a congestive neurosis; and it is a morbid entity, because it presents special phenomena; palpitation and congestions of the thyroid gland and of the eyeballs. It is a pathological variety of the great class of neuroses, with a paroxysmal course, and should be regarded as entirely distinct from ophthalmias due to organic diseases of the heart, while it cannot be confounded with goitre proper, of accidental or endemic origin.

I will now relate to you the particulars of a case which seems to me to throw the greatest possible light on the disease which we are now studying. The history of this patient shows, indeed, the undoubted influence of deep mental emotion on the generation of exophthalmic goitre, and some of the anatomical lesions found explain the influence of the sympathetic nerve on the functional disorders which are peculiar to the disease, and on the secondary structural lesions.

A woman, aged sixty years, is admitted into St. Bernard Ward on July 3, 1863, suffering from highly marked exophthalmos, and with the following history: In 1856, that is to say, seven years previously, she lost her father, whom she had attended at the cost of great fatigue. This loss caused her very deep grief. One night, after she had been crying for a long time, she suddenly felt her eyes swell and lift up her eyelids, her thyroid gland increase notably in size and throb in an unusual manner: she had at the same time violent palpitation of the heart. Simultaneously with the development of this train of symptoms, she bled copiously from the nose throughout the night. Four days after this, she consulted Mr. Desmarres, who recognized exophthalmic cachexia.

A year afterwards, she went to Africa, where she soon caught intermittent fever. She was admitted for this into the Algiers Hospital, and there, while under Dr. Bertherand's observation, her goitre, which had been very marked, diminished rapidly. The two other symptoms, palpitation and prominence of the eyeballs, continued, however, to the same degree.

The fever lasted for nearly a year, and brought on a cachectic condition, from which she never recovered completely. In January, 1863, she had an attack of angina pectoris, which lasted a few hours, with radiating pains in the right shoulder. It seems that a fortnight after her arrival at Algiers she had œdema of the lower limbs and ascites, which disappeared after four or five days. In 1863, this dropsical condition recurred on several occasions, but without continuing. When she came under my care, she showed no trace of œdema or ascites, and was in the following condition:

Eyeballs considerably prominent; the free margin of the lower lid is more than four millimetres distant from the transparent cornea, instead of being in contact with it. The upper eyelid does not cover a segment of the cornea, as it usually does, and is more than two millimetres away from it. In consequence of the protrusion of the eyeballs, the eyelids no longer form regular curves, but intercept between them an hexagonal space with obtuse angles. The patient is readily dazzled by a bright light, which makes her feel as if she were drunk; she is long-sighted in spite of the prominence of her eyeballs. On the night when her complaint set in, she lost her sight for a time entirely, and for nearly a whole year she was hardly able to bear artificial light. She was at that time unable to read or sew, but can now do both by using spectacles for long sight.

At the commencement of her illness, her eyes were still bigger than they are now. She could very imperftly close her eyelids, and even now, during sleep, her eyelids do not entirely cover the eyeball.

Her heart beats with force, but much less so than at the onset of her complaint; it is found, on percussion, to measure 13 centimetres in a longitudinal direction (about 5¼ inches), and 12 centimetres (4⅘ inches) transversely. There is no blowing murmur at the base or apex, systolic or diastolic; nor is any heard in the vessels of the neck, although the arteries pulsate with force.

Pulse 96; habitual dyspnœa.

The liver comes down a little beyond the false ribs.

The thyroid gland is of small size: there is no goitre.

Some time after the exophthalmos set in, the patient seems to have had a ravenous appetite for more than a year; she was obliged to take some food every two hours. She had a copious diarrhœa at the same time.

She menstruated for the first time at the age of 20; she had been chlorotic for five years previously, but menstruation, by degrees, caused the symptoms of chlorosis to disappear. The patient was menstruating at the time when the exophthalmos first came on; the catamenia were suppresed on that night, and have not since shown themseves.

Her father died of epileptiform seizures, which had occurred for several years. On admission, she complained of neuralgic pain in the ophthalmic nerve, in the occipital, and the first two cervical pair of nerves. Since the commencement of her illness, she has had trifling epistaxis every month, and about the same period.

She was ordered digitaline and Baumé's bitter drops.

When she left the hospital in the month of August, she suffered less from palpitation of the heart, but her eyeballs were as prominent. She was readmitted on December 3, in a weaker condition.

Six days afterwards, she was suddenly struck with apoplexy after a few trifling cramps in the legs; she fell out of bed without uttering any complaint, and was picked up in a state of asphyxia, with rigidity of the four limbs. A few hours afterwards, the left side got better, but the right side continued to be powerless, although not rigid. The patient did not recover consciousness, and died in the most complete state of coma, twenty-four hours after the attack.

On examining the body, a large hemorrhagic centre was found in the left hemisphere of the brain, near the corpus striatum and optic thalamus.

The heart was of very large size, the walls of the left ventricle being the most hypertrophied. The free edge of the mitral valve was thickened, but there was no constriction of the aperture or incompetence of the valve. The aortic valves were a little roughened along their free borders, but were perfectly competent.

The aorta was incrusted with a calcareous deposit along its arch, and there were atheromatous patches in its descending portion.

The vessels at the base of the brain presented, however, no appreciable alterations to the unaided eye; the capillaries in the vicinity of the hemorrhagic centre, and in that centre itself, were examined under the microscope by Mr. Peter, and were found free from calcareous and atheromatous changes.

The spleen was of voluminous size, measuring 12 centimetres in one direction, and 6 in the other. The capsule was not thickened; the splenic tissue was firm, and on section the glomeruli of Malpighi were found to be hypertrophied.

The liver was of nearly normal size, but of cirrhotic tint, and in an incipient lobular condition; its fibrous capsule was thickened, the trabeculæ considerably hypertrophied, and the tissue of the organ indurated. On microscopical examination, the hepatic cells were found to be normal, but to have diminished in quantity, and the interstitial connective tissue to be hypertrophied.

The kidneys had not increased in size; their capsule was not thickened; they had a granular aspect, were red on section, and showed traces of interstitial nephritis.

The thyroid body was of very small size, and its lobes hard, almost of the consistency of scirrhus; they had a lobulated quasi-cirrhotic aspect, owing to the retraction of their fibrous elements. On section, the glandular tissue was interrupted, and, as it were, squeezed by trabeculæ of an extremely thick fibrous tissue, of a mother of pearl color, and creaking when cut.

The thyroid arteries were small, not flexuous in the least, and showed no calcareous or atheromatous changes.

The eyeballs were thrust out of the orbit by the amount of cellular and adipose tissue which nearly filled the socket, was redder than usual, and contained a good deal of fat. The ophthalmic artery was not tortuous, nor was it abnormally large; the eyeballs, when removed from the cushion of fat on which they lay, were not of larger size than in health. They were not altered in structure.

The bones of the skull were extremely vascular, and were of more than double their natural size; in fact, they were all hypertrophied.

The cervical ganglions of the sympathetic were carefully dissected and examined on both sides by Dr. Peter and Dr. Lancereaux, clinical assistants at the Hôtel-Dieu. The superior and middle ganglions were of normal size and aspect. But the lower one, especially on the right side, was not only of larger size than usual, but was much redder also; numerous vessels were seen to ramify on its outer surface and throughout its interior, when examined with a power of 50 diameters. When examined under the microscope, a great many vessels were seen in its interior, with a thick admixture of connective tissue, and, in the midst of its fibres, nuclei and fusiform cells. There were a great many fat-globules; the ganglionic cells were very few in number, small, and with a mulberry aspect; some of them were reduced to a mere granular condition; the nerve-tubes were in small numbers.

All these details were well made out on examining a transverse section, with a power of 300 diameters. A very close network of fibres and of connective tissue was thus seen to inclose pretty narrow spaces containing small nerve tubules compressed, and, as it were, crushed by connective tissue. The examination thus made out two facts, to which I wish particularly to call your attention, namely, predominance of the connective tissue, and diminution of the nervous elements.

The cardiac plexus showed no apparent alterations, except perhaps some redness of the branches forming it. The ganglion of Wrisberg was unfortunately destroyed on an assistant cutting through the aorta. This case seems to me to be in the highest degree interesting, on account of its mode of invasion, its progress, the pathological changes found after death, and the probable connection of the lesions. It shows, in the first place, the immense influence of violent mental emotion on the production of exophthalmic goitre. In the course of a single night, the three great symptoms of Graves's disease showed themselves: palpitation, swelling and throbbing of the thyroid body, and exophthalmos. Of all morbid phenomena, congestion is the only one which can develop itself with such rapidity; and we have a proof that there were really multiple congestions present in the fact that the patient bled profusely from the nose at the same time, thus showing that there was simultaneously hemorrhagic congestion of the pituitary membrane.

After a year, the swelling of the thyroid body disappeared, but the palpitation and exophthalmia continued, so that the disease became incomplete in its manifestations. This is far from being rarely the case, but it is interesting to see in the same person this complaint assume these various symptomatic forms.

I just now stated that the disease had set in with rapid and multiple congestions, but all congestion which does not disappear in a short time brings on hemorrhage, effusion, inflammation, or what is termed hypertrophy. Now, this patient had frequent attacks of epistaxis and diarrhœa. Chronic congestions, in the majority of instances, cause interstitial plastic exudations, and from the organ having visibly increased in size, its parenchyma is supposed to have hypertrophied. The truth is, however, that there is, on the contrary, atrophy of the proper substance of the organ. For the plastic lymph becomes organized, is converted into fibrous tissue, and becomes a parasitical element, which develops itself by choking the proper tissue of the organ, or which is arrested in its evolution on account of its lower grade of vitality, and degenerates into fat. In other words, and in the language of the German school, hyperæmia may lead to exudation of a plasma in which the elements of cellular tissue get developed, namely, nuclei, fusiform cells, and fibres: there is proliferation of the connective tissue, and then one of two things may happen, either the proliferation goes on, and the connective tissue becomes changed into fibrous tissue, which, from its exuberant growth, as much as from the retractile force with which it is endowed, determines constriction of the parenchyma, or it undergoes retrograde changes, becomes infiltrated with fat-globules, and finally converted into adipose tissue. In the former case, cirrhosis is the result, in the second, fatty degeneration. Well, in the woman under my care, the cirrhotic process is the one which predominated, as shown by the condition of the thyroid body and of the liver. The tissue of the thyroid body was intersected by exceedingly thick fibrous partitions which compressed the globules, and gave rise to cirrhosis of the organ. The liver contained also a good deal of fibrous tissue, and the lobules were beginning to atrophy.

The kidneys exhibited what is termed interstitial inflammation, namely, an exudation of fibrin between the convoluted tubules of the cortical substance, and perhaps Bright's disease would have come on, if the disease had lasted longer. The heart was evidently hypertrophied; its muscular fibres were more abundant than usual, and there was no predominance of fatty tissue in it.

We found, therefore, cirrhotic atrophy of the thyroid body, consequent upon great and prolonged congestion, incipient cirrhosis of the liver, hypertrophy of the heart, hypertrophy, with hyperæmia, of the cellular tissue within the orbit, and hypertrophy of the cranial bones.

I now proceed to inquire into the proximate cause of these congestions, and their consequences. We know that excision of the upper cervical ganglion is followed by hyperæmia of the ear, and that paralysis, or weakness, of the vaso-motor nerves causes relaxation of the coats of bloodvessels, blood-stasis, and consequently congestion. I can hardly believe that excessive grief did not cause in my patient a primary modification of her ganglionic nervous system. No appreciable change was found, it is true, in some of the cervical ganglions, but the lower ones, that on the right side particularly, were congested, and there was in the latter proliferation of the connective tissue, and diminution in the number and size of the nerve-tubules. Such a structural lesion must necessarily have interfered with the functions of the organ, and have been followed by consequences somewhat analogous to those caused by excision of the ganglions, that is to say, hyperæmia and all its results.

In conclusion, then, this autopsy authorizes us to believe that the very numerous functional disorders which occur in Graves's disease are either due to temporary congestion of the sympathetic nerve or to a permanent structural alteration of the ganglionic nervous system; either of which becomes the origin of transient congestions or of irreparable lesions in various organs, which hypertrophy or atrophy in consequence of this by a process which I explained to you.

I regret not to have been able to examine all the divisions of the sympathetic nerve; my investigations were stopped by unavoidable circumstances, but I believe that a fertile mine remains to be explored in that direction, and I strongly recommend industrious men to make the attempt.

The *treatment of exophthalmic goitre* has been complicated in the majority of instances. Stokes says that he has used with success lowering remedies and preparations of iodine. When the nature of a disease is not understood, its symptoms can be alone treated. Hence, against this form of goitre, which was not recognized as a variety, iodine was used *internally* as well as *externally* by nearly all observers. But it was soon abandoned by nearly every one, as under this plan of treatment it was found that the symptoms grew worse.

Sir Joseph Oliffe has lately communicated to me the history of a young lady aged twenty-six, who for several years has been afflicted with exophthalmic goitre. She was treated with iodine, which brought on a slight diminution in size of the thyroid body, but the eyeballs remained prominent and the iodine caused very rapid emaciation and so great a debility that the patient was almost unable to take any exercise at all. She had only taken internally, however, about 25 or 30 grains of iodide of potassium in the course of three weeks. The medicine was dropped, and Sir Joseph prescribed antispasmodics and tonics, upon which the patient immediately felt better. It was only after the lapse of two months, however, that she regained her strength, and was enabled to resume her usual mode of life. The exophthalmos was as marked as ever.

Dr. Oliffe thought, and I might have done the same, that this young lady had suffered from iodism. I cannot hold that opinion now, however, although it has Rilliet's authority in his favor. For when we find that such a small dose of iodide of potassium as one-fifth of a grain a day is sufficient, at Paris and Geneva, to produce iodism, and, according to Rilliet, that a mere sojourn at the sea-side can bring on the same symptoms, I feel inclined to believe that the persons thus affected were the subjects of exophthalmic goitre. Otherwise, how is the contradiction between everybody's experience and the cases of iodism published by Rilliet to be explained away, unless we admit the existence of a morbid element, which, under the influence of a certain remedy, gave rise to more marked manifestations? Iodide of potassium is given every day in large doses, in every country, at Paris as well as at Geneva, in doses of twenty to forty grains in the twenty-four hours. No accidents ever occur, although the drug be continued for several weeks in the same doses, and if, on the other hand, we should find that nearly infinitesimal doses bring on one of the chief symptoms of iodism—an increase in the size of the thyroid gland, with bulimia and various nervous symptoms—I am of opinion that such exceptional cases should be regarded as examples of exophthalmic goitre.

Iodine, therefore, has, I believe, been wrongly accused by Rilliet. We all knew—and Rilliet knew it as well as we do—the great improvement which follows the use of iodine in ordinary goitre; but it should be known also that iodine is a dangerous remedy to use in exophthalmic goitre, and that it can give rise to paroxysms. When in a case of goitre, you find palpitation, protrusion of the eyeballs, and a strange look of the eyes, never give iodine. You have to deal with exophthalmic goitre, and iodine will only increase all the symptoms of the disease.

In some rare cases, however, preparations of iodine are borne without any ill effects, and even with a semblance of improvement by persons suffering from Graves's disease.

About the middle of June, 1862, Dr. Bruneau (of Villaines) sent to me a lady, who generally resides in Paris. Her history is of sufficient interest to deserve being related in some detail. She presents us, besides, with an instance of acute exophthalmic goitre. She is thirty-five years of age. About the beginning of the year 1861, she felt curious sensations about her heart, which she compared to a kind of itching. Her heart, from that time, and ever since, has beaten with greater rapidity, and even after she had rested in my consulting-room for more than an hour, I counted 120 pulsations in the minute.

Since the month of February, 1862, the catamenia have diminished in quantity, and become somewhat less pale. About the middle of March, however, she noticed that her throat swelled, especially on the right side, and she had pain in the eyeballs. Eight days later she noticed herself, like everybody else, the prominence of her eyes. She suffered from nervous irritability and difficulty of breathing, and although her appetite was markedly increased, she lost flesh. The bronchocele and the exophthalmos progressed so rapidly that, in the space of six weeks, they reached the point at which I saw them. The doctor who attended this lady in Paris advised her to go on a visit to her friends at Villaines (Mayenne), and to take daily twenty grains of iodide of potassium and some iron pills. The influence of country air and perhaps the treatment made the patient regain her strength. The point, however, on which I wish to insist is that the goitre diminished a little, in spite of the use of iodine in large doses, although, according to the patient's statement, the exophthalmos showed a tendency to increase. After this treatment had been carried out for a month, the disease remaining stationary, all remedies were ceased, and in a few days, the thyroid body increased again to its former size.

When I examined this lady, I found a large bronchocele (larger on the right than on the left side), and considerable protrusion of the eyeballs, the left of which was rather painful when pressed; she felt a sensation as if dust had got into her eyes. Strangely enough, she had become long-sighted since her complaint had set in three months ago, and she could not read or sew, except by holding the object at some distance from her.

When I held the bronchocele between my fingers, I had a sensation of expansion, and on listening with the stethoscope, I heard over the swelling a double bellows-sound, which was single above it, on a level with the bifurcation of the carotid artery, and corresponded to the ventricular systole. This afforded a proof that the double bellows-sound heard over the bronchocele was not merely due to transmission of sounds originating in the common carotid, since the arterial bruit was single.

There was no hypertrophy of the heart, nor any abnormal valvular bruit.

My object in relating this case was to show you that although in the great majority of cases iodine exerts a bad influence on the exophthalmic neurosis, it seems occasionally to produce temporary improvement of the patient's condition. I would not, indeed, leave on your mind the impression that iodine is invariably hurtful in Graves's disease. I once attended with my excellent friend, Dr. L. Gros, who was the first in France to call attention to Graves's disease, a man, about fifty years of age, whose condition was singularly improved by the prolonged administration of iodide of potassium.

This case had not, however, converted me with regard to iodine, when I happened to see a case in which an error which I committed enlightened me.

In the course of October, 1863, I was consulted by a young married lady, who habitually resides in Paris. She was suffering from a subacute exophthalmic goitre. The bronchocele was of great size. When I examined her for the first time, although I had let her rest for a long while, and although I repeated the examination several times, and at sufficiently distant intervals, so as to make sure that she was no longer under the influence of emotion, I still found that her heart beat at the rate of 140 to 150 times in the minute. I recommended hydropathy, and I wished to administer at the same time tincture of digitalis, but, preoccupied with the idea that there would be some danger in giving iodine, I wrote iodine instead of digitalis, so that the patient took from 15 to 20 drops of tincture of iodine a day, for a fortnight. When she then came back to me, her pulse was only 90. I found out my mistake, and I substituted tincture of digitalis for that of iodine, but, after another fortnight, the pulse had again gone up to 150, so that I at once returned to the iodine.

Notwithstanding these exceptional cases, however, bear in mind that iodine generally does harm in Graves's disease.

Now, what does chemical experience say respecting the preparations of iron? The patient sometimes comes to you in a state of very marked anæmia, with a pale complexion, with œdema, bellows-murmurs over the base of the heart, extending up the vessels of the neck. Steel seems to be indicated then, and nearly all observers have recommended it. But read the cases that have been published, and you will find how little good steel has done, when it did not do much harm, and note that it was nearly always given together with digitalis, while the patient was kept on low diet, and topical applications were used to the swelling to prevent congestion. The administration of iron would probably have been followed by still worse results, if it had not been counterbalanced by the influence of the other drugs, by digitalis in particular. As to me, I believe that iron does harm in exophthalmic goitre, and you will concur with me if you recall to mind how we were compelled to stop it in the case of the woman who lay in bed No. 34, in St. Bernard Ward, who soon became quieter, and had less palpitation when I substituted tincture of digitalis for the iron.

Dr. Graefe has before me pointed out the dangers and counterindications of a treatment by preparations of iron. It should be avoided, he said, when there is considerable vascular excitement, and the pulse is more than 100 or 110 in the minute, because all the symptoms are intensified by it. We have seen that in some cases, however, the administration of iron is not followed by bad results, and the case which I related just now is an instance in point. Bear in mind also the happy results which I obtained in the case of the boy T. from bleeding, drastic purgatives, digitalis in large doses, and the application of ice to the thyroid swelling.

I can, from experience, recommend you to have recourse, in this singular affection, to bleeding, digitalis, and hydropathy. When I advise bleeding, I do not do so in an absolute manner, and, of course, not with the view of combating the anæmia and the nervous element of the disease. It is only with one end in view, namely, that of averting the imminent danger which may result from congestion of the thyroid body, of preventing asphyxia by depleting the bloodvessels, and of quieting palpitation. The first indication during a paroxysm is to prevent suffocation. This is effected by diminishing the size of the swelling; the continuous application of cold prevents the flow of blood to it, and artificial congestion may be induced in other regions, as the lower extremities, for instance, by means of Junod's boots, large mustard poultices, &c. By and by, when the paroxysm is over, and there is no danger of suffocation, act on the supposed cause, on the nature of the disease. Exophthalmic goitre is, in my opinion, a neurosis which principally affects the heart and the supradiaphragmatic arteries; while Stokes thinks that it is pre-eminently a cardiac neurosis, characterized by violent palpitation. Administer digitalis, therefore, the sedative par excellence of circulation. Be not afraid of giving it in large doses; yet, feel your way with it, and only stop when you have induced symptoms of incipient poisoning, when the patient complains of vertigo, of cephalalgia, and of nausea.

The pulse will also furnish you with indications when the medicine should be given in small doses, or should be dropped altogether. When the pulse falls to 60 or 70, stop the digitalis, or diminish the doses.

When the patient's life was in danger, I have obtained good results from the administration of tincture of digitalis, given every hour in doses of eight or ten minims. You have not to fear in such cases the accumulating effect of the medicine. The boy T. took without harm 109 drops of tincture of digitalis in the space of only ten hours.

I will now tell you what good results may be expected of a treament by hydropathy. Three years ago I was summoned to Crest, in the department of the Drôme, to see a lady who, for the sixth time during the last six years, presented all the symptoms of exophthalmic goitre: prominence of the eyeballs, swelling of the thyroid, palpitation of the heart, pulsation, with bellows-murmur, of the carotids, obstinate vomiting, and congestion of the liver. In 1858, I was consulted again, and I advised hydropathic treatment. Dr. Gilbert-d'Hercourt superintended the treatment himself in his establishment at Longchêne. Bearing in mind, says Dr. Gilbert-d'Hercourt, in his relation of the case, that all the relapses of Mrs. B. had been preceded by a diminution or a complete suppression of the catamenia, he decided on carrying out the hydropathic treatment in such a way as to bring on congestion of the uterus, and thus produce a healthy revulsion. The hepatic engorgement soon disappeared; the protrusion of the eyeballs and the swelling of the thyroid became less and less marked. Mrs. B. was able to resume her ordinary occupations, and to sing for several hours without fatigue. In 1859, in the month of June, she had another relapse, or rather a fresh paroxysm, preceded by suppression of the menses. A hydropathic treatment again mastered the disease, and I have since been able to verify the perfect health of Mrs. B. She walks and sings without getting out of breath, she no longer suffers from palpitation; her pulse is not so frequent as it used to be; her appetite is good, digestion easy, and she sleeps well.

Hydropathy has several times been followed with the same good results in similar cases; it is a plan of treatment which should not, therefore, be neglected. You know what good effects may be obtained from it in anæmia, chlorosis, and hysteria, and you are aware also that many visceral engorgements have been cured by it. You must, therefore, think it quite natural that exophthalmic goitre, which I have regarded as a congestive neurosis, should be favorably modified by it.

The continuous application of ice over the precordial region, and to the thyroid body, is a powerful measure which I cannot too strongly recommend to you.

Now is perhaps the best time for dwelling on the therapeutic indications, and for analyzing the reasons which make certain measures successful in the treatment of this complaint. I will be brief, and will merely remind you that bleeding and revulsive applications to the extremities are employed against the congestion of the thyroid gland, avert the cause of asphyxia, while digitalis quiets palpitation, diminishes the frequency of the cardiac and arterial pulsations, and the hydropathic treatment offers the twofold advantage of causing a violent revulsion to the skin and of rendering innervation and nutrition more perfect. I should perhaps dwell more on the necessity of re-establishing menstruation. This is certainly an important therapeutic indication, but in order that it should succeed, one must wait till a hemorrhagic tendency shows itself in the uterus. It would be bad medicine to try anyhow and at all times to bring back menstruation. One should know how to wait and to act only when nature seems to indicate it. Revulsives may then be had recourse to, and a few leeches be applied to the lower limbs, &c.

Lastly, if you have not succeeded in averting the paroxysm, and if it should be accompanied by a sense of choking threatening life; if revulsives and the application of ice to the swelling do not remove the risk of asphyxia, you may have recourse to tracheotomy. But bear in mind that the operation cannot be performed under more serious circumstances, and that the patient may die under the surgeon's knife. I have laid much stress already on the extreme vascularity of the thyroid gland in exophthalmic goitre, and I have known a case in which death from hemorrhage occurred during the operation: you should take every precaution, then, against hemorrhage. With this object in view, Mr. Demarquay proposes the use of the écraseur, which has done so much good service since its invention by Chassaignac. One of the great advantages of this method is to secure one in nearly every instance against the grave hemorrhages which so frequently follow upon the use of the knife in regions where ligatures cannot be easily applied.

The following is the manner of operating recommended by Mr. Demarquay: The thyroid gland is to be exposed by dissection, and all the subcutaneous and the subaponeurotic vessels liable to bleed should be secured by ligature; and after this the chain of the écraseur is to be passed underneath the isthmus of the thyroid. If the removal of the gland be accomplished in this manner without hemorrhage, the trachea can next be divided, and a canula introduced into it.

Mr. Chassaignac thinks that the knife need not be used at all in this case, and he recommends instead that the skin be pinched in a transverse direction, and that all the soft parts situated in front of the trachea be included in the chain of the écraseur. There would then be two stages in the operation: in the first, all the soft parts would be cut through by the écraseur; in the second, the trachea would be opened with a knife, and a canula introduced.

These are, as you may see, two different processes of one and the same method, namely, removal by means of the écraseur. This method offers the immense advantage of considerably lessening all risks of hemorrhage, and future experience will pronounce on its value. But whichever mode of operating you may prefer, never forget to get ready beforehand all the means which medicine and surgery place at your disposal for arresting bleeding, which in these cases may in a few minutes endanger the patient's life.

LECTURE LIX.

PROGRESSIVE LOCOMOTOR ATAXY.

(PROGRESSIVE LOCOMOTOR ASYNERGIA.)*

§ 1. *Definition.—Prodromata: Pain, Disorders of Innervation.—Nocturnal Incontinence of Urine.—Spermatorrhœa.—Paralysis of the Third and Sixth Cranial Pair.—Diplopia.—Amaurosis.—Symptoms: Defect of Co-ordination of Movement with Retention of Muscular Power.—Transient, Persistent Pain.—Impotence.—Deafness.—Varieties: Painful Ataxy; Ataxy more marked on one Side of the Body.—Etiology: Hereditary Influence.—Symptoms of the fully-developed Disease.—Disorders of Progression.—Spasms.—Variable Anæsthesia, which is sometimes completely Absent.—Return of the Paralytic Symptoms.—Progressive Locomotor Ataxy may be Imperfectly Developed.—Course of the Disease.—Prognosis Extremely Grave.—Locomotor Ataxy independently of Cutaneous and Muscular Anæsthesia.—A few Words respecting Sir Charles Bell's Muscular Sense and Gerdy's Sense of Muscular Activity.—Differential Diagnosis between Progressive Locomotor Ataxy, Various Forms of Paralysis, and Cerebellar Ataxy.*

GENTLEMEN: You have had occasion to see several cases of *progressive locomotor ataxy* in my wards, and I have repeatedly called your attention to them. In 1861 and 1862 I devoted several lectures to the study of this singular malady, and I return to-day to the subject because recent discussions have imparted fresh interest to it. Formerly the pathological anatomy of the disease was very incompletely known, but cases that have occurred in my own practice, and others recorded by physicians of note, have since supplied us with interesting facts which I desire to bring before you. First, however, allow me to give Dr. Duchenne (de Boulogne) the credit which is due to him, and which has lately been contested by some. There is nothing surprising indeed that, before Dr. Duchenne wrote on *progressive locomotor ataxy*, cases, evidently referable to this disease, should have been seen and recorded by others. Locomotor ataxy is not a new disease, and Dr. Duchenne has never pretended that he was the first to suspect its existence. Such cases, however, had not been seen in their true light, and the few descriptions of the disease given by foreign authors, under different and more or less appropriate names, were, to say the least, very incomplete. I do not even except that of Professor Romberg (of Berlin), whose monograph on the subject has, however, been called a masterpiece of conciseness and exactness. I admit that it is concise, but I deny that it is exact, both as regards the description of the symptoms and the pathological anatomy. I make this assertion after a careful perusal of the translation, which Dr. Zubelsky (of Varsovia) kindly wrote for me, of the chapter on Tabes dorsalis, in the edition of 1851 of Romberg's work.

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* The word *asynergia* would be better than that of *ataxy*, which has already a

Even admitting, for the sake of argument, that the researches of German and English physicians on this subject be as complete as they are stated by some to be, it must be acknowledged that in France, as well as in England and Germany, the attention of the profession has been drawn to this subject only since Dr. Duchenne (de Boulogne) published his memoir.[*] It is to him, therefore, that we are really indebted for the knowledge we now possess of an affection which, until then, had been confounded and mixed up with such very different diseases.

As to the name, *progressive locomotor ataxy*, given to the complaint by Dr. Duchenne, I accept it, however long it may be, because it conveys to the mind, I believe, the most complete idea of the disorders of locomotion which constitute the most striking phenomena of the disease. The terms *atrophy of the posterior columns of the cord*, and *tabes dorsalis*, which have been suggested in its stead, are not better, in my opinion. The name of *tabes dorsalis* has only its antiquity to recommend it, and it has the disadvantage of having been applied by the ancients, and by others, since, to very various affections, especially, as in the works of Hippocrates, to special paralysis brought on by sexual excess.[†]

I likewise reject the denomination of *atrophy of the posterior columns of the cord*, first, because it is as long as the one which I adopt, and secondly, because it is not so precise as some would have us believe. For, as I shall have to tell you when we come to the pathological anatomy of the disease, cases have been recorded in which the distinctive characters of progressive locomotor ataxy have been present, and that during several years, whilst

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definite sense in medical language, different from its meaning in locomotor ataxy; but as this latter term has been almost universally adopted in France, I have hesitated before changing it.—TR.

[*] "De l'Ataxie locomotrice progressive" (Archives Générales de Médecine, Décembre, 1858, Janvier, Février, et Mars, 1859), and "le Traité de l'Electrisation localisée," by Duchenne (de Boulogne), 2^{e} édition, Paris, 1861, pp. 547–620.

[†] Consult on this point, chap xiv De Internis Affectionibus, and chap. xix of lib. ii, De Morbis, in the works of Hippocrates.

after death no material alteration of the posterior columns of the cord has been found.

Now, gentlemen, what is meant by *progressive locomotor ataxy?*

According to Dr. Duchenne (de Boulogne), the fundamental characters of the disease are—"Progressive abolition of the faculty of co-ordinating movements, and apparent paralysis contrasting with the integrity of the muscular power."* This is a very incomplete definition, however; but, for the present, I shall not attempt to give you another myself, for definitions in general—and in medicine perhaps more than in any other science—are not easily framed. They become still more difficult, nay impossible even, when they must be applied to a recently known disease, or, at least, a disease which has been but recently studied, and presenting an infinite variety in its manifestations, and the order of their sequence.

If you ask an individual suffering from ataxy to walk, he staggers, makes great efforts to maintain his equilibrium, and feeling that his muscles do not respond to the influence of his will, he seeks for a point of support. It is especially at starting that this difficulty in maintaining the equilibrium of the body is remarkable. When once started, the patient is able to walk, although he does it badly, and throws his legs about to the right and to the left. Occasionally he loses his equilibrium entirely and falls down, unless he be supported, especially when he turns round. Formerly, a man whose gait was uncertain, whose legs were thrown to the right and to the left, was set down as suffering from paralysis, and if no serious impairment of the intellect were present, the disease was localized in the cord, and called paraplegia. No physician, before Dr. Duchenne (de Boulogne), ever thought of testing the muscular power of these so-called paralytic patients. The idea first occurred to this *savant*, and he it was who detected that their muscular power was considerable, and that they only lacked the faculty of co-ordinating their movements. You have yourselves examined my patients in St. Agnes Ward who are suffering from locomotor ataxy. The one in bed No. 2, is a young man whose muscular power is so great that his limbs cannot be flexed or stretched against his will. Although his gait be so vacillating, he is strong enough to bear on his shoulders, when standing, a weight of 160 lbs., on condition, however, that he may rest on a friend's arm, or on a piece of furniture; and I showed you that he could carry on his shoulders several students in succession. Surely this is not muscular weakness, and still less paralysis.

At No. 23, the patient was about 40 years old. He too, looked as if he were paralyzed, for his gait was tottery, the least touch sufficed to throw him down, and he could not walk across the ward, except by going from bed to bed. When sitting or lying down, however, his limbs could not be extended or flexed against his will.

Look now at that woman in bed No. 23, St. Bernard Ward, and at that man lying at No. 11, St. Agnes Ward. Both of them possess considerable muscular power, yet when they are up, even though they be propped up under the arms, they cannot move a single step, they thrust their legs forwards, backwards, and laterally, in a strange disorderly manner. When their eyes are closed, this disorder knows no bounds; their movements become so extravagant that they baffle description, as you saw yourselves. If the strength of their muscles be tested, however, whilst they are in a sitting or a lying posture, one is surprised to find it unimpaired, or nearly so, and to find also that unless considerable efforts be made, the limbs of these so-called paralytics cannot be flexed or extended against their will.

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* "De l'Electrisation localisée et de son Application à la Pathologie et à la Thérapeutique." 2^e édition, Paris, 1861, p. 547.

The difficulty which these patients have in co-ordinating their movements, is still more marked when they have not the sense of sight to guide them. But it must be observed, however, that the sight can never completely remedy the want of co-ordination in ataxy, whilst this obtains in cases of the mere loss of tactile sensibility, as we shall more fully state when treating of the differential diagnosis. The difficulty which the patients have in guiding their movements is much more marked when they first start, and when they turn round. It diminishes when they can rest on something, especially on a friend's arm. In some rare instances, the disease is restricted to this defect in the power of co-ordinating voluntary movements, and is unaccompanied by impairment of muscular sensibility, by analgesia, or by cutaneous anæsthesia. In other words, all the functions of the cerebro-spinal system are performed normally, with the exception of the faculty of co-ordination.

Note, however, that this form is very rare, I may even say, exceptional. Since my attention has been drawn to locomotor ataxy, I have seen more than fifty cases of the disease, and in three only have I seen it consist merely in a want of co-ordination. One of these cases occurred in a gentleman eighty years old, residing at Tours, and a patient of Dr. Duclos. He was suffering from paraplegia, and as his case seemed to his medical attendant to differ from one of ordinary paraplegia, I was consulted. The patient looked in excellent health, although he had not for a long time been able to walk. He generally sat up in a chair, and for the last twelvemonth he had had some paralysis of the bladder. A few days previous to my visit, Dr. Duclos had been struck with the extraordinary suddenness and violence with which the patient had stretched out his leg, when asked to do so. On my testing, in my turn, his muscular power, I could not succeed in flexing or extending his legs against his will. I then made him get up on his feet, and by letting him rest on my arm he was able to carry on his shoulders his own medical attendant who had pronounced him to be paralyzed.

The mistake, however, was very excusable, and every one made it a few years ago. Even one of the most distinguished professors of the faculty, a man of very extensive knowledge, was deceived himself in the case of a patient whom we saw together at Tivoli. But he was easily convinced, however, that there existed no muscular paralysis, when there was only a want of co-ordination. This case of the old gentleman, was one of simple ataxy, and as to the slight paralysis of the bladder, it could be ascribed to his advanced age. In 1860, however, I was asked by my excellent friend Dr. Deguise to see with him a superior cavalry officer, who was very markedly ataxic. The sensibility of the skin, the muscles, and joints had undergone no modification. There was nothing wrong with the eyes, the bladder, or intestines. The case being clearly one of uncomplicated ataxy, I showed it to Dr. Duchenne.

Now, how does the disease begin?

Its accession is marked by various neuroses, and one of its premonitory symptoms is *pain*. How many patients, that have been sent to the baths at Néris, Bourbon-Lancy, Bourbon-l'Archambault, and Bourbonne, for rheumatic or pretended neuralgic pains, and who derived no benefit from the baths, were perhaps suffering from the pains which usher in locomotor ataxy? The characters of these pains are peculiar: they come on and go off with the rapidity of lightning or of the electric spark; in some cases, however, lasting from a few seconds to a minute. They recur ten, fifteen, twenty times in an hour, and they come on in paroxysms several times in the year, or in a month, often without any other exciting cause than variations of temperature. At other times they are of a boring character, and either simultaneously or successively attack limited, perfectly well-defined spots, which the patient quickly compresses or rubs so as to diminish the pains. When the disease is confirmed, as I shall tell you by and by, these pains may become continuous and gradually increase in intensity. These have been described by some authors under the names of *general neuralgia* and *neuralgic rheumatism*, but Dr. Duchenne was the first to point them out as the prelude of locomotor ataxy. They are the most constant premonitory symptom of the disease, and yet in September, 1861, I had under my care at the Hôtel-Dieu a man aged 37, suffering from well-marked ataxy, who had never had any pain.

Nocturnal incontinence of urine is another neurosis which may precede locomotor ataxy. More frequeptly, and in nearly half the cases which have come under my observation, there had been spermatorrhœa. The seminal losses were either diurnal or nocturnal. In the former case they occurred chiefly during defecation, from compression of the vesiculæ seminales. In Lallemand's work on spermatorrhœa, you will find several cases of paraplegia which were certainly cases of locomotor ataxy. At No. 23, St. Agnes Ward, was a patient of mine who had for years been subject to spermatorrhœa, which had exhausted him considerably. Frequently these nocturnal emissions are accompanied by erection and voluptuous sensations; but in some cases there is anaphrodisia instead of spermatorrhœa, marked by an imperfect erection or a complete absence of sexual appetite.

There is again another form of genital neurosis in ataxic patients; namely, a singular aptitude for repeating the venereal act a great many times within a short period. This is an abnormal condition in man; for if birds, and some mammalia, such as the ram, the bull, and deer, can have connection rapidly, and repeat the act at short intervals, in man the act must extend over a certain time, and if performed too quickly it indicates a deviation from health. Men who possess this semblance of exaggerated virile power are often subject to spermatorrhœa. Only yesterday you heard the patient lying in bed No. 2, St. Agnes Ward, confess that before his admission into the hospital he was able to have connection as many as eight and nine times in one night. Recently again I saw in my consulting-room a gentleman in the prime of life, and suffering from ataxy, who told me that he could have connection eight or ten times in the twenty-four hours. That this condition is abnormal is proved by there having most frequently existed incontinence of urine at some previous period, and that involuntary seminal emissions often occur.

Certain forms of transient paralysis also precede the want of co-ordination. I was lately consulted by a gentleman from the Côte-d'Or, who nine months ago was suddenly seized with left hemiplegia. There was no impairment of the intellect, and he could resume his occupation at the end of a week. The hemiplegia could not have been due to cerebral hemorrhage or softening, nor was it probable that it could have been caused by cerebral congestion, since there had been no loss of consciousness, not even temporarily. Paralysis of the fifth cranial pair, which had occurred simultaneously with the hemiplegia, persisted, and in July of the same year the patient was seized, on two different occasions, with paralysis of the tongue, of a few seconds' duration only. From that time, however, his gait became uncertain, and the locomotor ataxy soon made frightfully rapid progress.

These instances of transitory paralysis are rare. The paralysis very often lasts some time, as when it affects the sixth cranial nerve, producing on a sudden internal strabismus, or when it attacks the third or motor oculi nerve, causing external strabismus, diplopia, and ptosis. The duration of these forms of paralysis is very variable; they may last for the remainder of the patient's life, or they may go off after a few months, or even after a few days only. In some cases they recur, when the disease is fully developed, after having disappeared for several years.

This is the form of paralysis which, as it gets well spontaneously, has made the fortune of so many methods of treatment, whilst the very success of the treatment contributes to leave the medical man in error. Paralysis of the third and sixth pair has been looked upon by many pathologists as dependent on constitutional syphilis, and when a treatment by mercury and iodine has been followed by apparent success, the diagnosis seems to be confirmed, and the other phenomena which characterize ataxy are ascribed to the same cause. Within a short time, however, the same remedies prove utterly powerless. Vision itself may be deeply affected. Amblyopia, for instance, may be present for some time; or the patient may have amaurosis on one side, and discover it by chance; or the amaurosis may be double, as in the case of the man in bed 23, St. Agnes Ward.

On carefully examining ataxic patients in the intervals when they are free from pain, there is often noticed an injection of the conjunctiva, sometimes as marked as in the most violent conjunctivitis, and in some cases giving rise to a sort of chemosis. There is at the same time contraction of the pupil, reducing it to the smallest possible size, and so powerful sometimes that it resists the influence of belladonna. On the other hand, during the paroxysm of pain, especially when the pain affects the head, the contraction of the pupil is replaced by more or less marked dilatation, and generally also the vascular injection of the conjunctiva disappears at such times. I merely mention these facts now, but further on I will revert to them, and try to interpret them.

Other cranial nerves may be affected as well, although this is the exception, and not the rule. These affections may coincide or alternate with those I have previously mentioned. Thus, the auditory nerve has been found paralyzed either on one or both sides, and I shall give you an instance of this presently. Dr. Duchenne has twice met with paralysis of the fifth pair concurrently with that of the third. "In one of those cases the two nerves were affected on the same side; in the other the fifth was paralyzed on both sides, and the third on the left only: in this case there was also paralysis of the soft palate and larynx."

Some of these premonitory nerve affections may be absent, but it very rarely occurs that they are all absent in the same case. I have nearly always found them, and Dr. Duchenne is right in attaching great importance to them for diagnosing the disease at the outset. Remember, besides, that they may have been transitory, and been forgotten by the patient, so that the physician must needs make careful inquiries in order to discover their existence in the patient's previous history.

The accession of the disease is again marked by strange sensations, by a sense of constriction of different parts of the body. The patient feels as if his chest, his arms, or legs, were compressed by an india-rubber cuirass. His shoes feel too tight, and he often has the sensation of a belt constricting his abdomen. And just as in the most confirmed cases of paraplegia, there is paresis of the rectum and bladder, or even paralysis of their sphincters.

The etiology of the disease is still very obscure, and Dr. Duchenne and I have not been able to discover constant causes in the cases which have come under our observation. The cases on record are, however, sufficiently numerous now to admit of my making a few remarks on the influence which age, sex, and hereditary predisposition seem to have on the production of the disease.

Locomotor ataxy is chiefly met with about the middle period of life, from 20 to 40, although it may occur late in life, as in the case of the gentleman 80 years old, which I related to you. It is a remarkable fact that males are more prone to it, and that in a very large proportion. Dr. Duchenne has only seen it four times, and I three times, in females. The general paralysis of the insane is another affection which greatly preponderates in the male sex.

Now, what influence has hereditary predisposition on locomotor ataxy? If this question can be answered, with great difficulty only, in the case of progressive muscular atrophy, the difficulty is greater still in the case of locomotor ataxy, which has been but recently studied. If you find, however, in the patient's family history, that there have been cases of various nervous diseases, you will be in a certain degree authorized in connecting ataxy with those diseases, and ascribing to them a common origin.

When treating of epilepsy, as you may remember, I related to you the history of a family, the different members of which were afflicted with monomania, hypochondriasis, epilepsy, seminal losses, and locomotor ataxy —thus illustrating what I told you of the transformation of neuroses into one another.

On July 17th, 1861, a physician of Rouen brought me a patient, aged 45, who was suffering from locomotor ataxy, in a very advanced stage. His intellect was perfect, but an uncle and an aunt of his were insane, one of his brothers was ataxic and another and younger brother was hemiplegic.

Dr. Duchenne and I know a gentleman who has been ataxic for more than twenty years. He has never manifested any intellectual disorder himself. But his father committed suicide, and his two sons have labored under the most peculiar nervous affections. One of them, although of perfectly sound mind, is irresistibly impelled to shriek in a most extraordinary manner nearly all day; the other has had, and still has, singular muscular spasms. These are examples again of the transformation of nervous affections through hereditary influences.

I now pass on to the study of locomotor ataxy, when the disease is fully developed.

When children walk along a narrow plank or the edge of a boat, you must have noticed the peculiarity of their gait. In order to maintain their equilibrium, they take one step forward, stop, sometimes go backwards again, and incline their body to one side or the other, instinctively putting their arms out like a sort of balancing-pole. In fact, their movements resemble those of an unskilled rope-dancer.

The gait of an ataxic patient is something like this. At the outset of the complaint, he staggers a little, especially as he gets up after having sat down for a long time. He rests on a stick or on the chair which he has just left, and he starts. As he takes the first step, the arm which does not rest on the stick leaves his side and oscillates like that of a rope-dancer, and his body inclines a little forwards. His walk is at first slow and uncertain, but becomes involuntarily hurried. Whereas in true paralysis, the leg is slowly lifted off the ground and is dragged along; in ataxy, the foot is thrust forward in variable directions, and comes down suddenly. Instead of the measured flexion of the knee-joint, which obtains normally, the flexion is sudden and followed by forcible extension.

When the disease is in a more advanced stage, if the patient does not rest on a stick, he throws his legs about with still greater disorder, and the inequality of his steps renders the loss of equilibrium still more imminent. Both his arms are then moved about like those of a rope-dancer, and his trunk itself is inclined or straightened according to the displacement of his centre of gravity.

This uncertainty and difficulty of progression do not prevent the patient from walking several miles on even ground, and he will often tire out persons free from any nervous affection. We had an instance of this in the case of a stonemason, who was admitted under me, September 18th, 1861. He had great difficulty in walking a few paces over the waxed floor of the ward, and yet on the previous day he had walked (almost without fatigue) from one end of Paris to the other.

When the disease, however, has made pretty considerable progress, the violence and irregularity of his movements soon exhaust the patient's strength, and he can scarcely walk a hundred paces before he gets out of breath, and is thrown into profuse perspiration.

There even comes a time when, although he still possesses muscular power, he cannot move a single step without falling down. If he be then supported by two persons under the arms, whilst he tries to walk, his legs move like those of a puppet, and are thrust to the right and to the left, forwards and backwards, with inconceivable disorder. From this time forwards he is obliged to keep in bed. The muscles of his trunk become affected also, and he can no longer sit up in a chair, unless he holds on to it with his hands, when his arms are not themselves implicated.

You can easily understand, gentlemen, how grave the prognosis must be in such cases. Death inevitably supervenes, and all the more quickly that sloughs form on the nates and about the trochanters, and that the suppuration to which they give rise rapidly exhausts the patient.

Instances, however, occur of patients who even at this advanced stage of the disease regain, sooner or later, some degree of motor power, and you had occasion to see this in the case of a man at No. 11 in St. Agnes Ward. After having been for a long time compelled to keep to his bed, he improved so much that he was first able to get down his bed by himself, next to walk a few steps, resting on a companion's arm or taking hold of a chair or going from bed to bed, and later he could come up or go down stairs. This amelioration lasted several months, and I was indulging the hope that he would get well, when he was seized with hæmoptysis accompanied by all the signs of phthisis, which ultimately carried him off.

In the same ward, you can at present see another patient afflicted with locomotor ataxy and amaurosis, who after having been on several occasions compelled to keep perfectly quiet, can now walk by resting on a chair, and guiding himself with a cane.

When the disease has reached one of the stages which I have just described, the diagnosis is in general easy, even if the affection has been studied in books only. At the outset, however, great care is required, and few physicians, unless familiar with the neurosis, are able to recognize it.

In the early part of August, 1861, I was consulted by a chemist residing in a western province, who complained of some weakness of the lower extremities and the bladder. The lightning-like pains of which he also complained, and the deafness of one ear which I detected (in his case replacing diplopia or amblyopia) led me at once to suspect locomotor ataxy, and a more careful investigation only confirmed my suspicions. For this I used a test which is of the hightest importance, and to which I am desirous of calling your attention most particularly.

You have noticed already that, at an advanced stage of the disease, when the patient is in the dark, or when he voluntarily shuts his eyes, the uncertainty of his gait increases so much that he is absolutely incapable of moving a single step without falling down. This phenomenon, which is a symptom of very great value, manifests itself, although in a less marked degree, yet strikingly enough, from the very outset of the complaint.

The last patient, whose case I was relating, although complaining of weakness in the legs, which weakness did not really exist, yet walked without tripping, and maintained his equilibrium perfectly. As soon as he closed his eyes, however, he immediately staggered like a drunken individual, and would have dropped down if I had prolonged the experiment for some time.

The irregularity of the patient's walk, when his eyes are closed, is of later occurrence, and therefore of less diagnostic value than the next one which I am now going to mention.

If you ask an ataxic individual to stand up, and keep his feet closely applied together along their inner edges, he manages to do it with some difficulty when his eyes are open, even at an early stage of the disease. But when he shuts his eyes, he immediately oscillates and falls down, unless he be supported, or unless he opens his eyes and takes hold of a point of support, or, again, unless he makes considerable exertions to recover his equilibrium.

Thus, the walk of the patient whom I first mentioned presented little uncertainty only; but when his feet were closely approximated, he found it perfectly impossible to maintain his equilibrium on shutting his eyes. This sign, then, is of great value; and all the more so, that in paralysis nothing of the kind is observed. I have often had in my wards patients afflicted with hemiplegia, sequential to cerebral hemorrhage, and sometimes also individuals attacked with general paralysis. I have made them walk and stand in your presence, with their eyes alternately closed and open, and you have been able to satisfy yourselves that they did not lose their equilibrium when their eyes were shut.

Every patient, however, who suffers from locomotor ataxy does not walk in the manner which I have described above. Thus, the patient at No. 23, in St. Agnes Ward, who has double amaurosis as well, walks very much like a blind man. He carries a stick in his left hand, and in his right a small cane, with which he guides himself, whilst he walks *in a hurried manner*. A blind man walks, in general, in slow and measured steps, regularly balancing himself from right to left, but this patient constantly hurries forwards, and trots more than he walks, with a jerked step, oscillating when he stops. A blind man can remain perfectly motionless when he stands; an ataxic patient, on the contrary, loses his equilibrium, because his muscles are always in a state of exaggerated spasmodic contraction. In some exceptional cases, the patient's limbs are stiff when he walks, and his body moves all of a piece, as it were.

I was lately consulted by a patient whose intelligence was perfect, and who had mydriasis, but no strabismus. He had, besides, paralysis of the sexual organs, dating one month back, cutaneous anæsthesia, and what he termed paralysis of the lower extremities. He was constantly tripping, and dared not go out alone; when he walked he was obliged to take short steps only, otherwise his movements became disordered. He was not really paralyzed, because his muscular power was still considerable, and he was only in the first stage of the disease.

At an advanced period of locomotor ataxy, spasmodic contractions are frequently observed, not only when the patient wills a regular movement, but even in the state of rest. In the latter case they consist in very powerful jerks of the limbs, and are an important symptom of this singular neurosis.

Patients then state that whilst they are walking, or even whilst they are merely standing, they feel as if the ground suddenly gave way beneath their feet. The cause of this is that the flexors of the limbs have been suddenly seized with spasm, and, overcoming the resistance of the extensors, have produced the sudden flexion of one of the lower limbs.

You may remember a woman lying at No. 23, in St. Bernard Ward. When her legs were exposed whilst she was lying down, we could often see them shake and quiver with extraordinary violence. If, with both my hands, I encircled her thigh, I could feel the quivering of her muscles, whilst her foot moved with extraordinary violence and rapidity, without her knowledge and against her will.

Dr. Duchenne and I saw a patient at Montmartre suffering from well-marked locomotor ataxy, and who presented equally violent spasmodic movements.

In July, 1861, an old patient of mine, who had been suffering from this disease for more than twenty years, fractured both bones of one of his legs. In spite of the apparatus applied, the injured limb was constantly shaken convulsively, and the treatment considerably interfered with.

The pains, which I described in the first period of the disease, are usually, but not always, more intense when the disease is fully developed. They torture the patient, and extend to the trunk and upper limbs. Bodily fatigue and the least moral emotions suffice to bring them on again.

They most frequently recur in paroxysms, that is to say, they show themselves for a few hours or a few days, every week or every month, and then disappear. In other cases they are continuous, recurring from ten to thirty times in an hour, and deprive the patient of sleep for months and even years. We had an instance of this in the case of a picture-dealer who was in St. Agnes Ward, and who was subject to such intense and frequently recurring pains, that his face always wore an expression of suffering. Belladonna and opium only gave him very transient relief.

Whether frequent or rare, these pains usually set in and go off suddenly. Sometimes, however, the patient is warned of their coming by some morbid sensation in the stomach or the genital organs. Thus a lady, who often consults me, is seized at intervals of two or three months with shooting pain in the lower limbs or the walls of the chest, sometimes preceded by epigastric *malaise*, sometimes by a dragging sensation about the region of the womb. It is a sort of *aura* which starts from those regions, and which ascends and descends to the spots which are suddenly seized with acute and transient pain. At other times, however, these pains set in of a sudden, unpreceded by any sensation, so that this form of *aura* may be after all due to a special susceptibility of the stomach and womb; and I am the more inclined to think so because the lady is liable to frequent attacks of gastralgia, and has suffered, for several years, from dropsy of the left ovary.

The premonitory anæsthesia becomes general after a time. The patient feels the ground imperfectly, and when to cutaneous anæsthesia is superadded the loss of muscular and articular sensibility, the patient can no longer feel the resistance of the ground, and if he shuts his eyes, he may, as Dr. Duchenne and I found out last year, have the sensation of being suspended in the air. Sometimes the patient fancies the ground is elastic, and that he is walking on india-rubber or on compressible balls, and this strange sensation persists even when sight can help him to correct his mistake.

Mucous membranes may also become anæsthetic. The patient at No. 2, in St. Agnes Ward, suffers from anæsthesia of the mucous membrane of the mouth. He does not feel bodies placed in contact with his lips, sometimes drops the food which is between them, and cannot distinguish the temperature of what he eats or drinks. His teeth have lost their special sensibility, and cannot distinguish substances which are easily broken down from those that are not. The mucous membrane of his tongue obscurely perceives sapid substances, especially on the left side. The upper extremities are likewise affected sometimes, and the patient loses his sense of touch, occasionally also all muscular, osseous, and articular sensibility; but retains normally the sensibility to differences of temperature.

In some cases, and this is an important fact to remember, locomotor ataxy may be unattended with impairment of sensibility. I have myself seen a few cases of this kind, and several have been recorded by other physicians; as the one published by Dr. Oulmont, and the case you may have seen at the Lariboisière Hospital, in Dr. Hérard's ward. Dr. Lecoq has also published two analogous cases, in the "Archives Générales de Médecine," 1861, in which no special pains had ever been felt, and sensibility had remained normal.

However exceptional these cases may be, I admit they are still highly important, as proving categorically that cutaneous and muscular anæsthesia are only secondary phenomena of the disease.

When the disease is fully developed, the various affections of the eye, which I mentioned as occurring at the outset, show themselves again, and may remain persistently. Thus diplopia, amblyopia, and amaurosis, as well as paralysis of the third or sixth nerve, may again be noted. Both eyes may be affected, although this rarely happens; whilst in other cases, as I have noticed several times, these various affections may be totally absent.

I have also met with paralysis of the fifth nerve, as shown by anæsthesia of the mucous membranes of the eye, nose, and mouth, and the skin of the face.

Anaphrodisia is often present, together with paralysis of the sphincters of the rectum and bladder. Yet, in three cases which I saw with Dr. Duchenne, the sexual power was unimpaired, although the bladder and rectum were seriously affected. In some cases the fundus of the bladder is paralyzed, but not the sphincter, producing retention of urine; or the rectum is paralyzed, whilst the sphincters of the anus act normally, so that obstinate constipation is the result. The retention of urine may be followed by grave consequences, as in cases of paraplegia; cystitis, for instance, may set in, and if the inflammation spreads upwards to the kidneys, death may result, preceded by symptoms of urinæmia or of pyæmia.

Locomotor ataxy, like many other diseases, is pretty frequently incompletely developed. Thus, at the outset, and sometimes for a period of several years, it reveals its existence by a few symptoms only, the significance of which may escape the observer. In one case, for instance, there will be merely paralysis of the muscles supplied by the third and sixth cranial nerves; in another, there will be more or less complete amaurosis, which, after having resisted every treatment, gets well spontaneously; or again, acute pains in the lower extremities will alone be complained of, which the patient compares to electric shocks, and which are so characteristic that, whenever they are mentioned to me, I immediately suspect incipient locomotor ataxy. Indeed, since my attention has been drawn to this point, I have seen so many patients whom I at one time regarded as suffering from vague neuralgic pains, or from muscular rheumatism, present, from a few months to two or three years afterwards, the most characteristic symptoms of locomotor ataxy, that I now keep on my guard. I have often been able to find out by closely questioning patients affected with such shooting pains, that they also presented some of the premonitory symptoms of ataxy; such, for example, as impotence and spermatorrhœa.

The *progress* of this disease is usually slow, and it may extend over a period of ten, and even twenty years. A friend of mine has been ataxic for twenty years; and I am at present attending a Polish officer who has been ataxic since 1846, and who yet took a very active part in the Hungarian war of 1848. He can now ride his horse every day; and although he does not feel his stirrups (so great is the insensibility of his feet), he yet manages to sit his horse well, through the great strength of the adductors of his thighs, and on one occasion, as I was testing his strength, he gave me very great pain by squeezing my hand between his knees. In some cases, however, the disease may run a rapid course, as in a patient from Saulieu, whom I had under my care, and in whom the disease became generalized in the space of six months. You yourselves saw an instance of this in the case of the stonemason who was in St. Agnes Ward.

The *prognosis* in this disease is relatively of extreme gravity, for if in some cases it may remain stationary for a long time, it does not, however, get well. When speaking of the treatment, I will discuss the question whether it can be arrested.

I shall now endeavor to analyze the principal phenomena which characterize the disease, and attempt to give an idea of the defect of co-ordination of movement which constitutes the most striking symptom of confirmed ataxy.

In the act of skating, there is required a very remarkable co-ordination of all the movements of the foot, leg, and trunk; for as the impulse forward is given, the skater must keep in equilibrium on a single skate, that is to say, on a very thin blade of iron placed vertically. He must put one foot down on the ice at the very instant that he lifts the other up. And when both his feet are together, learned combinations of muscular actions are necessary to enable the skates to avoid or get over an obstacle; he must often bend his body forwards, backwards, or sideways, and he must be careful to use his arms as balancing-poles. Now the necessity for muscular co-ordination, which is strikingly marked in the act of skating, exists for all the movements of the body as well. Those which look the simplest require a precision, the difficulty of which we forget, owing to the habit we have formed of executing them automatically. Indeed, there is, in reality, no simple movement: when we flex our fingers, for instance, the flexors are not the only muscles which are called into play, but the extensors, which antagonize them, must also contract. For the performance of every movement there is required a common action of several muscles tending to the same end, and this common action, or muscular synergia, as it is termed, produces the harmony of movements. When this is at fault, defect of co-ordination results, and this constitutes one of the chief characteristics of locomotor ataxy and of St. Vitus's dance.

In the majority of patients who are afflicted with ataxy, tactile sensibility diminishes, and is even abolished, especially in the sole of the foot and the skin of the leg. This anæsthesia extends sometimes to the trunk, although it gradually diminishes from below upwards. One kind of sensibility persists to the last, namely, that which takes cognizance of differences of temperature.

The anæsthesia may extend deeper than the skin, and affect the muscles and articular surfaces. The irregularity of the movements may be considerable, even when there is no loss of sensibility. At the end of August, 1861, I was consulted by an eminent barrister from Dublin, who had formerly been a patient of the illustrious Graves, and had lately been under the care of Drs. Corrigan and Carmichael. In his case sensibility was perfect, and yet the defect of co-ordination was so great that he was not able to walk, unless supported by the arm of a companion. When both the cutaneous and deep sensibility, however, is lost, the incoördination of movements reaches its maximum.

I now proceed to discuss a very important physiological point, to which several eminent physicians have ascribed the principal share in the production of locomotor ataxy.

Sir Charles Bell observed the following case:

"A mother, while nursing her infant, was seized with paralysis, attended with the loss of muscular power on one side of her body and the loss of sensibility of the other. The surprising, and indeed the alarming circumstance here was, that she could hold her child to her bosom with the arm which retained muscular power only so long as she looked to the infant. If surrounding objects withdrew her attention from the state of her arm, the flexor muscles gradually relaxed, and the child was in danger of falling."

Sir Charles, therefore, believed that muscles are supplied by nerves endowed with two distinct properties—the one giving muscular power, and the other muscular sensibility; and according to him, "muscular power is insufficient for the exercise of the limbs, without a sensibility to accompany and direct it."

Sight, however, can supply the absence of this sensibility, as shown in the above case. To the consciousness of exertion, Sir Charles Bell gave the name of *muscular sense;* but Gerdy, who perhaps was not aware of Bell's essay on the subject, suggested for the same faculty the name of *sense of muscular activity;* and in 1855, Dr. Landry published a memoir on "The Paralysis of the Sense of Muscular Activity." For my part, I confess that I am by no means convinced of the existence of this sense, and I do not see that it is proved by the case recorded by the illustrious English physiologist.

An important distinction must be drawn between the consciousness of a movement which has been executed and the consciousness of the muscular contraction which performs the movement. When, after shutting our eyes, we execute, without effort, a pretty extensive movement, we are unable, even on paying the strictest attention, to feel the contraction of our muscles, although we may feel the movement communicated to the levers by the contracted muscles. This fact is so true, that when we ask an intelligent person, who knows nothing of anatomy and physiology, which is the seat of the movements through which the fingers are flexed or extended? he immediately points to the hand, and never to the forearm. It is only when the muscular effort is considerable, or kept up for a long time, that it is perceived where the contraction really occurs. Normally, then, we have no consciousness of muscular activity, but merely the consciousness of the movement itself, which is a perfectly different thing.

Another proof of this is the following: If we make the hand, the fingers, or limbs of a healthy individual go through a series of passive movements, the extent and variety of these movements are perfectly appreciated by the person. But although his muscles are completely inactive, he is not conscious of this, but feels the movement which is performed, although he does not know by what means it is executed.

Every one may repeat these experiments, and will be then convinced that this so-called muscular sense of Bell, or sense of muscular activity of Gerdy, has no real existence in ordinary and normal contraction. Rest your elbow on a table, for instance, and flex or extend passively one of your arms, or the fingers of one hand. You will be perfectly conscious of these movements, even with your eyes closed; but the sensation which makes you affirm their existence, is partly psychical and partly dependent on a local impression.

Allow me, gentlemen, to explain my meaning. When I will a movement, I am conscious of its being performed, first, because every-day experience has taught me that our limbs invariably obey our will; and this is the psychical act which I mentioned just now. But when I carefully analyze the impressions which are excited during these various movements, I perceive a very evident sensation, which is not seated, however, in the muscles of the arm. When my elbow is placed on a table, as in the above experiment, there is a sensation of pressure on the olecranon, which sensation is only felt in the skin. As the forearm is extended, a portion of the skin which covers its posterior and ulnar aspect will be in contact with the table; but when it is flexed, this latter contact ceases, while other points of the lower and posterior portion of the arm come in contact with the table, so that this double impression, entirely confined to the skin, tells me that I have completed a movement of flexion or extension.

The same thing happens when the hand is moved. If we shut our eyes whilst we move one hand, for instance, we feel in the palm, and in the palmar surface of the fingers, a sensation of dragging when the hand is opened wide, and a sensation of relaxation when the hand is closed, in addition to a special sensation in the joints themselves, which latter is always striking, and even painful, when we wake up from sleep. As to muscular sensations, they only exist when the contraction is extreme, or when the muscle is in a painful condition, as after a contusion or great fatigue, for example.

Understand me well, gentlemen; I do not deny muscular sensibility, as I have been said to do, but what is a very different thing—I deny the existence of a sense of muscular activity. Muscles are, indeed, endowed with an obtuse sensibility, as surgeons in their operations have found out thousands of times. This sensibility, which scarcely feels the incisions of a sharp knife, is very acute in cramp, in what is called muscular rheumatism, in inflammation of the muscular tissue, and after extreme fatigue; but from an abnormal state of sensibility we must not infer the existence of a physiological sensibility. Ligaments and articular surfaces become very painful in cases of arthritis or of sprains. The neck of the womb, in metritis, is sometimes exquisitely sensitive, and yet you are aware of the slight degree of sensibility to pain in a condition of health.

A healthy muscle, when galvanized, feels pain. If the biceps be pinched sharply, pain is felt, which, although not very acute, is perfectly distinct from the pain in the skin, and which must, therefore, be seated in the muscle. Muscles, then, are endowed with sensibility, but, I repeat, this sensibility is totally different from the sense of muscular activity, the existence of which I deny, as a non-psychical phenomenon. If, in the last experiment which I mentioned, the movements of the hand and arm be passive, instead of being spontaneous and active, the person experimented on will know perfectly, even with his eyes shut, that the movements have been executed. But he will be informed of this, not by his sense of muscular activity, for his muscles shall have been inactive, but by the nature of the pressure made by another person's hand on his own, and by the sensations which he will feel in his skin and the vicinity of his joints. Cutaneous and deep sensibility, therefore, plays in this case a very important part, and this it is which regulates the movement.

It is this sensibility, that is to say, the impression made first on the skin, then on the deeper structures, and next on the articular surfaces, which enables us to appreciate the form, weight, and resistance of objects. If you lay the back of your hand flat on a table, and then place an object in your palm, you will at once appreciate, without making any muscular effort, part of its shape and weight; and this notion will at once tell you how much muscular power you shall need to take hold of and displace the object. The impression received will be responded to by your will, and your muscles commanded to act in proportion. But suppose, now, that the superficial and deep structures of your hand have become insensible. If you then successively place in your hand two objects of the same shape and color, but of different weight and consistency, on trying to raise them in turn, you will either go beyond or stop short of the mark, and your movements will be marked by morbid irregularity. Yet your muscles will have retained their power, and what Bell, Gerdy, and Landry call muscular sense, or sense of muscular activity, will not be in the least impaired. Cutaneous and deep sensibility alone is affected, which is the monitor of the mind, and consequently of the will.

When a person, whose cutaneous and deep sensibility is lost, attempts to execute movements, he is like a deaf individual who tries to speak. The pitch of a man's voice is raised proportionately to the distance at which he wishes to be heard, but persons who are deaf, having no means of judging of the pitch of their voice, either raise it most inopportunely, or lower it so as to be inaudible. If I do not admit, then, the existence of this so-called muscular sense of Bell, or sense of muscular activity of Gerdy, I need scarcely add that I cannot admit Dr. Landry's theory, that locomotor ataxy consists in the loss of this sense of muscular activity. If you study this neurosis in its gravest form, and in its most advanced stage, I confess that you will find muscular sensibility and the consciousness of resistance and pressure gone. But in no instance is muscular sensibility deeply impaired, without the sensibility of the skin and articular surfaces being equally so; and I do not see why we should resort to a function and to properties, the existence of which is by no means proved, when we can interpret the facts in the simple manner which I explained to you just now.

But remember, gentlemen, that you may, on the one hand, meet with patients suffering from ataxy, in a very advanced stage even, who have still retained their muscular sensibility, as in the case of the barrister from Dublin whom I mentioned to you, of three of my patients in St. Agnes Ward, and of another patient who was under the care of my regretted colleague, Dr. Legroux. On the other hand, there may be muscular anæsthesia, and yet no locomotor-ataxy, as in the case of a house-painter who was admitted into the St. Agnes Ward in May, 1861. There was complete anæsthesia of the skin over his whole body; he did not feel pricking or pinching, but could distinguish differences of temperature, and when a vase filled with cold water was placed on his thigh, he complained of very disagreeable sensations. He did not feel violent pressure of his muscles; and when he contracted his muscles powerfully, he only knew of their acting because he had willed it, but he had no feeling of their doing so. The sensibility of his hands and feet was nearly perfect. Yet, in spite of this complete muscular insensibility, he walked naturally, even when his eyes were shut.

This case, which I studied with the greatest care, proves, therefore, that muscular insensibility, which necessarily implies the loss of the sense of muscular activity, does not suffice for producing locomotor ataxy, and there must, in my opinion, be superadded another element, to which I shall revert by and by, namely, *spasm.*

I may add, gentlemen, that in some cases, rather uncommon, it is true, even at a pretty advanced period of the disease, the patient complains of an extraordinary degree of cutaneous and muscular hyperæsthesia. In July, 1861, a practitioner of Rouen sent me a patient suffering from locomotor ataxy, in a very advanced stage. Both the patient and his medical adviser had been particularly struck with the exaggerated sensibility of the skin and the deeper structures of the limbs, so heightened, indeed, that the least contact or pressure gave unbearable pain. At the time when I examined the patient these curious phenomena had passed away, but they had lasted several months, during which the irregularity of the patient's movements, and the difficulty he had in maintaining his equilibrium, were already considerable.

The following case, which I studied carefully, would seem, at first sight, to favor Dr. Landry's theory; but a more exact analysis of the case admits of its being interpreted in a manner contrary to those views.

On January the 26th, 1863, I was asked by Dr. Collongues to see, with him, a lady about 40 years old, who had been seized with left hemiplegia, about the middle of the year 1862. There had been at first complete loss of sensation and power of movement, without notable impairment of the intellect. By degrees the power of moving returned, and when I saw the lady she was sewing with her right hand, and held her work in her left hand, the one which had been previously palsied. Sensibility was *completely abolished* in the arm and hand, and the whole lower limb, except in the sole of the foot, which still retained a certain amount of *very obtuse* sensibility. Hard pinching of the skin, violent compression of the muscular masses, gave absolutely no sensation at all, nor did pressure of the articular surfaces against one another give rise to any.

Her hand could be opened out or closed, her forearm bent or extended, without her knowledge. Differences of temperature she perceived, however, perfectly. When I asked her to open out her closed hand, which I held in mine, she performed the movement; but although she *knew* that she performed it, she yet *did not feel it.* Even when she shut her eyes, she could do as directed. If, when her eyes were closed, I opened out her hand, and then asked her to extend her fingers, she moved them as if trying to do as she was bid, and if I told her that her hand was closed, although it was wide open at the time, she exerted herself to stretch out her fingers in an exaggerated and disorderly manner.

Without looking at them, she could bend each of her fingers in turn, into her palm, although her movements in so doing lacked precision.

In order not to drop any object which she might be holding, she was obliged to keep her eyes on her hand, and she squeezed the object with unnecessary force. If she closed her hand forcibly, when empty, she did it with such violence that her nails wounded her palm.

When she was engaged in conversation, and her attention could not thus be kept fixed on any object she might be holding, she had recourse to a peculiar stratagem, in order not to drop it. She pressed it against her chest, which had still retained its sensibility, and she could thus rectify, and in some degree measure, the contractions of the muscles of the forearm which moved the hand.

By a *constant exertion* of her will, she could supply the absence of sensibility of the skin, muscles, and articular surfaces. Normally, however, no *manifest* exertion of the will is needed to enable a person to hold an object in his hands, even when his attention is diverted from it, as shown by the familiar circumstance of a man carrying a stick or an umbrella without his attention being always directed to it.

The muscular power of the patient was *nearly normal.* The left forearm, when bent on the arm, resisted extension nearly as much as the right; and the patient opposed as great a resistance also to the forcible abduction of her left arm from the trunk as when the same experiment was tried on the right side.

The movements by which she alternately closed or opened her hand, were marked by the same irregularity as in the most confirmed cases of ataxy. Thus, instead of closing her hand by at once bending her fingers and thumb, she bent each of them in turn, in a strange and disorderly manner. This became considerably more marked when her eyes were shut, and was less so when she kept looking at her hands whilst moving them. In the alternate movements of flexion and extension of the hand, it was easy to perceive that the antagonistic muscles no longer regulated those movements, which were therefore more extensive than they should have been.

At night, the patient did not know where her left arm was, unless it was in contact with some sensitive portion of her body; it might hang out of bed without her being aware of it.

When she walked, she threw her left leg and foot forward exactly as an ataxic individual. She was obliged to keep her eyes on that leg, or else she would have fallen down. She knew, however, that her left foot rested on the ground, from a sensation of resistance in the hip-joint, and a very faint sensation in the sole of that foot. But this twofold impression was not sufficiently distinct to allow her to walk in the dark. At night she did not know the position of her left leg unless it was in contact with the right.

This case is interesting in many regards. First of all, the age of the patient and her previous history exclude all possibility of an hysterical affection, and therefore all idea of deceit, which, nervous women unfortunately practice in too many cases. Secondly, it is very evident that the case never had the aspect of locomotor ataxy. The disease had set in with sudden hemiplegia, probably the result of an extravasation of blood into the brain. There had never been the characteristic pains of ataxy, and no impairment of the sight. The anæsthesia of the skin, the muscles, and joints, had occurred suddenly; and this fact excluded all idea of ataxy. Now the defect of co-ordinating power was as great as it could possibly be, and no one would have hesitated to call the patient ataxic, had she presented the symptoms which usually precede and accompany locomotor ataxy. Cases such as this, if they were frequent, would favor Dr. Landry's theory; but as I have already said there are a good many instances of locomotor ataxy, and in which cutaneous, muscular, and articular sensibility is thoroughly preserved, and I have drawn your attention to some of them in my own wards.

On the other hand, recall to mind the case of that young man, suffering from diphtheritic paralysis, who was in the St. Agnes Ward, in January, 1863, and that of a young woman, similarly afflicted, who was in the St. Bernard Ward, in the beginning of the year 1862. In both those patients the sensibility of the feet and skin of the legs was singularly diminished, in fact, nearly abolished. Pressure of the muscles was not perceived, movements of the knee and ankle were not felt, and yet the gait of those patients was not in the least like that of ataxic individuals, but merely of persons struck with paralysis. They dragged their feet with difficulty along the floor, and the uncertainty of their walk was no greater than that which results from ordinary muscular paralysis. When they were asked to shut their eyes, they could still walk, although with increased difficulty.

You see then, gentlemen, that if one case seems to tell in favor of Dr. Landry's theory, other, and more numerous cases, decide against it. In the case of the lady mentioned above, hemorrhage into the cerebellum, or softening of that organ, might be suspected from the suddenness of the seizure, and the form assumed by the first symptoms which manifested themselves. If so, the case would come under the category of *cerebellar ataxy*, to which attention has been drawn by Drs. Bouillaud, Hillariet, and Duchenne.

The circumstance that passive movements of his limbs are not perceived by the patient, has by some been regarded as a characteristic symptom of locomotor ataxy; but this is a grave error, which is disproved by clinical observation. I admit that in hemiplegia due to cerebral hemorrhage or softening, passive movements of the palsied limbs are perfectly perceived, and that the perception is often attended with pain, provided, however, that the patient be not comatose. I will add, that compression of the muscles, in such cases, often gives rise to pain and to a sensation of cramps on the paralyzed, and not on the healthy side. But this does not happen in most cases of paraplegia (and it is especially with paraplegia that locomotor ataxy may be confounded). In July, 1861, I saw a young lady from Bernay, who had complete paralysis of the lower limbs. Pressure and pinching of the skin were feebly felt. When her eyes were shut, she felt imperfectly if her leg was touched, but she was not aware of my pressing the muscles of her calf, although they were then thrown into violent and convulsive contractions. If, after extending her leg, I held her foot in my hand, and then forcibly adducted, abducted, flexed, or extended the limb, or pressed strongly the articular surfaces against one another, she felt no sensation whatever. At night she did not know the position of her limbs. These were really the symptoms ascribed by Dr. Landry to loss of the sense of muscular activity, and yet the case was one of paralysis, and not of ataxy. Dr. Landry has been led into error by the fact, that in the last stage of locomotor ataxy, anæsthesia supervenes; but it is a symptom which belongs to most cases of paraplegia.

In locomotor ataxy which has not yet reached its final stage, even when the irregularity of the movements clearly points to the disease, muscular and articular sensibility may be preserved, as in the cases of the superior officer and the Dublin barrister, which I have already mentioned. The uncertainty of the gait may be considerable, especially when the patient shuts his eyes, although he may feel the ground perfectly, and although sensibility may not seem in the least impaired.

The fact that the patient's gait becomes much more uncertain when he shuts his eyes, is not a symptom either of the loss of the sense of muscular activity. The same thing occurs in perfect health, and happens as much, after all, in a clear case of paraplegia as in one of locomotor ataxy. Any one of us here will walk badly on shutting his eyes; and even when sure of meeting with no obstacle, he will walk in a peculiarly hesitating manner. The well-known bet of the Versailles lawn has clearly proved that no one can walk, with a handkerchief before his eyes, from one end of the lawn to the other, without getting into the gravel-walks. This is a proof that walking must of necessity be guided by sight, and that what has been called the sense of muscular activity is not sufficient for this purpose.

A blind man who walks on a pavement, however much he may be used to do without his sight, is yet obliged, in order not to deviate from his course, to use a stick, which brings him back into the normal direction which he is constantly losing.

Now, in order to explain the simultaneous existence in ataxic patients of extreme incoördination of movements and very slight diminution, or even perfect preservation, of cutaneous and muscular sensibility, the existence of a *deep* or *common sensibility* (*cœnesthesia*) has been assumed, the impairment of which is said to account for the phenomena. The author* of a recent critical review on the subject defines this new kind of sensibility thus: "It is the very distinct perception (although almost unnoticed from its being so continuous) which we all have of the presence of our organs, their volume, weight, shape, situation, and relations. From all the points of the organism there constantly ascend to the nervous centres an infinity of sensitive currents which, luckily for us, are not noticed by the encephalon, but the *interruptions* or *irregularities* of which strike us forcibly as soon as they occur."

I confess, gentlemen, that I cannot conceive a common, unnoticed sensibility, which reveals itself to us only when it is *interrupted*, in other words, when it ceases to exist. The very men who admit its existence, confess that physiology cannot teach us what it is, and that pathology alone can do so. Yet the few examples which have been given in illustration have not convinced me. They all consisted of cases of individuals "who could not tell positively the position of their limbs, unless they saw them, and who suffered merely from a very slight impairment of tactile sense, limited to a circumscribed region of the trunk." The same author cites the cases of two women, observed by himself, one of whom "was obliged to note the place where she lay her hands before going off to sleep, in order to be sure of finding them in the dark." This case would appear of little value to me, even if the observer had noted the condition of the *articular sensibility*, which he forgot to take into account.

Remember, however, that this so-called *deep* sensibility should not be confounded with the *organic* sensibility which nobody denies. This latter manifests itself, only when it is exalted in pathological conditions. The stomach, for instance, which in health performs its functions silently, makes its presence felt in bad digestion; and other organs, which are normally insensible, acquire, under the influence of what is called inflammation, an exaggerated sensibility, and become the seat of the most acute pain.

I now pass on to the *differential diagnosis* of locomotor ataxy. It most frequently attacks the lower limbs first: the patient believes that he is seized with paralysis, and his mistake is shared by some medical men, very excusably indeed when there is real paralysis of the bladder and rectum at the same time, with a sense of circular abdominal constriction, tingling of the lower extremities, &c. In order to avoid this mistake, however, the patient's muscular power need only be tested, and the absence of real paralysis will thus become evident.

Tumors of the cerebellum give rise to a form of ataxy. Dr. Hérard has lately published, in the Union Médicale (t. iii, 1860, p. 230), a very remarkable case of the kind. The patient had lost the power of associating, combining, and co-ordinating the movements which make up the complex act of locomotion, standing, &c. There was no paralysis of sensation or motion, general or partial, direct or crossed. So far, the symptoms of the case resembled those of locomotor ataxy, since the latter disease need not be accompanied by paralysis. A distinction, however, was afforded by the extreme frequency of vomiting, in Dr. Hérard's case, which symptom, according to the interesting researches of Dr. Hillairet, accompanies cerebellar diseases. Moreover, the patient had never presented the premonitory symptoms of genuine locomotor ataxy, namely, the characteristic pains in the limbs and the trunk, the disturbances of vision, and the various forms of local paralysis, &c.

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* Dr. Axenfeld: "Des Lésions atrophiques de la moelle épinière." Archives Générales de Médecine, août et octobre, 1863, p. 486.

I need not go into the differential diagnosis of ataxy and the general paralysis of the insane and chorea: these two last diseases are attended with such characteristic symptoms that a mistake seems to me impossible.

When speaking just now of cerebellar ataxy, I should have mentioned Professor Bouillaud as having been the first who experimentally and clinically studied the effects of injuries to the cerebellum. As far back as 1828, and later in 1847, in his "Nosographie Médicale," my learned colleague described *the various co-ordinated movements* which injuries of the cerebellum impaired; namely, walking, standing, and the maintenance of equilibrium. For a detailed account of his researches, I must refer you to some lectures delivered by Professor Bouillaud, and which have been recently published by Dr. Auguste Voisin.*

§ 2. *Pathological Anatomy of Progressive Locomotor Ataxy.—Relation of the Lesions to the Symptoms.—Nature of the Disease.—Treatment.*

In his treatise on "L'Electrisation localisée," in which he was the first to give the clearest and most complete description of the symptoms of progressive locomotor ataxy, Dr. Duchenne (de Boulogne) says not a word of its *pathological anatomy*. At least, he gives one case only in which he had occasion to seek, after death, for the alterations which might characterize the disease; and in that case, the subject of which was an individual who, in 1858, died in one of Dr. Nonat's wards at the Charité Hospital, "*the encephalon and spinal cord, on the most careful examination, presented no anatomical lesion appreciable to the naked eye.*"† These negative results did not support the theory which Dr. Duchenne had, *à priori*, broached of the nature of the disease. Indeed, reasoning from the fact that, since the beautiful researches of Flourens and Bouillaud, the cerebellum was looked upon as the seat of the faculty of co-ordinating movements, Dr. Duchenne suggested that, in locomotor ataxy, the defect of motor co-ordination, which constitutes its primary phenomenon, must be "*necessarily dependent on some structural or functional lesion of the cerebellum.*" Secondly, when he took into account the order of sequence, and the progress of the symptoms, he was led to believe that the central morbid process which gave rise to the symptoms, generally began in the motor nerves of the eye and the corpora quadrigemina, from which they extended to the superior peduncles of the cerebellum, and lastly to that organ itself.

Now that a pretty good number of cases have been recorded, showing that in progressive locomotor ataxy it is the spinal cord which is diseased, and nearly always a limited portion of the cord, the dorso-lumbar especially, and very rarely the cervical, whilst the cerebellum is not notably affected, Dr. Duchenne has given up his first theory. The anatomical appearances found in ataxy are confined, as a rule, to the posterior columns of the cord and the roots which issue from them; it is only in exceptional cases that the antero-lateral columns are implicated as well. These appearances consist sometimes in a kind of gray degeneration, and sometimes in a gelatiniform and translucent condition, in a diminution of consistency, or in a state of induration, called *sclerosis*. In the greater number of cases the posterior columns are sensibly diminished in size, but in some very rare instances the volume is increased. The alterations of the posterior roots are proportionate to those of the cord, that is to say, they are most marked in the roots which are connected with the most diseased portions of the cord.

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* Union Médicale, 18, 25, et 28 juin, 1859.

† "Traité de l'Electrisation localisée," p. 608.

With regard to the microscopical appearances, allow me to quote the following extract from a recent memoir of Dr. Axenfeld,* in which are summed up the observations made by a good many authors:

"In the *white matter* of the posterior columns, which has now become yellowish or gray, are seen scattered nerve-tubes, pale, shrunken, or varicose, sometimes reduced to their neurilemma only or filled with granular contents, a few still retaining their *cylinder axis*. On the other hand, the connective transparent substance (*neuroglia* of Virchow), the blastema in which these tubes are imbedded, has become fibrillated, and presents, amidst a large quantity of amorphous granules, a smaller quantity of elongated nuclei, and a smaller one still of cells (perhaps the nuclei, or at least most of them, belong to the nerve-sheaths). Corpora amylacea, also, are met with in variable quantity, distinguishable by their usual reaction with tincture of iodine. Lastly, the bloodvessels are considerably developed, and their thickened walls, composed of several layers, are incrusted with a deposit of fatty granules.

"In the posterior cornua of the *gray matter* the same alterations are found, but less markedly. The reddish tint of this part is due to the injection of its capillary network, and occasionally its tint is darker, blackish, owing to the presence of numerous granules of pigment. The nerve-tubes in these cornua are sometimes destroyed, and the nerve-cells altered in shape, although in general both the tubes and cells are normal.

"The changes noticed in the posterior roots are the same as those of the corresponding columns; and they are the same again in the diseased portions of the bulb, pons, optic nerves, &c.

"On the whole, all these alterations clearly point to *atrophy* of the nervous tissue."

The first two cases in which I had occasion to note these pathological appearances are the following. The first one was published by Dr. Bourdon, under whose care the patient was at the time of his death.†

Mr. W., a man of letters, aged 38, was admitted March 22, 1861, under Dr. Bourdon's care, into the Maison Municipale de Santé. He has formerly led a rather dissipated life, and has often had painful emotions and known real grief. When about 25 years old, he had well-marked epileptic fits, probably due to his excessive use of absinthe, for the fits disappeared after two years, and on his giving up drinking absinthe.

The symptoms of his present complaint first showed themselves about six years ago. At first slow and uncertain in its course, the disease afterwards kept on the increase, especially for the last six months, during which time he has had a good deal of trouble and undergone great fatigue. The first symptoms were uncertainty in the movements of the limbs, and mere stiffness when walking, followed by some difficulty in going up and particularly down a staircase, in spite of the most powerful exertion of the will.

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* Dr. Axenfeld: "Des Lésions atrophiques de la moelle épinière." Archives Générales de Médecine, août, 1863, p. 224.

† See "Etudes cliniques et histologiques sur l'Ataxie locomotrice progressive." Archives Générales de Médecine, novembre, 1861.

Eighteen months ago, weakness of sight supervened, with occasional diplopia. For the last six months he has had slight incontinence of urine with marked diminution of sexual power. Since this last date, he has for the first time been seized with suboccipital pain extending to the nucha and shoulders, becoming less violent, though not entirely removed, when he was lying down, but intensified by the standing or sitting posture, so much so as to be unbearable, and to compel him to lie down.

For some time now, he has been free from this pain. The left upper eyelid drops slightly; there is external strabismus of the left eye, the pupil of which is markedly more dilated than that of the right eye. In addition to the diplopia, which depends on the paralysis of the third nerve, there is a diminution in the range of distinct vision. Thus, although he can see perfectly a person when very close to him, he cannot distinguish his features when standing at a very short distance. The upper limbs and trunk are not affected, either as regards sensation or motility; but the lower extremities are strangely diseased. The act of walking is extremely painful and laborious, from the limbs moving with considerable stiffness and irregularity. The patient cannot control his movements, and is unable to come down a staircase without falling. He keeps looking at his feet when he walks, as if sight were needed for guiding them; and, indeed, when he shuts his eyes he cannot move a single step, unless he can rest on, or guide himself by, a piece of furniture. At night he has occasionally been obliged to go on all fours when he wanted to move a short distance from his bed. Yet, it is easily ascertained that his muscles have preserved their contractile power, for if an attempt be made to flex his legs on to his thighs against his will, he resists it with great vigor. When he has placed himself in equilibrium, he can stand very well, and can even bear a heavy weight on his shoulders. He is conscious of the contractions of his muscles, and knows when they are compressed. Even when his eyes are shut, he performs any movements which he may be asked. Tactile sensibility and sensibility to pain are perfectly normal in the lower limbs, even in the soles of both feet.

He has never had any boring pains, like those described by Dr. Duchenne (de Boulogne). For the last month he has been completely impotent; he has never had spermatorrhœa, or the rapid emission during coitus to which Dr. Trousseau has drawn attention. Professor Trousseau himself noted all the above symptoms, when he saw the patient in consultation.

The gastro-intestinal affection, which set in ten days after his admission, resisted all treatment. Intense and incoercible diarrhœa came on; the motions were passed involuntarily, vomiting and hiccup next set in, and the patient died of exhaustion, his intellect being unaffected up to the last. The post-mortem examination was made thirty-four hours after death. The weather was rather cold, and there were no signs of putrefaction. The cerebrum, cerebellum, and pons Varolii were injected in certain spots only, and looked healthy. The spinal cord, on the contrary, was deeply diseased. The examination was carried on by a distinguished micrographer, Dr. Luys, and the results are therefore highly important.

1. The *dura mater* (of the spinal cord) was very vascular in its whole length, and of a dark-red hue. It was also very markedly thickened in its upper portions, and somewhat œdematous. There was no trace of old exudations.

2. The spinal *pia mater* was likewise abnormally injected, more markedly so over the lower third of the cord and along its posterior columns. In those points, in fact, it strongly adhered to the posterior columns, and, like

them, had a yellow tint. It could not be separated from the cord without tearing away some of the nerve-substance.

3. The *posterior columns* were the parts most peculiarly diseased. They looked like two transparent vitreous bundles, in some parts of an amber-yellow color, and in others of a reddish-yellow tint, according to the greater or less degree of vascular injection. Their consistency was diminished, but they were not diffluent, and, moreover, there was no solution of their continuity. By spreading out their fasciculi with a needle, they could be easily traced for some distance.

This *degeneration* of the posterior columns was most marked in the lumbar region, but it extended to the dorsal also, being exactly limited to the space comprised between the posterior cornua. It gradually diminished, and finally disappeared in the brachial region, although, even on a level with the upper portion of the bulb, the white matter near the gray commissure could be seen to present appreciable traces of a similar degeneration.

This special discoloration of the posterior columns was owing to the transformation of their *nerve-tubes*. Most of these, indeed, had been destroyed; and there remained of them nothing but their empty sheaths, the walls of which were applied against one another. The nerve-tubes which had not wasted away completely, still retained their cylinders, with this difference, however, that these cylinders, instead of having smooth edges and a ribbon-like aspect, and of being of the faintest pale-yellow color, were now rough, jagged, and of a yellow tint like that of amber. Amidst the nervous elements, were scattered capillary vessels in large numbers.

4. The *lateral columns*, with the exception of a very slight and superficial yellowish discoloration in their lowest portions, were perfectly healthy in all their extent, from the lowest to the highest limits of the cord.

5. The *anterior columns*, in the lumbar region, were less thick, and less firm to the touch than usual; their color was normal, and differed totally from that of the posterior columns.

6. *Gray Matter.*—In the lumbar region, and for the lower fourth of the cord, it had lost its consistency, especially in its central portion. Its fibres were all more or less ruptured in places, although a few could still be traced; and in those points the shape of the anterior and posterior cornua was still perfectly distinguishable. Thus, on examining transverse sections of the cord, networks of cells extending from the posterior to the anterior cornua could be seen in some sections, whilst in others made one millimetre higher up or lower down, only ruptured fibres could be found, accumulation of fatty granules, and a shapeless detritus. Even in those degenerated portions, however, all the *nerve-cells* had not totally disappeared; a certain number of them could still be seen, with their prolongations, but most of them, either those of the anterior or of the posterior cornua, of the gray substance of Rolando, or of the intermediate regions, were shrunken, with jagged edges, and covered with a larger quantity than normal of granular pigment; in a word, they were undergoing a process of *involution*. The capillaries of the gray matter were considerably enlarged. The capillary plexus had evidently been the seat of partial and transient congestions, for in those parts where the gray substance was less consistent than usual, there were found amorphous and diffused deposits of hæmatin, pointing to antecedent congestive processes.

7. *Posterior Roots.*—The nerve-fibres were unfortunately examined only in that part of their course from the posterior columns to the ganglions, not beyond the latter. (A) First, as regards the condition of *the ganglions*. All those of the lumbar region were of larger size than normal, and unusu-

ally red and vascular. Their consistency was not diminished, and their enveloping membrane was notably thickened. On section, in addition to enormously dilated capillaries, there were found evident traces of old congestions, with diffusion of hæmatin. Moreover, the *ganglionic cells*, instead of being only partially covered by a few granules of brownish pigment, as they normally are, were sprinkled over with reddish-yellow granulations. Some of the cells were shrunken, with lacerated edges; others, on the contrary, were voluminous, pale, discolored, almost spherical, and looking very much like fat-cells, with which they might have been confounded, were it not for remaining traces of the old nuclei, and the vestiges of their nerve-tubes still adhering to their walls. A few of these ganglionic cells still retained their normal relations to the nerve-tubes which surrounded them.

The ganglions of the lumbar roots were the only ones which presented these lesions, limited to portions of ganglions.

(B) *Roots.*—The nerves of the cauda equina presented very characteristic appearances. Instead of their usual cylindrical form, firm consistency, and whitish color, they were flattened out, ribbon-like, and looked like strips of parchment which have long been macerated in water. Those which came from the anterior columns were transparent and of a grayish color, whilst those which were connected with the posterior columns had a vitreous aspect, and were of a uniform yellowish tint. Large vessels, more numerous than in health, accompanied the nervous fasciculi which were connected with the posterior columns.

All the nerve-fibres which connected the ganglions with the posterior columns were of the same yellowish color. The nature of the degeneration was the same, and produced the same collapse of the walls, and the same amber-yellow aspect of the cylinders, when these were still present.

The posterior roots were diseased in the lumbar region only; in the dorsal region, they by imperceptible degrees resumed their normal aspect, and in the upper regions of the cord they were not in the least modified. Thus the roots of the *glosso-pharyngeal, vagus, auditory,* and *trigeminal* nerve, were not appreciably affected.

8. *Anterior Roots.*—Generally speaking, the anterior roots were infinitely less affected than the posterior. In the lumbar region these roots were less firm than usual, grayish, and transparent. The nerve-tubes were not very sensibly altered, and in most of them continuous cylinders could be seen, unbroken and contained in a normal sheath. The intervening nerve-substance was very markedly diminished, so that these nerves, which are usually of large size and of a white color, looked, in consequence of this atrophy, like the nerves found in the gray portions of nervous centres, and which are without white substance.

In the dorsal region, the anterior roots looked normal again, as well as higher up in the cord. The roots of the *spinal accessory,* and of the *facial,* looked healthy on both sides, as well as the roots of both *hypoglossal* nerves.

The *abducens oculi,* however, and the *motor oculi,* on both sides, looked remarkably altered. The latter had the aspect of grayish cords somewhat œdematous, and shrunk to nearly half their usual size. They readily gave way when, by gentle pulling, an attempt was made to remove the brain from the cranial cavity. The *abducens oculi* was similarly affected on both sides, although to a less extent. They were both of diminished volume and consistency, and of a grayish color. The walls of their nerve-fibres were collapsed, and in some their contents (white substance and cylinder axis) had been completely reabsorbed. Numerous capillaries were interlaced round the nervous fasciculi.

On following up, in the gray matter of the fourth ventricle, the trunk of the abducens oculi as far as its real origin, large vascular trunks were found in a series along the course of the primary fibrillæ of this nerve, which they must probably have compressed considerably.

The roots of the *fourth nerve* had the same color and consistency.

The next case which I am now going to bring under your notice is that of a patient who died in Dr. Vigla's ward, and I owe the following details to Mr. Dumontpallier, who assisted Mr. Sappey in making the post-mortem examination :

Pothel, aged 55, formerly a messenger, has enjoyed good health previous to his present ailment. There has been no nervous complaint of any kind in his family. He has been a messenger since the age of 18, and has thus been exposed to changes of weather and to great fatigue, which did not, however, affect his general health. He has never had syphilis.

In 1849 he felt shooting pains always attacking the same parts of the trunk or lower limbs, and recurring in paroxysms about every fifteen days for half an hour. At first he paid little attention to them, on account of their short duration and the distant intervals between the paroxysms, and he went on with his usual occupation. These pains, however, by degrees returned at shorter intervals.

Two years afterwards, in 1851, he became impotent; in 1852 his gait became a little uncertain, and by the end of the year he could neither walk nor even stand, and he had to be carried. He noticed nothing peculiar about his eyes or bladder.

In 1861 he was admitted, under Dr. Vigla's care, whilst suffering from bronchitis, and the presence of pulmonary tubercles was diagnosed.

Dr. Duchenne (de Boulogne) having been requested by Dr. Vigla to examine the patient, ascertained from him the above details. He found, besides, that cutaneous and muscular sensibility was perfect everywhere, that partial movements were performed with normal power when tested whilst the patient was in a *sitting* or *lying* posture, and that, nevertheless, the patient could not maintain his equilibrium when standing, nor *take a single step unless supported by two attendants*. The want of harmony in the movements was such that the patient could not walk with the slightest regularity. He left the hospital after a two months' stay.

In April, 1862, he was again readmitted with symptoms of galloping phthisis. With regard to his locomotor ataxy, the following notes were taken: His *muscles* are very much wasted, but not more in one place than in another. Cutaneous and muscular sensibility is considerably diminished in the right leg. When he is pinched, the painful sensation is only perceived three seconds afterwards. In the left leg cutaneous and muscular sensibility is more acute than in the right, but is still abnormally diminished except on the posterior aspect of the limb. Pinching is faintly perceived.

In both thighs the various kinds of sensibility are normal. In the soles of the feet the sensibility to tickling and pressure is considerably diminished, especially on the right side. Sensibility to pinching has almost entirely disappeared.

The sensibility of the upper limbs and trunk is everywhere normal. Partial movements are still executed, although very feebly in consequence of the wasting of the muscles. The patient is incapable of standing or even of sitting. There is no affection of the bladder or of the eyes.

Six days after the above notes were taken, he died of his pulmonary disease.

The post-mortem examination was made twenty-four hours after death, and Mr. Sappey examined the spinal cord under the microscope. The brain was well formed, of normal consistency, and without a trace of injection. Successive sections of the organ showed it to be healthy throughout.

The cerebellum, pons Varolii, and medulla oblongata were also healthy.

The cervical and dorsal portions of the spinal cord were of normal size, color, and consistency; the lumbar portion alone was slightly diminished in size.

On dividing the latter transversely at its upper limit, the surface of section of the posterior columns was seen to be of a grayish hue, evidently pointing to an alteration of these columns, which were still, however, of normal consistency.

The anterior roots of the lumbar portion of the cord were normal; the posterior, on the contrary, were very considerably atrophied, this atrophy being peculiarly striking when a healthy cord was placed by the side. The roots were thus shown to have lost about two-thirds or three-fourths of their normal size. Their aspect was also considerably modified; they were not white, but of a reddish-gray color, looking pretty much like bundles of capillary bloodvessels. Besides, they did not project sufficiently from the surface of the cord, as they issued from it, but spread out like delicate ribbons of scarcely any thickness.

Under the microscope, the nerve-tubes of the posterior roots were seen to have lost a considerable portion of their medullary substance, although some of them looked still normally full, and contained a cylinder axis. Those that were reduced in size were contracted in one point, swollen out at another; in a word, they were very irregular. In a great many, the medullary substance had completely disappeared, so that they looked constricted here and there. In some, again, vestiges of the medullary substance reappeared at long intervals. Where it was completely absent, the tubes, when examined with a power of 400 diameters, had a filiform appearance, without, however, presenting a perfectly regular contour.

As the nerve-tubes were unequally altered, the progressive series of their degeneration could be traced out. Those that were normally filled with medullary substance, accounted for the retention of sensibility in several portions of the integuments; whilst the empty or nearly empty tubes explained the impairment of sensibility in the lower limbs.

The rapid post-mortem alteration of the cord unfortunately prevented an examination of the posterior columns.

Now, gentlemen, do you not find singular contradictions between the symptoms of progressive locomotor ataxy and the pathological appearances found in the cord?

In a disease essentially characterized by disorders of motility, and in which the loss of sensibility is relatively of secondary importance, since this property may be more or less unimpaired, one might have expected to find the *anterior* not the *posterior* columns diseased, according to the physiological doctrines professed by Mr. Longet. Yet the absence of disease in the anterior columns accounts for the absence of real paralysis, and the defect of co-ordinating power may be physiologically explained by disease of the posterior columns. This want of co-ordination, as I have several times told you before, is not due to anæsthesia, for this may be transitory only, or very slight in degree, or even be completely absent.

As to the persistence of sensibility, notwithstanding the grave lesions of the posterior columns and their corresponding roots, microscopical examination has proved that there still remained in the degenerated nerve-tissue a variable number of healthy nerve-tubes, from which it has been concluded course, to use a stick, which brings him back into the normal direction which he is constantly losing.

Now, in order to explain the simultaneous existence in ataxic patients of extreme incoördination of movements and very slight diminution, or even perfect preservation, of cutaneous and muscular sensibility, the existence of a *deep* or *common sensibility* (*cœnesthesia*) has been assumed, the impairment of which is said to account for the phenomena. The author[*] of a recent critical review on the subject defines this new kind of sensibility thus: "It is the very distinct perception (although almost unnoticed from its being so continuous) which we all have of the presence of our organs, their volume, weight, shape, situation, and relations. From all the points of the organism there constantly ascend to the nervous centres an infinity of sensitive currents which, luckily for us, are not noticed by the encephalon, but the *interruptions* or *irregularities* of which strike us forcibly as soon as they occur."

I confess, gentlemen, that I cannot conceive a common, unnoticed sensibility, which reveals itself to us only when it is *interrupted*, in other words, when it ceases to exist. The very men who admit its existence, confess that physiology cannot teach us what it is, and that pathology alone can do so. Yet the few examples which have been given in illustration have not convinced me. They all consisted of cases of individuals "who could not tell positively the position of their limbs, unless they saw them, and who suffered merely from a very slight impairment of tactile sense, limited to a circumscribed region of the trunk." The same author cites the cases of two women, observed by himself, one of whom "was obliged to note the place where she lay her hands before going off to sleep, in order to be sure of finding them in the dark." This case would appear of little value to me, even if the observer had noted the condition of the *articular sensibility*, which he forgot to take into account.

Remember, however, that this so-called *deep* sensibility should not be confounded with the *organic* sensibility which nobody denies. This latter manifests itself, only when it is exalted in pathological conditions. The stomach, for instance, which in health performs its functions silently, makes its presence felt in bad digestion; and other organs, which are normally insensible, acquire, under the influence of what is called inflammation, an exaggerated sensibility, and become the seat of the most acute pain.

I now pass on to the *differential diagnosis* of locomotor ataxy. It most frequently attacks the lower limbs first: the patient believes that he is seized with paralysis, and his mistake is shared by some medical men, very excusably indeed when there is real paralysis of the bladder and rectum at the same time, with a sense of circular abdominal constriction, tingling of the lower extremities, &c. In order to avoid this mistake, however, the patient's muscular power need only be tested, and the absence of real paralysis will thus become evident.

Tumors of the cerebellum give rise to a form of ataxy. Dr. Hérard has lately published, in the Union Médicale (t. iii, 1860, p. 230), a very remarkable case of the kind. The patient had lost the power of associating, combining, and co-ordinating the movements which make up the complex act of locomotion, standing, &c. There was no paralysis of sensation or motion, general or partial, direct or crossed. So far, the symptoms of the case resembled those of locomotor ataxy, since the latter disease need not be accompanied by paralysis. A distinction, however, was afforded by the extreme frequency of vomiting, in Dr. Hérard's case, which symptom, according to the interesting researches of Dr. Hillairet, accompanies cerebellar diseases. Moreover, the patient had never presented the premonitory symptoms of genuine locomotor ataxy, namely, the characteristic pains in the limbs and the trunk, the disturbances of vision, and the various forms of local paralysis, &c.

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[*] Dr. Axenfeld: "Des Lésions atrophiques de la moelle épinière." Archives Générales de Médecine, août et octobre, 1863, p. 486.

I need not go into the differential diagnosis of ataxy and the general paralysis of the insane and chorea: these two last diseases are attended with such characteristic symptoms that a mistake seems to me impossible.

When speaking just now of cerebellar ataxy, I should have mentioned Professor Bouillaud as having been the first who experimentally and clinically studied the effects of injuries to the cerebellum. As far back as 1828, and later in 1847, in his "Nosographie Médicale," my learned colleague described *the various co-ordinated movements* which injuries of the cerebellum impaired; namely, walking, standing, and the maintenance of equilibrium. For a detailed account of his researches, I must refer you to some lectures delivered by Professor Bouillaud, and which have been recently published by Dr. Auguste Voisin.*

§ 2. *Pathological Anatomy of Progressive Locomotor Ataxy.—Relation of the Lesions to the Symptoms.—Nature of the Disease.—Treatment.*

In his treatise on "L'Electrisation localisée," in which he was the first to give the clearest and most complete description of the symptoms of progressive locomotor ataxy, Dr. Duchenne (de Boulogne) says not a word of its *pathological anatomy*. At least, he gives one case only in which he had occasion to seek, after death, for the alterations which might characterize the disease; and in that case, the subject of which was an individual who, in 1858, died in one of Dr. Nonat's wards at the Charité Hospital, "*the encephalon and spinal cord, on the most careful examination, presented no anatomical lesion appreciable to the naked eye.*"† These negative results did not support the theory which Dr. Duchenne had, *à priori*, broached of the nature of the disease. Indeed, reasoning from the fact that, since the beautiful researches of Flourens and Bouillaud, the cerebellum was looked upon as the seat of the faculty of co-ordinating movements, Dr. Duchenne suggested that, in locomotor ataxy, the defect of motor co-ordination, which constitutes its primary phenomenon, must be "*necessarily dependent on some structural or functional lesion of the cerebellum.*" Secondly, when he took into account the order of sequence, and the progress of the symptoms, he was led to believe that the central morbid process which gave rise to the symptoms, generally began in the motor nerves of the eye and the corpora quadrigemina, from which they extended to the superior peduncles of the cerebellum, and lastly to that organ itself.

Now that a pretty good number of cases have been recorded, showing that in progressive locomotor ataxy it is the spinal cord which is diseased, and nearly always a limited portion of the cord, the dorso-lumbar especially, and very rarely the cervical, whilst the cerebellum is not notably affected, Dr. Duchenne has given up his first theory. The anatomical appearances found in ataxy are confined, as a rule, to the posterior columns of the cord and the roots which issue from them; it is only in exceptional cases that the antero-lateral columns are implicated as well. These appearances conin progressive locomotor ataxy were not the cause, but the effect, the product of the disease.

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* Union Médicale, 18, 25, et 28 juin, 1859.

† "Traité de l'Electrisation localisée," p. 608.

When we examine the lesions found, we are struck with three facts: First, the atrophy of the nerve-tissue of the posterior columns and the corresponding roots; secondly, the development of cellular tissue, or if you prefer the term now generally adopted, the hypertrophy of the *neuroglia;* thirdly, the vascularity of the diseased tissues.

The atrophy of the nerve-substance is the most striking phenomenon. It evidently does not belong to the category of those simple forms of atrophy which takes place in organs condemned to prolonged physiological inactivity, and which is the counterpart of hypertrophy due to an excess of functional activity. But, although it is the most striking phenomenon, this atrophy is only a consequence of the pathological *evolution* of the cellular element, which, by developing itself, has crushed the nerve-elements contained within its areolæ; and this abnormal development of the cellular tissue is itself dependent on the increased vascularity of the tissues.

Now is this increased vascularity sufficient to characterize inflammation, and are we from its presence to conclude that progressive locomotor ataxy is only *a variety of chronic myelitis?* If so, how are we to explain why this myelitis is always so exactly limited to the posterior columns of the cord and to the roots issuing from them? and particularly why, during life, it is attended with symptoms differing so much in their form, course, and changeability, from those common to all varieties of myelitis?

This abnormal vascularity of the posterior columns of the cord, which is again observed in the motor oculi and optic nerves and the tubercula quadrigemina, seems to me a consequence of frequently repeated congestions, analogous to those which we see during the patient's life affecting the conjunctiva. This membrane, as I mentioned to you already, gets injected in the interval between the paroxysms of pain, simultaneously with the occurrence of contraction of the pupil, which is sometimes carried to an extreme degree. Generally, however, this injection disappears on the supervention of pain, especially of pain in the head, whilst the pupil dilates more or less at the same time.

These congestive phenomena show themselves in other diseases acknowledged to belong to the class of neuroses, such as hysteria, asthma, and Graves's disease (exophthalmic goitre); and they belong in my opinion to the same category as those which in his experiments Professor Cl. Bernard produces at will by dividing the sympathetic.* They point to some disturbance in the functions of that nerve, of which we neither know the nature nor the cause.

It now remains for me to speak of *the treatment of progressive locomotor ataxy.* I am unfortunately compelled to be brief on this point, for the great number of remedies which have been tried against this disease, is a proof of their inutility and of the impotence of medicine. If as yet, however, we do not possess any means for curing this affection, or even for arresting its progress, we can still, in certain cases, modify and moderate some of its symptoms, and thus procure some alleviation. Above all, we are to avoid remedies which have proved not only useless, but dangerous. Thus we should reject bloodletting absolutely, whether general or local; purgatives also, which, when often repeated, act in the same way; revulsives, cauteries, moxas, or setons, which, by causing irritation of the skin, may bring on the special pains of ataxy in the spots where they are applied.

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* "Leçons sur la Physiologie et la Pathologie du Système nerveux." Paris, 1858, t. ii.

Flagellation, however, used methodically, and in moderation, in some of my cases, has diminished the pains.

Dr. Duchenne (de Boulogne) also states that he has often known *cutaneous faradization* diminish the cutaneous and muscular anæsthesia which, at an advanced period of the disease, aggravates the want of co-ordination of the movements; and the result, he adds, has been great improvement in the manner of walking.

In order to calm the pain, which is sometimes very acute, I have recourse to *belladonna* and *spirits of turpentine*, which I prescribe alternately, for ten or fifteen days successively, in gradually increasing doses.

Lately, Professor Wunderlich has published several cases in which the progress of the disease has been apparently checked by the internal administration of *nitrate of silver*. Drs. Charcot and Vulpian, who tried this remedy in their turn, have reported favorably of it.* Since then, other cases have been published in favor of it; but, unfortunately, cases may be opposed to these, in which the nitrate of silver failed completely. I myself have very often prescribed it both in my private and hospital practice, and although I, *à priori*, expected some good from a remedy which I had found useful in a good many neuroses, I must confess that in locomotor ataxy it has not fulfilled my expectations.

Hydropathy and *sulphur baths* are indicated in this disease as general modifiers; and above all, the patient's strength should be supported by all the means in the physician's power.

LECTURE LX.

PROGRESSIVE MUSCULAR ATROPHY.

Pathological Anatomy.—Lesions of the Muscles.—Lesion of the Nervous System.—Is the latter constant?—Symptoms.—The Atrophy generally begins in the Upper Extremities.—Of the exceptions to this rule.—Prognosis, fatal.

GENTLEMEN: I am enabled to-day to complete the details of the autopsy, which we made a few days ago, of a patient who died at No. 10 in St. Agnes Ward. M. Ch. Robin, who kindly undertook to make a microscopical examination of the muscles, has sent me a report which I am going to read to you. I wish first to state, however, that the autopsy was made in my presence by M. Sappey, who is at the head of the anatomical department of our Faculty, and that this skilful anatomist found no other lesion, besides the alterations of structure of the muscles, than marked atrophy of the anterior roots of the spinal cord, and *perhaps* a slight diminution in size of the anterior columns.

M. Robin's report is as follows:

"In all the muscles, even in the palest, there is found a certain number of fasciculi, the fibres of which are marked by transverse striæ, although these striæ are, it is true, paler and less distinct than they normally are. The fasciculi of the diseased muscles are one-third less in diameter than the bundles which have retained their normal structure, and which have a diameter varying from $\frac{7}{100}$ to $\frac{12}{100}$ of a millimetre. The granular condition, paleness, and transparency of the atrophied bundles are most marked in the spots where their diameter is most notably diminished. This fact is particularly striking in the interossei muscles, in which a pretty large number of bundles are seen, the sarcolemma of which is completely empty, and so collapsed as to be reduced to a diameter of from $\frac{8}{1000}$ to $\frac{10}{1000}$ of a millimetre. The paleness and transparency of these muscles is remarkable. By the side of fasciculi which are not very atrophied, some are seen in a perfectly granular condition, and others still marked by very pale striæ; lastly, the most atrophied bundles are mixed up with others presenting, nearly all of them, the same degree of atrophy. There is no lesion of the intermuscular cellular tissue. *Fat-cells are not seen in greater numbers than normally.* The degree of paleness of the muscles is evidently proportionate to the number of bundles presenting varying degrees of atrophy, from a simple diminution in size, with indistinctness of the transverse striæ, up to a granular condition without apparent striæ, and with extreme diminution of size.

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* CHARCOT et VULPIAN: "De l'Emploi du Nitrate d'Argent dans le traitement de l'Ataxie progressive." Bulletin Général de Thérapeutique, 1862.

"Fat-cells are found in as large numbers in the reddest muscles (which yet present a few bundles that are smaller and paler than the rest, and some of which are already granular and have lost their striæ) as in the palest ones, such as the interossei muscles of which I have spoken, and the muscles of the hypothenar eminence. This anatomical fact was most apparent in the intercostal muscles, a very small proportion of the fasciculi of which have become pale, and in which the granular condition is still more rare."

This case, gentlemen, so far as the results of a microscopical examination of the diseased parts are concerned, cannot, according to M. Duchenne (de Boulogne), be regarded as a perfect type of progressive muscular atrophy, or the anatomical lesion had not at least reached its maximum, namely, the stage of fatty metamorphosis. M. Ch. Robin, it is true, declares that he has never met with this change, but his views are opposed by men whose opinions in microscopical matters is law in science. I need only mention Professors Virchow (of Berlin), Friedreich (of Heidelberg), and Lebert (of Breslau), who believe, as do also Professor Cruveilhier and M. Duchenne (de Boulogne), that progressive muscular atrophy is anatomically characterized by the diminution in size of the muscular bundles (the transverse and longitudinal striæ of which are seen to disappear at a more advanced stage), and, lastly, by the production of granulations, which ultimately become fatty. I declare myself incompetent to decide the question. But whether these granulations be fatty or not, the point, however interesting it may be in pathological histology, is of little importance in a clinical point of view. It would be of greater use to us if we could know whether the muscular lesion is primary, or whether it is dependent on a lesion of the nervous system, either of the cerebro-spinal centres, or, as it has been stated, of the anterior roots of the spinal cord. You are aware, gentlemen, that in his memoir on Progressive Atrophic Muscular Paralysis, read at the Academy of Medicine in March, 1858, and published in the "Archives of Medicine" in May of the same year, Professor Cruveilhier adopted the conclusion that this motor paralysis, which was sometimes local and sometimes general, and coincided with the complete retention of sensation and intelligence, was due to atrophy of the anterior roots of the spinal nerves.

My honored colleague quoted in support of his view the case of a man named Lecomte, which had been already given in detail in a memoir by Dr. Aran,* and has been since reproduced in the work of Dr. Duchenne (de Boulogne).† There was, in that case, marked atrophy of the anterior roots of the spinal cord, in the cervical region chiefly; and in the case which suggested the present lecture, there was found atrophy of the same parts. From other cases, however, in which these roots are said to have been in a perfectly normal condition, one may be led to believe that this peculiar lesion was by no means the primary cause of the progressive muscular atrophy.

As to a lesion of the nervous centres, the integrity of the intellectual functions, the absence of all symptoms during life of paralysis proper, and after death the absence of anatomical changes in the spinal cord and the brain, prove conclusively that the great centres of innervation are not in the least involved in this complaint. And yet I have told you that M. Sappey thought he had found in my patient a diminution in size of the anterior columns of the cord.

Even though progressive muscular atrophy belong to the class of neuroses, as I believe and admit, the morbid process which characterizes it still begins primarily in those muscles themselves which are involved, and in their intimate elements. However interesting it may be for a physician to acquire precise notions as to the proximate cause of a disease, what he requires above all, and what is to him of immediate use, and he cannot dispense with, is a full knowledge of the symptoms, and a correct appreciation of the characters, by means of which alone he will be enabled to diagnose the disease. Let us, therefore, inquire what are the *symptoms of progressive muscular atrophy*. They have such a peculiar physiognomy that, when they have been once observed with attention, they can be very rarely mistaken. The first sign of disease of which the patient complains is a diminution of muscular strength, at first confined to one limb only, and increased by cold and by exercise—the difficulty which he has in executing a movement sometimes increasing to such a degree that he is perfectly incapable of performing it. This weakness, which is local at first, confined to a single limb, and even to a portion of one limb, and affecting certain movements only, is generally attended with cramps (*subsultus tendinum*), whilst the diseased muscles are often the seat of fibrillary contractions. I say *often* only, because one must not think that these fibrillary contractions are a symptom *sine quâ non* of progressive muscular atrophy, and constitute one of its fundamental characters: for, on the one hand, it is not rare to find that they are completely absent during the whole course of the complaint; and, on the other hand, they are observed in other muscular affections which are very different from the one under consideration. They are analogous to the contractions which are noticed in diseases of the spinal cord, and, like them, they are independent of the will, occur spontaneously, but are never so violent and so repeated as when the muscles in which they show themselves are excited, either by being percussed, compressed, pinched, or maintained in a state of forcible contraction, or when they are galvanized. They last a very short time, and sometimes follow one another with such rapidity, and such frequency, that the affected muscles seem to be in a state of constant action: at other times, on the contrary, they are so very rare that one must watch the movement when they occur, and even irritate the muscles in order to produce them. When they do occur, they give rise to an appearance of very fine whipcords, which are alternately tightened and relaxed under the skin with extreme rapidity: at other times the movements are slight and of a vermicular character. As they are unattended with pain, and are involuntary, they are often unperceived by the patient, although they are sometimes spoken of by some as a very slight quivering. When these fibrillary contractions affect a muscular bundle of pretty large size, they give rise to very perceptible convulsive movements, chiefly in the limbs; and patients state that their fingers are alternately flexed and extended suddenly, independently of their will.

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* Archives de Médecine, Sept., 1850.

† "De l'Electrisation localisée." Paris, 2e édit., 1861, p. 437.

This sensation of quivering is not the only one, according to Dr. Duchenne (de Boulogne), which is mentioned by individuals suffering from progressive muscular atrophy. In a pretty advanced stage of the disease, they complain of a sense of cold in the atrophied limb, consequent on a real lowering of its temperature, which can be appreciated by the physician. The capillary circulation has by this time become less active, whilst the cutaneous veins enlarge, and the skin gets livid under the influence of cold. The muscular weakness, which is the first striking symptom of progressive muscular atrophy, very rarely sets in suddenly; and even when this seems to have been the case, the accuracy of the patient's statement might very well be questioned. Nearly always, not to say always, the disorders of locomotion manifest themselves slowly and gradually. At the outset, as I told you a moment ago, this weakness is localized in and restricted to a limb, or even a part of a limb, interfering with some movements more than with others, increasing under the influence of cold or of fatigue to such a degree as to take away all motor power. By degrees the affection becomes general, and involves the whole of the limb—not all its muscles in an equal degree, however, and even sparing some of them entirely. The opposite limb is in its turn attacked, and, at last, the whole muscular system of the life of relation is more or less generally implicated.

According to Dr. Duchenne, this weakness is not due to deficient nervous excitability, but is the result of changes in those muscles, the fibres of which are more or less destroyed, and which consequently lose the power of performing movements. Voluntary muscular contractility remains normal unto the end, even in the most advanced stage of the disease, in those fasciculi which have not undergone a morbid change. This is an all-important fact, which has been and could only be brought to light by means of localized faradization. I hasten to add that the discovery of this fact is solely due to Dr. Duchenne (de Boulogne).

No one is more disposed than I am, gentlemen, to give Dr. Duchenne his due, and to declare that to no physician is the study of diseases of the nervous system more indebted than to him for real progress, but I cannot completely indorse his opinion touching progressive muscular atrophy. I read to you, in the beginning of this lecture, the details of the microscopical examination made by Dr. Ch. Robin, and you may remember that there still remained in that case a good many muscular fibres which were apparently sound, whilst others were already altered, and others again were so very discolored, and so deeply modified, that one could conceive that they had lost all contractile property. You may remember also that Dr. Duchenne himself, who honored us with his presence during our round, had shown that most of the muscles of the arm and forearm still contracted under the influence of electricity, whilst the patient could not voluntarily move his hands or his forearms. One could but suppose, therefore, that previous to any anatomical change which seemed not to exist then, the peripheral extremities of the nerves had undergone a modification, in consequence of which they had lost the power of rousing muscular contraction.

A loss of excitability of the peripheral extremities of nerves would therefore precede the degeneration of the muscular fibres, a fact perfectly in accordance with pathological physiology.

The difficulty felt in the performance of movements generally coincides with very marked thinning of the diseased parts. I say generally, because cases have occurred (and Dr. Duchenne has at least recorded a remarkable instance of the kind) in which muscular atrophy coexisted with a considerable degree of plumpness, and was only characterized by a diminution of motor power. The emaciation presents, besides, a very characteristic feature. It is only seen in those parts which correspond to the diseased muscles, while the rest retains its normal size; and it differs, therefore, from the general emaciation which obtains in individuals exhausted by prolonged suffering, or which is sequential to paralysis, even when localized, as in the case of lead-palsy. The destruction of the muscular masses, which is the cause of these alterations of shape, also produces changes in the attitude of the limbs and trunk during muscular rest, in consequence of the loss of equilibrium between antagonistic muscles. Lastly, apart from motor weakness, there is also inability to co-ordinate voluntary movements, as the diseased muscles can no longer act in concert with the rest.

I shall presently revert to these peculiarities; but a most curious one, to which I am desirous of first drawing your attention, is the favorite seat of progressive muscular atrophy, at its onset, in the upper extremities.

Usually—at least 9 times out of 11, according to an analysis of cases made by Aran—the disease primarily attacks the upper limbs, and more especially the right limb, 7 times out of 11. Its seat is still more localized, for the muscles of the hand are the first to be involved—those of the thenar eminence in the very beginning, those of the hypothenar next, and the interossei afterwards. You must not think, however, that all the muscles of the diseased region, or even all the fasciculi of a single muscle, are simultaneously affected. This is far from being always the case, for by the side of atrophied muscles, there may be others in the same region, their congeners even, which are untouched and act in the place of the former, as these are necessarily unable to discharge their functions. Again, there may be in the same muscle, by the side of fasciculi which have undergone a morbid change of structure, other fasciculi which may be called into contraction by means of faradization, and which must therefore be made up of healthy fibres. Thus, in the case of a patient lying in bed 23, St. Agnes Ward, which I shall presently relate to you, the atrophy commenced in the left deltoid muscle, and yet, at the end of three years and a half, the posterior fasciculi of that muscle were still in a nearly normal condition. They contracted in obedience to the will and under the influence of electricity, whilst after the patient's death, they were found to be of normal color and size, and under the microscope they exhibited very regular transverse striæ. The fibres of the anterior and middle fasciculi, on the contrary, either showed no transverse striæ at all, or only in spots here and there; or even, as in the ultimate stage of the degeneration, the sarcolemma only contained extremely fine granulations, mixed up with fat-cells in greater or less numbers.

This is the rule, but there are exceptions to it, for Dr. Duchenne (de Boulogne) has pretty frequently seen the disease commence in the muscles of the trunk. Thus, he saw it once begin in the sacro-lumbales muscles; in another case, the pectorales, trapezii, and latissimi dorsi were destroyed before the upper limbs became involved; and then a large portion of the biceps in the arm, and the supinator longus in the forearm, were affected, whilst the motor muscles of the hand escaped entirely. In another case, again, the muscles of the trunk, the pectorales, trapezii, rhomboidei, latissimi dorsi, serrati magni, some muscles of the lower limbs, among others the flexors of the leg, were atrophied, whilst in the upper limbs the supinatores longi were alone destroyed; in two cases the lower limbs were the first involved. Lastly, in a case which I had the opportunity of seeing with Dr. Duchenne (de Boulogne), and which he has published in his treatise on Localized Electrization, the disease became general in less than two years, and ran a most irregular course.

The patient was a Spaniard, aged 32, who had come from Barcelona to Paris. The motor muscles of the right hand were the first to waste away, and after them the flexors of the left foot. The left hand atrophied next, and after it the flexors of the right foot and those of both thighs. Lastly, the atrophy extended in a variable degree and in the following order: to the biceps, the deltoidei, the muscles of the trunk, those of the neck, and those of the face. At the time when these notes were taken, the diaphragm and the muscles of deglutition were so seriously involved, as to threaten the patient with the risk of dying of starvation or of asphyxia.

At the close of the year 1860, a gentleman, practicing in the south of France, sent me a lady, aged 30, who had been paralyzed for several years past. She was remarkably plump and fresh-looking, and I only saw that she had lost all muscular power, but could not find out the cause of it. Having asked Dr. Duchenne to join me in consultation, I must declare that before he had asked the lady four questions, he had recognized, and made me recognize, with the greatest facility, progressive muscular atrophy, bidden under heaps of fat. What had contributed to deceive me, was the fact that the lady, who was an excellent musician, tried to console herself of her sad infirmity by playing the piano, so that she had lost none of the strength and precision of the movements of her hand and forearm. In her case the deltoidei, rhomboidei, serrati magni, sacro-lumbales, psoæ and iliaci muscles, were more particularly diseased.

I repeat, gentlemen, these are exceptions, which do not in the least invalidate the general law, that muscular atrophy first shows itself in the upper extremities. The course of the disease has been carefully studied, and it has been ascertained that after the muscles of the hand, especially those of the thenar and hypothenar eminences, have been involved, the interossei, and the flexors and extensors of the fingers, and in some cases the muscular masses of the posterior region of the forearm, atrophy in their turn.

The disease, being thus restricted to these localities, may remain stationary and not spread beyond these limits for several years; but when it passes them, it affects almost simultaneously the muscles of the arm and of the trunk, although in every case only partially and very irregularly. In the arms, the biceps wastes and then the deltoid—in some cases the former before the latter; in others, on the contrary, the reverse obtains. The triceps is the last to undergo change. According to Dr. Duchenne, the course of the disease is generally the following: the trapezius disappears first, but it is a remarkable fact that its lower portion alone does so; whilst its clavicular portion is, on the contrary, the last involved of all the muscles of the trunk and neck. Next in succession come the pectorales, the latissimi dorsi, rhomboidei, levatores anguli scapulæ, the extensors and flexors of the head, the sacro-lumbales, and the abdominal muscles. In nearly every case Dr. Duchenne has found that, at this period of the disease, the muscles of respiration, of deglutition, and of the face become involved.

If, in very rare cases, muscular atrophy shows itself first in the lower limbs, in general the muscles of those regions undergo transformation only after those of the upper limbs and trunk have been to a great extent destroyed. The disease seems to concentrate itself in the flexors of the foot on the leg, and those of the thigh on the pelvis, the remaining muscles undergoing change in the long run only.

The disease never shows itself at once in the two sides of the body, but when it has attacked a certain group of muscles, their homologues are not long before they are similarly affected, previous to the extension of the disease to other regions.

The modifications in the shape of parts which have lost their muscles, the changes in the attitude of a limb and the trunk, consequent on the destruction of the muscles, are characteristic of and special to progressive muscular atrophy. I mentioned to you just now the emaciation which accompanies the weakness of the patient's movements, and the loss of freedom in their performance, of which he complains; and I stated that the emaciation was by no means like the one which follows long-continued and exhausting diseases, or which obtains in paralysis, when the parts which have lost the power of moving diminish in size. In such cases, even when the paralysis is confined to a certain group of muscles, as in lead-palsy, the emaciation is uniform, while it is merely partial in muscular atrophy—so much so, indeed, that by the side of parts almost deprived of all muscular substance, others may be seen which have preserved their regular shape, and the prominence of which contrasts with the depression of the former.

This characteristic alteration of shape and these contrasts may affect all the various regions of the body, or they may be confined to more or less limited areas, and present an infinite variety of aspect and seat. Thus, an individual whose chest-walls are perfectly bony from the atrophy of his pectorales muscles, and whose scapula and its bony eminences stand out in relief through the disappearance of the fleshy masses of his back and of his trapezius and rhomboidei, may have arms which are still strong and with well-developed muscles, a face with perfectly regular features and of its usual plumpness. In another case, in which the disease has not spread beyond the upper extremities, you will be surprised to find that the hand and forearm have wasted considerably, whilst the muscles of the arm have undergone no change. In another case, again, the atrophy being incipient only, and having attacked the hand alone, the thenar eminence has disappeared, and in its place a hollow is seen, caused by the disappearance of the superficial and deep layers of muscles; and later, deepening of the interosseous spaces is noticed, consequent on the degeneration of the muscles which filled them. I shall not pursue this analysis further, for the best descriptions cannot give an accurate idea of these alterations of shape which are special to atrophy, and which need be seen but once in order to enable one to recognize the disease at first sight.

There is one point, however, to which Dr. Duchenne (de Boulogne) has called attention, and which I must not omit—namely, that this alteration of shape, which is a pathognomonic feature of atrophy, may be absent in some cases, even when a great many muscles are entirely atrophied, from its being masked by a considerable degree of stoutness. I mentioned to you just now a remarkable instance of the kind, and another is recorded by Dr. Duchenne—namely, that of a man afflicted with progressive muscular atrophy, localized on each side in the trapezii, rhomboidei, latissimi dorsi, and serrati magni, coinciding with extreme obesity. The case is so interesting on many counts, that I must request permission to quote it *in extenso:*

"M. R——, of Aix, in Provence, aged 22, tall, extremely stout, and of a robust constitution and sanguine temperament, has had no other ailment but the one for which he is now applying for advice. His great-grandfather, his grandfather, and his father, all of them *eldest sons*, were attacked with the same complaint—the first between 20 and 22 years of age, the second when 24 years old, and the third at the age of 17. In all of them the disease showed itself successively in the muscles which move the shoulder, next in those of the arm, and lastly in the flexors of the thigh on the pelvis, and those of the foot on the leg. M. R—— became affected at the age of 17. Until then he had been rather thin; but from the age of 18, he grew gradually stouter, so much so as to be exempted from the conscription on account of obesity. From the time that this obesity set in (which likewise showed itself in the case of the members of his family who labored under this muscular affection), there came on weakness of some movements. First, the act of raising the arm, or of carrying his hand to his head, became more and more painful. During this movement the scapulæ were extremely prominent, and their inferior angle, instead of moving outwards and forwards, moved backwards. These phenomena became gradually more and more marked, until the present date. M. R——, for the last twelvemonth, has complained of some fatigue in walking, and especially in going up a staircase. He has never felt any pain, and has not had articular or muscular rheumatism, or any syphilitic affection.

The disease, which has for the last three generations attacked the eldest sons in his family, has until now remained a mystery. Although he has already lost to a great degree the power of raising his arms, he still hopes that he may escape the fate of his predecessors, because he believes that the disease is, in his case, limited to the muscles which serve to raise the arm, and grounds his belief on the development of his limbs and the soft parts of his trunk.

On testing the condition of his muscles, however, by electricity, a large portion of the pectorales were found to have disappeared, and no traces could be detected of the trapezii, rhomboidei, latissimi dorsi, and supinatores longi. The other muscles were well developed, and contracted very powerfully when galvanized. M. R—— could not raise his arms above the horizontal line, and even then he had to make very great efforts. During that movement the spinal border of the scapula moved away from the thorax, whilst its inferior angle got nearer the median line. In that position the scapula formed a triangle, the apex of which was at the inner angle of the bone, and the base was formed by its axillary border. The atrophy of the other muscles did not seem to interfere much with his movements. Lastly, fibrillary contraction was not perceptible anywhere, and the patient declared that he never felt any quivering of his muscles. His general health was excellent. On merely looking at M. R——, one would not indeed suspect that atrophy had already destroyed many of his muscles. His chest is plump and well developed; the posterior aspect of his trunk looks normal and well nourished, when his arms hang alongside of it; and yet, on testing his muscles by galvanism, the trapezii, rhomboidei, and latissimi dorsi are found to be missing. As these muscles are only of secondary importance, their atrophy would not be suspected if the serrati magni were not involved. M. R—— was surprised to hear, therefore, that all these muscles had degenerated, and especially that the supinatores longi were destroyed, while his arms possessed vigorous muscles. He knew that he was seized with the complaint which was hereditary in his family, from the difficulty which he had from the beginning in raising his arms, owing to the absence of the serrati magni. The special deformity, which is observed when the arm is raised, from the want of co-operation of the serratus magnus, and which is pathognomonic of paralysis of that muscle, is the only sign which at first sight points to the existence of muscular degeneration.

Cases of this kind are too exceptional to detract from the value of alteration of shape as a pathognomonic sign of progressive muscular atrophy. In proportion as the disease makes progress, this character becomes more and more marked, and there comes a time when the skeleton-like aspect of the greater portion of the body contrasts with the plumpness of the face.

The disease derives a still more special physiognomy from the changes which occur in the attitude of the limbs and the trunk in the state of rest, and from the disorders of locomotion during the performance of voluntary movements. These phenomena have been accurately studied and analyzed by the author of the treatise on Localized Electrization:

"The attitude of the limbs during muscular rest depends on the tonicity of the muscles which move them. But there is no muscle which is not antagonized by another. If, therefore, one of the antagonistic muscles happens to be weakened or destroyed by atrophy, the equilibrium of the tonic forces, on which depends the normal attitude of the limbs, is disturbed, and the limbs are necessarily drawn in the line of the predominating tonic force, namely, of the predominating muscle or muscular bundle. If the mechanism of these faulty attitudes be well understood (and this may be done by studying the separate action of individual muscles and even of muscular bundles), it will be easy to deduce from it the kind of signs which constitute the principal characters of partial atrophies."*

The functional disorders, which occur during the performance of voluntary movements, affect, some of them, the movement special to a muscle or a portion of a muscle, and others the co-ordination of the diseased muscles; for every movement, in order to be regular, not only requires that one or several muscles should contract, but also that other muscles, which do not directly concur in the production of the principal movement, should come into play, in order to steady, regulate, and moderate that movement. The patients can supply the place of some of the muscles which they have lost, by instinctively contracting their congeners: as when, for instance, in the absence of the biceps, the forearm is flexed by means of the muscles which are inserted into the epitrochlea, and especially by means of the pronator radii teres. These supplementary movements are very irregular, it is true; but there are muscles which have no homologues, and then the movements which they used to perform become impossible; and if the patient attempts to execute them perfectly, contrary movements will result, through the antagonistic muscles contracting by themselves. I cannot too strongly recommend to you, gentlemen, to read in Dr. Duchenne's treatise the interesting details into which he has entered on this point, and which it would take me too much time to go into here.

However advanced the destruction of the atrophied muscles, and however general the disease may be, phenomena indicating a general disturbance of the system are absolutely wanting. The appetite remains good, and digestion is perfectly regular. Yet, when the muscles of mastication and deglutition become in their turn involved, these acts are more or less impeded in consequence. The lower jaw is depressed with an effort only (for it is the depressors of the jaw which are in general affected), and there may even come a time when, owing to the complete destruction of these muscles, the mouth cannot be opened, and the patient has extreme difficulty in feeding himself. Deglutition is generally then accomplished with difficulty, and attended with a copious flow of saliva. I need not tell you what a grave complication this will be in the patient's condition, in proportion to the degree of interference with the mechanism of deglutition. The amount of food taken being insufficient, the patient may die from gradual starvation.

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* "De l'Electrisation localisée, et de son Application à la Pathologie et à la Thérapeutique." 2ᵉ édition, Paris, 1861, p. 468.

As the rectum and bladder are never affected, the fæces and urine are normally expelled, at will and with perfect freedom, unless the abdominal muscles should be atrophied, in which case their expulsion is attended with difficulty.

The breathing is regular, until the time when the respiratory muscles are involved, but this generally occurs in a pretty advanced stage of the disease. When the diaphragm is affected, phonation is impaired, and becomes considerably more so when the expiratory muscles atrophy. Were these to disappear completely, simultaneously with the diaphragm, asphyxia would result from the complete inability to breathe; but before the lesions reach that degree, they can cause the most serious complications, in the shape of intercurrent diseases of the respiratory apparatus, and thus prove an indirect cause of death. If the patient be attacked with bronchitis, for example, the mucus secreted in the bronchial ramifications can no longer be expelled, and their accumulation in those tubes will bring on asphyxia more or less rapidly.

A patient whom you may have seen at No. 23 in St. Agnes Ward, died in this way. He was a cabinet-designer, and 46 years old. Three years and a half ago he first noticed that the strength of his left shoulder failed him, and that it was less easily moved. He felt, at the same time, in his deltoid, creeping and quivering sensations, which were the fibrillary movements characteristic of incipient muscular atrophy. The shoulder shortly became thinner, and then perfectly identical symptoms showed themselves in succession in the biceps, in most of the muscles of the forearm, and in those of the thenar and hypothenar eminences. Soon afterwards the right shoulder, arm, forearm, and hand were similarly affected. Note that in this case the disease spread from above downwards, from the shoulder to the hand, whilst the reverse usually obtains.

At the end of eighteen months the patient had to give up his occupation, and for the last two years he had only been engaged as a messenger, when he decided on being admitted into one of my wards, on October 2, 1863. You could then see that both his shoulders were atrophied, and that his humeri, being no longer supported by the deltoid muscles, dropped from the glenoid cavity of the scapula. His upper limbs hung alongside of his trunk, and when he wanted to feed himself, he knelt by a small table, rested on it his forearms (which were by that means flexed, in the absence of his wasted biceps), and took hold, with both his hands, of the pieces which he wanted to carry to his mouth, making the best use he could of the muscles or of the remnants of muscles which he still possessed. His chest-walls were fleshless, and it was evident that his pectoral and intercostal muscles were wasting away. The muscles of his abdomen and lower limbs were intact.

All the functions of vegetative life were perfectly performed; yet I told you beforehand, that the imminent danger which this patient ran was from the possibility of his having a chest affection, for he had an habitual small dry cough. On January 16, he became suddenly feverish, had difficulty of breathing, and died two days afterwards from this dyspnœa, which I vainly tried to combat. I subsequently found general congestion of, and miliary granulations in, both his lungs. You saw the pathological specimens which my clinical assistant, Dr. Peter, had prepared. The deltoid muscles, the biceps, coraco-brachiales, the superficial and deep flexors of the fingers, &c., the muscles of the thenar and hypothenar eminences, the interossei and lumbricales, were more or less considerably atrophied; whilst the triceps brachialis, the palmaris, and anconeus were untouched by disease, and their normal volume and their red color contrasted with the slenderness and the yellowish discoloration of the wasted muscles. The pectorales minores and majores were partially atrophied, and the intercostales very considerably so.

The degree of atrophy did not merely vary in the limbs, but also in various portions of the same muscle. Thus the muscles of the left arm were markedly more atrophied than their homologues on the opposite side, and the posterior bundles of the deltoids were normal, whilst their anterior and middle bundles were deeply altered.

You could ascertain, by comparing them with those of a healthy cord, how far the anterior roots of the cervical and dorsal nerves had atrophied, whilst the lumbar nerves and those of the cauda equina were of normal size. The roots of the cervical nerves were those especially affected, and more markedly so on the left side. Some of them, particularly those which go to form the brachial plexus, were reduced to one or two extremely fine threads. The circumflex, median, ulnar, and radial nerves were of somewhat diminutive size.

I was anxious that not only the structure of the anterior roots and of the nerves should be carefully examined, but also that the condition of the capillary bloodvessels distributed to the atrophied muscles should be ascertained. Now, Dr. Peter found that the nerve-tubes of the most diseased roots and nerves had diminished in number and size, and that the diminution in size was due to a considerable reduction in the amount of nerve-substance. The nerve-substance of some tubes had become finely granular, while it had completely disappeared from others, as well as the cylinder-axis; and there remaining nothing but the sheath, the tube had a remarkably constricted appearance.

(In the examination, a comparison was instituted between the above tubes and those of healthy roots and nerves.)

The changes of the nerve-elements consisted, therefore, in a diminution, a granulation, or a complete disappearance of the nerve-substance, with retention of the neurilemma. We shall presently see that these changes are perfectly analogous to those of the muscles. No appreciable lesion of the capillary vessels could be discovered.

As to the muscular fibres, they presented various degrees of alteration. They had diminished in size, and while, in some fibres, the transverse striæ had merely become more rare, and fat-globules were seen in pretty large numbers, in other fibres the striæ had completely disappeared, and in their stead were only seen very fine granulations. Hence, then, a microscopical examination revealed that the sarcolemma of the muscular fibres was preserved as the neurilemma of the nerves was, and that there was a diminution or a granular degeneration of the proper substance of both tissues, the muscular and the nervous. If this remarkable autopsy does not admit of our recognizing whether the lesion began in the nerve or the muscle, it authorizes us, at least, to affirm that the lesions were parallel and identical in both these tissues.

When I spoke of the pathological anatomy of progressive muscular atrophy, I reminded you that the absence of all kind of lesion of the nerve-centres was in accordance with the absence of nervous disorders during life. Scarcely is there, in some cases, cutaneous anæsthesia of the regions corresponding to the degenerated muscles, and this is the only nervous symptom ever observed. Everywhere else the integuments retain their sensibility; free from exaltation or diminution. The organs of the senses are in nowise disturbed in their functions. Lastly, intelligence remains entire unto the end; and it is as curious as it is sad to see poor unfortunates—who are, as it were, reduced to the condition of skeletons, through the loss of a great portion of their muscles—not only perform all the functions of organic life, but possess a perfectly lucid mind, and thus witness themselves their slow and progressive destruction.

I remember, gentlemen, the history of a lady at Tours, whom the illustrious Bretonneau attended for several years, and who, according to the description given me by my old teacher, died in the last stage of muscular atrophy. She could scarcely breathe, and no longer spoke; but her eyes still retained all their vivacity, and reflected her intelligence, which did not forsake her. She could still contract some of the muscles which support the head, and those of the index-finger of her right hand. During the last days of her existence, she conversed with her children by means of this finger. Several sets of alphabetical letters, like counters, had been got for her, and with her finger she put the letters together, composing words and sentences. By that means she was enabled to make her will.

The epithet *progressive*, applied to the word *atrophy*, sufficiently indicates the course of the disease. If it be not rare to see it remain stationary, after having affected one or more regions, too often, after a more or less prolonged pause, it spreads to other parts; and as I have already told you of the manner in which it progresses, in the rapid sketch which I gave you of the symptoms of the disease, I need not revert to it now. If we only look at what occurs in one or several muscles taken singly, the course of the disease is rapid, for the tissues may be completely destroyed in a few months; but if we look at the disease as a whole, and calculate the time which has elapsed from the period when the first symptoms showed themselves until the fatal termination, the duration of progressive muscular atrophy is generally long. If in some cases, as Dr. Duchenne (de Boulogne) has had occasion to observe, in less than two years a good many muscles of the upper limbs and the trunk, some of those of the lower extremities, the muscles of the face, those of respiration and deglutition, have been transformed in various degrees, the disease usually progresses much more slowly, although its duration is never very determinate.

On this point, therefore, we have no precise data for prognosis. But we know, unfortunately too well, that the prognosis is invariably most serious. Death, indeed, may be the consequence of these grave disorders of the locomotor apparatus. It may be directly brought on when the atrophy invades the muscles of deglutition and respiration, and the patient, as I told you, ultimately dies of starvation or by asphyxia; or it may be caused indirectly when, as I have already told you, there supervenes an intercurrent affection, bronchitis for instance, to the evolution of which the muscular atrophy adds a fatal complication. But even supposing that this fatal termination occurs as late as possible, and that, from the disease remaining localized, the patient suffers for many long years, the prognosis is not the less unfavorable on this account. The disease may, indeed, pause in its course, it may be arrested in its progress, but we cannot hope to see it retrograde, for the muscles that have been destroyed are ruined forever. You may imagine, gentlemen, the infirm condition to which the unfortunate patient, thus deprived of a greater or less portion of his muscular system, is condemned—a condition which is all the more cruel, that progressive muscular atrophy much more frequently attacks individuals in the strength of years, or belonging to the working-classes, and who need all the freedom of their movements in order to earn their living and that of their family.

With regard to prognosis, there is another peculiarity mentioned by Dr. Duchenne (de Boulogne), and which to me also appears of great importance—namely, that the disease becomes general, and terminates fatally, all the more quickly that no appreciable determining cause has brought it on. It then seems that the diathesis, of which the muscular lesion is only an expression, is much more active than when the disease has been excited by a determining cause. Lastly (and this is also a remark made by Dr. Duchenne), when the disease primarily attacks the muscles of the trunk, it remains stationary much longer, and spreads to other regions more slowly, than when it sets in first in the muscles of the limbs.

A disease which has such a characteristic physiognomy cannot be mistaken by a careful physician who has once seen it. There are cases, however, in which the *diagnosis* is attended with some difficulty. When, for instance, a considerable degree of plumpness prevents the production of the characteristic deformities, the disease may at first sight be unobserved, although from the disorders of locomotion it can be easily recognized.

Rheumatic pains are sometimes followed by muscular atrophy, which it is important not to confound with progressive atrophy, for the two affections are essentially different. The course and duration of the symptoms, the forms which they assume, and the seat which they select, suffice, before having recourse to the test of electricity, to guard one from error. Rheumatic atrophy is, indeed, preceded by more or less violent pains, which are intensified by the voluntary contraction of the affected muscles, and by pressure made on them, whilst progressive atrophy is generally painless. Besides, the whole muscular mass is involved, and not a few bundles only, as in progressive atrophy. Lastly, whilst in the latter electric contractility is considerably weakened in proportion to the number of muscular fibres destroyed, in rheumatic atrophy, on the contrary, whatever be its stage, and however restrained and impossible even voluntary movements may have become, galvanization is still all-powerful, for the muscle has merely diminished in size, and its fibres have undergone no transformation.

The differential diagnosis between lead-palsy and progressive muscular atrophy is attended with greater difficulty. In the former of these affections, however, electro-muscular contractility is completely abolished, whereas in the latter it is only diminished in proportion to the number of muscular bundles which have undergone the characteristic alteration. Moreover, even when saturnine paralysis has become general, the electric contractility of certain muscles alone is destroyed—namely, of the extensors of the hand on the forearm, and subsequently of the muscles of the arm, chiefly the biceps and deltoid. Lastly, the evolution of the symptoms (for in the lead-disease palsy precedes atrophy), the previous history of the patient showing that he has been exposed to the influence of lead, and other morbid phenomena special to saturnine poisoning, will throw light on the question.

The disease which most closely resembles progressive muscular atrophy is the atrophic paralysis of infants, but the latter differs from the former in being complicated by an arrest of development of the bones of the limb whose muscles have undergone fatty degeneration.

It is important also to distinguish from progressive atrophy, the wasting which is due to an injury to a nerve or the branch of a nerve. The wasting is, in such cases, exclusively limited to the groups of muscles supplied by the nerve or the branch of a nerve, and the complete limitation of the disease must immediately suggest the idea of an equally circumscribed lesion, and do away with that of progressive muscular atrophy, in which the disease settles here and there, at random as it were, and not according to the anatomical distribution of a nerve. There is even now, in the St. Jane Ward in this hospital, a man suffering from wasting of the left hand and forearm, and whose tongue is also atrophied in its left half. But it is easily recognized that the wasting involves the deep flexor of the fingers, the interossei, the two inner lumbricales, and the deep adductor of the thumb—that is to say, muscles supplied by the ulnar nerve. Now this man has had syphilis, and has now an exostosis on a level with the epitrochlea, which, by compressing the ulnar nerve, has probably altered its structure, and then caused wasting of the muscles which it supplies. It is pretty probable that another exostosis, depending on the same cause, compresses either the lingual nerve or the hypoglossal, and causes, in the muscles of the tongue, alterations of nutrition similar to those of the muscles of the arm.

Lastly, there is a disease of which few instances will come under your notice, which also causes muscular atrophy—namely, the *dry leprosy* of hot climates. But in this case also, the wasting is circumscribed, and limited to all the muscles of the hand. The skin of the hand is red, thickened, and completely anæsthetic; the fleshy masses have entirely disappeared, and the fingers assume the shape of claws. Such were the symptoms noted in a woman whose case M. Peter had occasion to study for a long time, whilst he was Dr. Cruveilhier's resident assistant. The differential diagnosis is easy, for apart from the leprosy, which is easily recognized, and from the complete anæsthesia which accompanies it, the flexor muscles of the fingers are found to be retracted, a fact on which Dr. Duchenne justly lays particular stress.

I told you, a moment ago, that progressive muscular atrophy never terminated in death so rapidly as when it was developed without any appreciable determining cause. The most frequent of such causes is continuous and excessive work, necessitating the exercise and exaggerated contraction of certain muscles. But determining causes are subordinate to an individual predisposition, to a diathesis which is more generally acquired, but is often also transmitted from one generation to another.

This influence of hereditary predisposition, or at the least of consanguinity, was, in 1851, pointed out by Dr. Meryon, who, in a communication to the Medico-Chirurgical Society of London, related the history of a family, in which three boys suffered from muscular atrophy. I have already mentioned to you similar instances, observed by Dr. Duchenne. The most remarkable point in Dr. Meryon's case is, that of the nine children composing the family, the three sons were alone affected, whilst the six daughters escaped.

It is, besides, an observed fact that this disease rarely attacks women, and that in them it does not become general, no instance of its doing so having been recorded as yet. When I have added that muscular atrophy is scarcely ever met with but in adults, I shall have told you the little that is known concerning its etiology.

Treatment is, unfortunately, powerless against this complaint; and if localized faradization has, in some cases, been able to arrest its development, the disease has made further progress after a more or less prolonged intermission.

Before dismissing the subject of progressive muscular atrophy, I wish, gentlemen, to discuss with you an important question, namely: whether this singular disease is an affection of the spinal cord, or whether it should be grouped with diseases of the muscular system. Those who ascribe the atrophy to a spinal lesion, ground their opinion on the results of post-mortem examinations, which have shown a singular diminution in size of the anterior roots of the spinal nerves. This pathological change is perfectly evident in the specimens which I now show you. M. Sappey has had the extreme kindness to prepare, with the greatest care, the spinal cord of my patient, and has placed by the side of it several cords taken from the bodies of individuals who had not suffered from any nervous disorder. You can see how great the difference is between them. It is supposed, then, that this lesion of the interior roots is the starting-point of the functional disorders, and that the muscles, no longer receiving any nerve-force as usual, first atrophy, and next become altered in their structure. They atrophy in the same manner as muscles do in cases of saturnine or rheumatic paralysis.

This view of progressive muscular atrophy is at first sight seductive, but it cannot be accepted when the question is closely examined.

First of all, the anatomical fact, namely, the atrophy of the anterior roots, proves nothing as to the priority of the nerve-lesion. It is known that if the brachial plexus, and the nerve-roots which concur in its formation be dissected in an individual whose arm has been amputated, the nerves are found to be atrophied as far as their entrance into the fissure of the spinal cord. In individuals who have lost an eye, not from cerebral disease, but in consequence of ophthalmia or of a wound, the optic nerve is invariably found to be atrophied as far as the commissure. The peripheral lesion and the cessation of function may, therefore, be the cause of the atrophy of the nerves; and it may be reasonably maintained that the atrophy of the anterior roots, found after death, in nowise decides the question at issue. But I pass on to other arguments. If the starting-point of progressive muscular atrophy were in the spinal cord, we should always see the disease affect a whole group of muscles, and not a few only.

Thus, for example, all the muscles which derive their nerves from the brachial plexus would be involved simultaneously and to the same degree; or, at the very least, all the muscles supplied by nerves from one of the cords of this plexus would atrophy and degenerate at the same time. But such is not the case. In one patient we see a perfectly isolated muscle, in the hand for example (and this is the most frequent case), atrophy completely, and lose its structure and functions, whilst other muscles, supplied by the same nerve-branch, are untouched. We see at the same time one or more muscles of the arm, trunk, and lower limbs, on either side indifferently, becoming affected in turn—a circumstance which is never observed in those diseases in which the primary lesion of the nerve-centres and the nerve-cords cannot be called in question.

I confess, gentlemen, that a careful study of the course of this disease does not allow me to group it with diseases of the spinal cord, in spite of the autopsies made by a celebrated anatomist, Professor Virchow, who declares that he has seen, with the aid of the microscope, alterations in the anterior columns of the cord itself. I dare not, on the other hand, adopt a perfectly opposite opinion, and say that progressive muscular atrophy is primarily a disease of the muscles. It is infinitely probable that the nerves of organic life which accompany the terminal and muscular branches of the arterial tree, are so modified in their functions as deeply to disturb the nutritive phenomena over which they preside; but when we find grave organic lesions and degenerations of tissue in a kidney or in the liver, although in every case, perhaps, the vaso-motor nerves may be at fault, we do not the less say that the lesion is renal or hepatic, if the supposed nervous disorder exist only in the kidney or liver. Until more is known on the subject, we may justly regard progressive muscular atrophy as a disease of the muscular system, if we wish to conform ourselves to the usual view taken of anatomical lesions; although this does not prevent us from admitting the existence of a diathesis which influences the whole system, and predisposes it to the strange lesions which we have just studied.

LECTURE LXI.

ON APHASIA *

There is not only loss of Speech, but loss of Memory also: 1. Amnesia of Speech; 2. Amnesia of Speech and Writing; 3. Amnesia of Speech, Writing, and Gesture; hence three principal forms of Aphasia.—Transitory Aphasia and Persistent Aphasia.—Special Anatomical Lesions of Aphasia.—Function of the Posterior Portion of the third left Frontal Convolution.—Unique Case in which the Seat of the Lesion was probably on the right side.—More or less profound, and undeniable Disturbance of the Intellect in Aphasia.

GENTLEMEN: Some of you may recollect a young mechanic, about twenty-five years old, who occupied bed No. 2 in St. Agnes Ward. He had walked to the hospital, he was not lame, he used both his hands perfectly, his face was full of intelligence, and yet he was not able to answer any of my questions, although his tongue was very mobile. He heard me well, and looked at me whilst I questioned him; his gestures, his looks, showed that he understood all I said; it seemed as if his mind were full of thoughts which he could not express in words. He knew how to read and write, and yet when I gave him a pencil and some paper, and asked him to write his name down, he held the pencil properly but only wrote meaningless letters, and then threw away the pencil in a fit of impatience. He, however, remembered a few words, which he kept constantly repeating, showing at the same time by his manner, that he well knew how little those words expressed his meaning. His illness had set in suddenly, after certain excesses.

It was evident that some local changes had occurred in the structure of his brain, which were certainly not due to hemorrhage or softening; and as there was no headache and no fever, I waited until more light could be thrown on the case. But before a fortnight had elapsed the young man recovered completely, without having been subjected to any treatment, and was able to leave the hospital. Every day a new word was added to his vocabulary; then incomplete or incoherent sentences were succeeded by very connected ones, and at last he managed to hold a conversation, although he from time to time felt some hesitation, and was sometimes unable to find the exact word which he wanted to express his thought. By the time that he left the hospital, all abnormal symptoms had disappeared. He had previously been able to give a pretty good account of what had passed in his mind. He well knew that he had lost all recollection of words, and he was besides conscious that his mind was not then so clear as it had been.

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* The affection which I am about to describe was, in 1841, termed *alalia* by Professor Lordat: and in 1861, Mr. Broca changed this name for that of *aphemia* But Mr. Chrysaphis, a very distinguished Greek scholar, and a Greek himself, although accepting the term *alalia*, proposed, however, as a better one that of *aphasia*, derived from α, privative and φασις, speech. Mr. Littré, whose authority is so great, and Dr. Briau have likewise preferred the word *aphasia*, and all three concur in rejecting *aphemia*. I had at first adopted the name of *aphemia*, after Mr. Broca, but I have now, on the authority of the savants whom I have mentioned, substituted for it that of *aphasia*.

On the 27th of February, 1861, the woman Desteben, aged fifty-eight, was admitted into the St. Bernard Ward. After she had left the hospital, she was admitted on the 12th of April following into the Salpêtrière, where she died on the 16th of April, 1863, of what is called *cerebral congestion* in the register, but no post-mortem examination was made. During the forty-four days which she spent at the Hôtel-Dieu, this woman was most carefully watched, and I remained every day a pretty long time by her side. I learnt that on several occasions, after slight apoplectiform seizures, she had experienced considerable difficulty in speaking, although she was not in any way paralyzed. When I saw her, the movements of her limbs were perfectly free; she moved her tongue about with as much facility as any other person, but she could not articulate anything besides, "Oh! how annoying!" It was thus she expressed her impatience on finding her inability to answer my questions. Although she looked intelligent, and behaved in the ward like a sensible person, I could never obtain another word from her. She knew perfectly the use of the objects which I pointed out to her, but she was never able to tell their name; and if an attempt was made to deceive her, by calling the object by a wrong name, she protested very clearly by her gestures, whilst she on the contrary made signs of approval, when the true name was mentioned. As she did not know how to write, it was impossible to ascertain the manifestations of the intellect which might have been revealed in writing.

This patient was remarkably clean in her habits: she made her own bed, combed her hair, and dressed herself with a certain amount of vanity, put away carefully all her toilet articles, and never exhibited, during her residence in the hospital, the least hesitation in her movements, which always remained perfectly regular.

The next case which I shall relate to you is all the more precious, from the patient being one of my most eminent colleagues in the Faculty of Medicine, and from his having paid special attention to the study of cerebral diseases. Mr. X—— had been confined to his bed for a few days in the country in consequence of an injury to one of his legs, and being alone there, he read all day, and thus fatigued his brain. He was engaged one day reading one of Lamartine's literary conversations, when he suddenly noticed that he did not clearly understand what he was reading. He paused for a short time, but, on beginning again, he made the same remark. Alarmed by this, he tried to call to his help, but to his extreme surprise he was unable to utter a word. Believing himself to be struck with apoplexy, he then executed various complex movements with both his hands and his uninjured leg, and thus found that he was not in the least paralyzed. He then rang the bell, and when a servant came into the room, Mr. X—— could not speak a single word; and yet he could move his tongue in every possible direction, and was perfectly aware of this singular discrepancy between the facility with which his vocal organs were moved and his inability to express his thoughts in words. He made signs that he wished to write, but on pen and ink being brought to him, he was as incapable of expressing his thoughts in writing as in speaking. As he had always paid particular attention to diseases of the brain, he tried, however, to analyze his symptoms, and to explain them by some special lesion of the brain, reasoning mentally on his own illness, as he would have done in a clinical conference. His servant immediately telegraphed to Mrs. X—— who was in Paris, and sent for a medical man, who came two or three hours afterwards. Mr. X—— then pulled up his shirt sleeve, and pointing to the bend of his arm, clearly intimated that he wished to be bled. No sooner was the bleeding over, than he was able to say a few words, although incoherently and imperfectly. Still some of these words clearly expressed an idea; whilst others seemed to have no direct relation to the principal idea. By degrees the veil was removed; he had a greater command of words to express his ideas which also became more numerous, and at the end of twelve hours recovery was complete. Let me here observe, and I shall revert to it by and by, that Mr. X—— had been suffering from diabetes for a few years.

The next case, which very much resembles the preceding, is that of a gentleman suffering from Bright's disease. He was sixty years old, and was sent to me on June 18, 1863, by Dr. Denoette (of Havre). He was a terrible sufferer from gout, and notwithstanding his doctor's advice to the contrary, had drunk to excess of Vichy water. He had therefore had internal gout, and had become extremely cachectic; for some years, he had passed albuminous urine. In 1861, he had a nervous attack, the details of which he related to me, and which were confirmed by his wife. He was playing whist at his club, and in the middle of a game, threw his cards on the table. On his then attempting to speak, he was unable to utter a single word, and yet until that moment he had noticed nothing strange, and had played with his usual skill. Feeling alarmed, however, he gets up, takes his hat, his walking-stick, and walks home quickly, more quickly even than usual. On arriving home, he tried to tell his wife what had occurred. By that time he could say a few words, parts of sentences even, but omitting words which he could not find, and getting impatient in consequence. The difficulty which he had in expressing his thoughts increased every moment, however, and before two hours had elapsed, he was incapable of articulating a single word. Yet his limbs and his tongue could be moved as freely as in health.

The usual medical attendant of the family, who arrived by this time, prescribed leeches round the anus, and whilst the servant was gone for them, the patient's wife wished to find out whether he could read. But he could not read the newspaper which she held before him, although his sight was perfect. On my questioning him on this point, he said that he could see the letters and the words, but did not understand their meaning well. The leeches were next applied, but he seemed to grow very impatient because they took slowly and with difficulty. He tried to explain why he was impatient, but his gestures, although normal, were not understood. Speech was completely gone. Three of the leeches, however, had scarcely begun to swell, when speech returned a little; the patient could then make himself understood, but still left out a few words in each sentence. He asked for better leeches, and when fresh ones were applied and drew blood in abundance, all the strange symptoms vanished, and the patient expressed his thoughts with as much facility as ever. He then related all the circumstances of his seizure, and explained that he was so impatient when the first leeches were applied, because he wished for better ones, and was annoyed at finding that his gestures were not understood. Since that time he has had no second attack of the kind, and the Bright's disease from which he suffers has continued, without presenting any abnormal symptoms.

The two following cases have been communicated to me by my friend Dr. Voyet, of Chartres.

M. X——, a veterinary surgeon of X. (Eure et Loire) aged forty-six, of robust constitution, single; no previous illness with the exception of a cancer of the under lip, successfully removed by operation in 1863. About the end of September, acute articular rheumatism. On the 29th of October, 1863, intense dyspnœa in the middle of the night; I was sent for, and saw the patient for the first time on the morning of the 29th of October. During the paroxysm of dyspnœa, the rheumatic pains disappeared, the pulse was intermittent, and so irregular that it could not be counted; there was the same disturbance in the heart's action, but no abnormal bruit; the patient's anxiety was such that he could not remain in bed. Sinapisms and a blister applied over the precordial region gave immediate relief; upon which the rheumatic pains returned, and the heart's action became regular again. On November the 4th, M. X—— was sitting by the fire and talking to a friend, when he suddenly stared at his friend with a stupid look, without being able to utter a single word. About five minutes afterwards he began to sputter out the word *monomentif*, which he kept repeating for four hours. Annoyed at not being able to make himself understood, he made signs that he wanted pen and ink, but only traced on the paper shapeless signs, like a child who does not know how to write. Four hours afterwards, on his renewing the attempt, as he wished to inform his brother of his condition, he only succeeded in writing legibly, "My dear;" the rest was as shapeless as on the former occasion. By this time, however, he began to articulate a few monosyllables, but always ended them by *tif;* and if he wished to say a word of several syllables, he only pronounced the first syllable, and added *tif* to it, saying, for example, montif for monsieur, bontif for bonjour, &c.

On the following day he could answer questions that were put to him, but could not construct a sentence: yet he could be understood. On the second day all abnormal symptoms had disappeared, whilst, during the forty-eight hours that the aphasia had lasted, there had been no trace of paralysis.

On the 12th of February, 1864, that is, more than three months after the above attack, M. X——, who still complained of rheumatic pain in the right wrist, was eating his soup, holding his spoon in his left hand, when he suddenly dropped it, and his arm became completely powerless. Dr. Voyet, who was immediately sent for, found, on his arrival, that there was paralysis of the whole left side of the body, together with great embarrassment of articulation and deglutition. By the next morning all the symptoms had disappeared. On this occasion, the difficulty in speaking was not in the least like what had been felt in November; at that time, M. X—— could not find words to express his thoughts, whereas now he found words, but had a difficulty in articulating them.

On February the 12th, 1864, Dr. Voyet saw in consultation Mrs. X——, aged 58, residing at Voves, and for many years afflicted with hypertrophy of the heart. He was told by her daughter, that in the beginning of February, she was awakened by an unusual noise proceeding from her mother's room, and, on her going there, she found her mother in the act of gesticulating and repeating constantly, *vousi, vousi.* After two hours this condition disappeared, and Mrs. X—— then related that she wanted to ask for ether, and to say that a doctor should be sent for, because she felt that something extraordinary was passing within her.

Another curious case is the following:

Mrs. B——, the mother-in-law of a medical man, had never been paralyzed, but labored, under a very singular intellectual disorder. Whenever a visitor entered her apartment, she rose with an amiable look, and pointing to a chair, exclaimed: "Pig, animal, stupid fool." ("Mrs. B. asks you to take a chair," her son-in-law would then put in, giving this interpretation to her strange expressions.) In other respects, Mrs. B.'s acts were rational, and her case differed from ordinary aphasia in that she did not seem to grow inpatient at what she said, or to understand the meaning of the insulting expressions of which she made use. In the seven cases, which I have just related, the nervous phenomena seem to affect the intellect alone, and especially the aptitude for expressing one's thoughts in writing and speaking. I now pass on to more complex cases, in which there evidently is a deeper lesion of the brain, characterized by some impairment of the motor power, as well as by the peculiar phenomena, to which I have just called your attention.

M. X——, a very eminent jurist, consulted me in the beginning of 1863, and his wife, a remarkably intelligent person, told me of details which the patient perhaps would not or could not have communicated to me. From time to time, in the middle of a conversation, he could not find the word he wanted, or substituted a strange one for it. On other occasions, he would call out to his wife: "Give me my—my—dear me! my—you know;" and he would point to his head. "Your hat?"—"Yes, my hat." Sometimes, again, he would ring the bell before going out, and say to the servant: "Give me my um, umbrel, umbrel, oh dear!"—"Your umbrella?"—"Oh yes, my umbrella." And yet at that very time his conversation was as sensible as ever; he wrote on, read of, or discussed most difficult points of law. He complained, however, of some heaviness of the head, and pretty frequently also of some numbness of the limbs, more marked on the right than on the left side.

Now, gentlemen, let this singular forgetfulness of words be considerably more exaggerated, and you will have aphasia with its usual characters. In the first cases which I related to you, there existed no symptom of paralysis, but in the last one, there is very probably, if not certainly, an organic lesion of the brain. The following case, which was in my own wards, is all the more interesting from the patient being now completely cured, and from her being able to analyze herself all the phenomena which occurred in her case.

On the 1st of April, 1862, Marie K——, aged 50, was admitted into the St. Bernard Ward. A month previously, she had begun to complain of certain symptoms which she afterwards described perfectly. She had violent headaches, followed, from time to time, by convulsive movements of the right half of the face which lasted a very short time only, and left behind them a momentary embarrassment of speech. She had never lost consciousness, and she even added that she had got up during an attack, and had fetched a handkerchief in order to wipe off the frothy saliva which ran out of her lips. Two days before her admission she had had a more violent seizure, during which she had bitten her tongue.

During the first week after her admission, she could only say a few words, devoid of any precise meaning. She looked intelligent, and yet she could neither tell nor write the name of the most commonplace objects, such as a watch, a key, a spoon, or a fork. She could articulate her name well, and write it easily; but if, after having written her name, she was asked to write the word *spoon*, she went on writing her name, and yet noticed her mistake, as could be seen by her look of annoyance. When I insisted on her repeating a word after me, she made signs that there was an impediment in the right side of her throat, and strangely enough, another patient, Adèle

A——, of whom I shall presently speak, complained of a painful tightness in the same spot.

Marie K——* read a good deal during the day, and we were all taken in by this semblance of intelligence, but after her recovery, she told us that she only read with her eyes, not with *her stomach*, a singular expression which she used, meaning thereby that she did not understand what she read. After she had been a few days in the hospital, she got better, and recovered the faculty of speech. She then told us that, in the preceding year, she had been subjected by her medical attendant to a very severe mercurial treatment. I therefore suspected that her peculiar affection was due to some grave syphilitic lesion of the left cerebral hemisphere, or of the base of the brain; and on my prescribing powerful antisyphilitic remedies, I had the satisfaction of seeing the abnormal symptoms disappear, and her health get re-established. She has now left the hospital for two years, but she has often come here to show herself. She has for several months taken, and still takes, from time to time, iodide of potassium.

On the 9th of December, 1854, a young laborer, aged 28, was admitted into St. Agnes Ward. Two days previously, according to the statement of his friends, he had been seized, suddenly and without any assignable cause, with complete mutism. His previous health had been good on the whole, he had led a regular life, and yet he had two years previously suffered from violent headache, and even been delirious. He was bled from the arm, and got well again; and since then, he had not been taken ill in the same way.

The affection, for which he had come to the hospital, consisted solely in an utter inability to speak, although his intelligence seemed to be unimpaired, and he could perfectly understand all the questions which were put to him. But to these questions he invariably answered "*No*," even when he nodded his head to signify assent. One of the students, however, informed me that when left alone with him, he had succeeded in making him say the word "*cloak*," after many repeated trials. I found only a marked deviation of the apex of the tongue to the right, but no other sign of paralysis; the face, the trunk, and limbs, could be moved with perfect freedom and force.

The second day after the patient's admission, I ordered him to be bled, and after this, he seemed to be able to move his tongue more freely than before, but there was the same complete aphasia. When I asked him to write his name down, he did so correctly, but when I told him to write down what had happened to him, he only wrote "*was, was, was.*" He knew perfectly well that this was not what he wanted to write, and annoyed at not being able to express his thoughts, he put down the pen. Two days after this, on my asking him to write down the name of his birthplace, he wrote "*alone, alone, alone*," and did so again when I desired him to write "*good morning*." His impatient gestures, all the while, showed that he was perfectly conscious that he was not writing what he had in his mind. On the following day, he wrote again words that had no sense, such as "*game*" for "*soup*," but he could say, "*Good morning, sir*," speaking, it is true, like a child who is learning to speak. A few days later, he said very distinctly: "*I am pretty well*," and then "*Good morning, sir, I am getting on well*," with a hesitating voice, however, like an habitual stammerer who endeavors not to stutter. When the attempt was renewed to make him write, he only scribbled on the paper series of syllables without any meaning, but he managed to write under dictation: "*I have eaten*." He left the Hôtel-Dieu on the 24th of December, although there had been no appreciable change in his symptoms. He asked himself for his discharge, however, saying very distinctly: "*I wish to go away.*"

In the first cases which I brought under your notice, gentlemen, you saw aphasia set in without paralysis. I next related to you cases in which this affection occurred together with a very slightly marked and transitory paralysis, and persisted, as in the instance of Marie K——, even after all trace of weakness of the arm and leg had disappeared. I will now pass on to cases in which the aphasia was extremely marked, and the paralysis of longer continuance, although still transitory.

I was consulted in the year 1863 by a gentleman, a recorder, aged 49. In the month of January of the previous year, he had got up one morning in his usual state of health, and had afterwards sat for five hours in his office, engaged in his usual occupation and without feeling any abnormal sensation. On getting up, however, he found that his right leg felt a little numb. He walked upstairs to his bedroom by himself, but on his way there, felt that his right arm was becoming affected. He spoke with facility, and dictated to his wife a very sensible letter addressed to the gentleman at the head of his department, as he was afraid that this attack of paralysis would keep him from his duties for a time. A few hours later, although the paralysis had not increased in degree, he could no longer speak. Yet he seemed to recognize and understand everything, but could only say: "*Nasi bousi, nasi bousi,*" repeating these unmeaning words whether he asked or answered a question, or pointed to anything. Eight days afterwards, he recovered completely the faculty of speech; and at the end of a month, the paralysis disappeared. This patient was obese, and troubled with piles; his heart was normal. A few days before his attack, he had felt pain in the back of his neck and the left side of his head, but he was pretty frequently liable to this pain, whenever he did any hard work. Two years previously, he had suddenly felt a kind of electric shock in his left hand, which had since then felt slightly numb. When he came to consult me, accompanied by his wife, who told me the above details, he was in the full enjoyment of his intellectual faculties, walked well, but still wrote with great difficulty. As soon as he took hold of a pen, his arm moved violently away from the trunk, and he could only manage to write, and even then with difficulty, by strapping his arm down. He was, therefore, affected with what has been called *writer's cramp*, or what Dr. Duchenne has designated by the more appropriate name of *functional spasm*. This patient was also slightly deaf on the right side, and complained of a sensation of burning in the skin of the right half of the body.

In some cases, the disease is more lasting, probably from its being due to a deeper and more persistent lesion of the brain. Thus, at No. 8 in St. Agnes Ward, you can now see a patient of the name of Marcou, who is affected with aphasia and *left hemiplegia*. Mark that I say *left hemiplegia*, and this is an important case, because, as far as I am aware, it is the only one as yet recorded, in which the paralysis has not been on the right side. This man is thirty years old. He came to the hospital on foot, but could give no information about himself, nor tell his name, occupation, and address, in the office where the names of patients are entered on a register before they are sent to a ward. His stock of words was restricted to these two: "*My faith!*" and when he was pressed hard, he looked impatient, and uttered the oath, "Cré nom d'un cœur!" A stratagem was thought of in order to find out his name and address. He was told that he could not be admitted into the hospital, and had better go home. The poor fellow understood, and went away. He was followed, and was seen to go to a stone-yard, where he sat down on a stone. The workmen there knew him, said what his name was, and added that he had come to the yard that morning, dragging the left leg a little, and unable to speak. He was then brought back, and admitted into the hospital.

The next morning I easily recognized that he was suffering from aphasia, when I questioned him. I asked him what his name was, and his occupation; he looked at me, and answered: "My faith!" . . . . I insisted, but in spite of his efforts, he only shook his head with an impatient gesture, exclaiming: "Cré nom d'un cœur." As I wished to find out how many words he had at command, I said to him: "Are you from the Haute-Loire?" He repeated like an echo, "Haute-Loire?" "What's your name?" "Haute-Loire." "Your profession?" "Haute-Loire." "But your name is Marcou?" "Yes, sir." "You are sure it is Marcou?" "Yes." "What department do you come from?" "Marcou." "No; that's your name." But with an impatient gesture, he exclaimed, "Cre nom d'un cœur." His mouth evidently deviates a little to the right, owing to the paralysis of the left side of his face. When he is pressed to say what he complains of, he lifts up both his arms at the same time, but whilst he moves his right arm briskly and closes his right fist with force, he looks sadly at his left arm, which is relatively powerless, although he can still use it pretty well. When he is in bed, or even when he walks, a certain degree of attention is required in order to recognize that he is paralyzed on the left side. I dare not say that he looks as intelligent as he may have done formerly, but he has not the dulness of aspect which sadly strikes us in persons who have had cerebral hemorrhage. It is very remarkable, also, that aphasic individuals (even when completely hemiplegic) do not shed tears like those who have had apoplexy.

I cannot say what influence has been at work to bring on this man's complaint; but on examining his organs, I thought I could recognize traces of an indurated chancre cured long ago. I accordingly treated him by mercury and iodide of potossium; and after alternating periods of very marked improvement and momentary aggravation of the disease, there is at last a permanent improvement, which, however, is not a complete recovery. The poor fellow, in spite of three months' lessons and efforts, can never remember the word *hair*, and can only say *cotton* when he wants his cotton cap, although he says the word *cotton* with evident complacency.

At No. 20, in the same ward, is a man forty years of age. He has been pretty well educated, since he was at one time destined for the clerical profession, and was in a seminary, so that it will be easier in his case to study the impairment of the intellectual faculties, and to appreciate its various manifestations. He is a married man, and a father, but he is far from having led a regular life. He is particularly addicted to drunkenness. Four mouths ago, after having complained of headache, which could be reasonably attributed to his intemperate habits, he had a fall in his bedroom; but his wife, who was accustomed to see him fall down after too copious libations, took no notice of it. That night she went, as usual, to bed with one of her children, but was awakened from her sleep by the noise of her husband falling down a second time, through his having got his foot entangled under a wardrobe. He got up without saying a word, his wife asking him no questions, went to the bed in which he usually slept by himself, and all through the night was violently agitated, as he used after all to be, whenever he came home drunk. In the early morning, however (it was summer-time), his wife, on going to his bed, discovered that the bottom of the bed was broken, whilst he was lying almost completely naked, and messed all over. His face had not its usual expression, and when his wife rebuked him, he looked at her in a strange manner, repeating, "*Cou si si, cousisi.*" These are the only words which he has spoken for the last four months, and he keeps saying them at every turn, when he is in a passion or when he wishes to express his gratitude, when he asks for or refuses anything. When he is very excited, however, he calls out, "Sacon, Sacon," probably an abbreviation of the oath, "Sacré nom de Dieu." When the poor wife recognized the gravity of her husband's condition, she tried to help him, and then discovered that he was paralyzed on the right side. Upon this, she brought him to the hospital.

You have seen him to-day, and you have found that he is less paralyzed than he was, for he easily moves his right arm and leg, although he cannot at all perform those movements of the hand which require a certain degree of precision, as the act of writing, for example. He can write with his left hand, however, and we shall thus be able to appreciate the state of his intellect. When I ask him his name, he answers, "*Cousisi;*" but when I ask him to write it, he writes down, "*Paquet.*" If then I wish him to write his address, he again writes, "*Paquet.*" Yet he perceives that he has made a mistake, and turns his head impatiently away, saying, "*Cousisi.*" He can write the word "*note*" when a printed copy of the word is set before him; but if this be then removed, and he be asked to write his name, he writes down "*note.*" As he had nodded assent when he was asked whether he could play backgammon and dominoes, several patients in the same ward were requested to play with him in turn, and they all declared that he played well, that he knew all the tricks of the game, and that he even cheated when he found that he was losing. Good luck made him laugh, whilst bad luck rendered him fidgety; in either case his gestures were very significant, or he kept repeating "Cousisi."

His wife, who had come to give me any information which I might desire, had brought with her her boy who had disease of the knee-joint. Whilst I was examining the joint, the boy's father made sign to him by repeatedly touching his own forehead with his hand to remove his cap, which he had kept on his head, and the man looked vexed at this want of deference on the part of his son. I lay great stress on all these details, because they will be useful when we come to examine how far the intellect is preserved in such cases. There is another detail, however, which I must not omit. This patient has several newspapers, containing tales, which he reads, and expresses by signs that he understands them perfectly; but his neighbor states that he reads them over again every day, and several times even on the same day. He surely could find no interest in them if he understood them well, or did not forget them. I have already told you, and I attach importance to the fact, that the hemiplegia has diminished, although, according to his wife's account, the manifestations of the intellect have not become developed in the same degree.

I now pass on to a series of cases in which there has been complete and persistent hemiplegia, whilst the aphasia has remained unchanged since the invasion of the disease.

The following case is sufficiently important to warrant my entering into minute details. I was asked to go and see the patient in the department of Landes, in the spring of 1863. His symptoms had been noted with considerable care by his relative, Dr. G. Hameau, of Arcachon, and I saw him in consultation with Drs. Sourouille, De Loustalot, Hameau, and Laffitte.

M. X—— is fifty-seven years old; he is possessed of great wealth, and has lived freely, without, however, committing any excesses. His father died of some chronic chest affection; his mother is still living, and eighty-seven years old. His grandfather, on his mother's side, and his great grandmother, died of apoplexy, the first when seventy, and the second when sixty-five years old. An uncle, on his father's side, died of apoplexy at the age of sixty-five, and an aunt, on his mother's side, died from the same cause, aged fifty-eight; whilst another aunt died recently at the age of eighty-four, in an epileptiform seizure following upon cerebral hemorrhage.

From his early youth M. X—— has been subject to paroxysmal headaches, of great violence and long continuance, recurring two or three times every month.

When he reached the age of forty-five, his headache was replaced by fits of normal gout, for which he went to Vichy, and drank the waters there, but without deriving any benefit from them. The attacks of gout had not been in the least modified, when three or four years after the thermal season at Vichy, M. X—— had one night an attack of vertigo, as he was going to take tea. He was standing at the time, when he suddenly felt giddy and his sight grew misty; he leant against the mantelpiece in order not to lose his equilibrium. This occurrence excited little attention, although there remained after it some unsteadiness of the right hand and a marked difficulty in writing. A few years previously, when M. X—— was only subject to headache, he once had suddenly noticed that his sight had become dim, and this weakness of sight had lasted for a month or two. It is very probable that on that occasion, as on the succeeding one, trifling hemorrhage had occurred in the brain. Two or three years elapsed without any fresh seizure; the gouty pains returned during the winter, whilst the hand recovered very slowly its former power.

A second attack of vertigo then occurred; the patient was at the time sitting on a chair and dressing. The giddiness lasted somewhat longer than on the first occasion, and from that day the right arm became more markedly weak and the tongue slightly embarrassed. This impediment attracted notice whenever M. X—— spoke with animation.

The intellect had failed a little. During the summer of 1857, he went to Ems, and was slightly better for a few months. In 1858, he went to Bagnères-de-Bigorre; in 1858, to Dax; but in December, 1859, his condition became less satisfactory; he had some fever and pain in the epigastrium. On the 2d of February, 1860, at 7 o'clock in the evening, he had a severe attack. As he got up to shake hands with the curate of the place, he suddenly staggered, stammered, and dropped into the arms of his visitor, who had rushed forward to support him. He remained in the most profound apoplectic stupor for more than ten hours, with complete paralysis of the right side. For a few days, he gave only very obscure signs of intelligence; but from that seizure, he entirely lost the faculty of speech. A few months afterwards, he almost completely recovered the power of moving his right leg, but the movements of his right arm have always been impeded. During the summer of 1860, he had, for the first time, an epileptiform seizure; he had three of these during that year, six in the following year, and four in 1862, up to the month of August; since which date, he has had no return of them.

When, in the spring of 1863, I saw M. X——, I found him looking very well and dressed with care, and even with elegance. His face was intelligent and smiling, and full of benevolence. He rose when I entered, and showed by his gestures, and especially by the expression of his face, that he was pleased to see me. He could not speak, and only uttered in a faltering voice unintelligible words, in which the monosyllable, "*Yes*," returned frequently. When I questioned him, he answered "Yes" to everything, even when he shook his head in denial. "How old are you?"—"*Yes!*" "How far back do you date your illness?"—"*Yes!*" &c., &c. It could be easily seen, however, that he was not satisfied when the word, "Yes," was wrongly applied, for he then made an impatient gesture. He looked pleased, on the contrary, when the word was used appropriately. He sat to table with us at dinner, used his left hand, and ate with great propriety, and with infinitely greater reserve than the generality of ordinary paralytics, who often eat voraciously and in a dirty manner. He looked after his guests during dinner, and took part in some of the discussions carried on. When the delicate flavor of the lamb of the country was praised, he nodded assent; whilst, on some of the guests saying that the kid of the country had a better flavor than the lamb, he shook his head in disapproval. He made signs to the servant to hand the wine round, and when wine of an esteemed vintage was going round, he made signs that it should be drunk in preference to the rest.

The dinner over, he rose with the rest of the company, resting on his stick, and politely let the gentlemen who were escorting ladies pass first. We next sat down, and I tried to make out how far he could give proofs of intelligence. As he always answered "*Yes*," I asked him whether he knew how that word was spelt, and on his nodding assent, I took up a large quarto volume with the following title on its back, "History of the Two Americas," and requested him to point out the letters in those words which formed the word yes. Although the letters were more than one-third of an inch in size, he could not succeed in doing as I wished. By telling him to seek for each letter in turn, and by calling out its name, he managed, after some hesitation, to point out the two first, and was very long in finding the third. I then asked him to point out the same letters again, without my calling them out first, but after looking at the book attentively for some time, he threw it away, with a look of annoyance, which showed that he felt his inability to do as I wished him.

He played every day at all-fours, hiding his cards behind a pile of books, and using his left hand. He often won when playing with the curate, the doctor, or his son, without their allowing him to do so out of kindness. Whenever he played a trump, he laid his hand on it with an air of authority which showed that he knew its value. His son and Dr. Laffitte declared to me that he played as well as he ever used to do. Sometimes his son sits by his side to advise him, and stops him when he takes a card which is not the proper one, but he insists on playing as he likes, and by winning the game proves to his adviser that if he sacrificed a card, it was because he could thus improve his game. Although his son manages all his affairs, he insists on being consulted about the leases and contracts, &c.; and the son stated to me that his father indicates perfectly well, by gestures which are understood by those habitually round him, when certain portions of the deeds do not please him, and that he is not satisfied until alterations are made, which are, as a rule, useful and reasonable.

Although his sight was good, he could not read, or, at least, understand the sense of what he read; he listened with pleasure, however, when he was read to. When I asked him his age, he told it me in such a remarkable manner that I must mention it. After first closing his left hand, he opened it out, showing me his five fingers one after the other; he then closed the hand a second time, and next stretched all his fingers out simultaneously and separating them at the same time. For a third time he closed his hand again, and then showed me two fingers. I confess I did not understand what he meant, and I said to him that he had only indicated twelve years. He laughed at this, as if he knew that the want of intelligence was on my side. He then began anew slowly to show me his five fingers, one after the other, making each time a movement with his head and arm as if he wanted to fix my attention more. On my saying that he meant to say fifty, he nodded approvingly with a smile, and then opened out his whole hand, and after closing it, showed me two fingers. He meant to say fifty-seven years; in fact, his real age.

He could not put together loose letters of the alphabet, nor write with his left hand, two things which most paralytics can do. It has often happened to him to say a word which he has not uttered for a very long time, as if an old impression were revived in his brain. Some time ago, he dropped his handkerchief, and as a lady near him picked it up and gave it to him he said to her, "*Thanks!*" in a loud and distinct voice. His friends were delighted at this, and thought that he had recovered his speech. He was asked, implored to say the word again, it was repeated to him several times, but all was in vain, he never could succeed. I will presently speak of an English banker whom I saw with Drs. Campbell and Blondeau, and who presented anomalies of the same kind.

I now come to a most important point. It seemed as if this patient was in full possession of his intellect, and the expression of his face clearly showed this, as well as his aptitude for card playing. Yet he had forgotten the words themselves, besides having become incapable of articulating them. When I took up his spectacles and asked him what they were, he seemed to make an effort, and yet answered only *yes* as usual. It was evident, however, that he was not satisfied with his answer; and, taking the spectacles with his left hand, he placed them over his nose, as if to show that if he could not tell the name of the object, he knew perfectly the use of it. I then asked him if he remembered the name of the article, but he shook his head. "It's a pen," I said, but he laughed, and moved his head and arm as if he understood that I was joking. "It's a knife, then," and he continued to laugh. "Perhaps it's a pair of spectacles?" "*Yes,*" he replied quickly, clearly showing by signs that I was not joking then. On my attempting, however, to make him say the word *spectacles*, he failed even to articulate the first syllable.

Whenever he grew impatient, he did not always show it exactly in the same way. With strangers, he contented himself with a very significant movement of the shoulder, shrugging it with a look of discouragement and *ennui*. But when he was alone with his wife, his son, or his servants, he showed his impatience by using a very well-known oath.

He sometimes proposed guesses to those about him. He seemed to take great pleasure in looking over collections of portraits in illustrated papers; and he would sometimes hide the name underneath the print, and ask whose portrait it was. As this somewhat childish game seemed to amuse him, his friends kept it up by pretending to make mistakes. "It's Napoleon's portrait," they say, "Alexander's of Russia," or "King Leopold's of Belgium." He laughs incredulously, and continues to ask. "It's Queen Victoria's," and he then takes his hand away, showing that the person has guessed right.

The young woman who was at No. 5 in St. Bernard Ward, was in exactly the same state, and the history of her case is as follows:

Adèle Anselin, aged thirty-two, of apparently good health, stout, and with a fresh complexion, was admitted under my care whilst suffering from acute pneumonia, as well as from right hemiplegia, and on examining her with care, I recognized the presence of chronic endopericarditis and mitral regurgitation. In the course of the year which she spent in my ward, she had slight attacks of hæmoptysis, perhaps depending on the state of her heart; but when she left the Hôtel-Dieu, her general health was as good as possible. Before she came under my care, she had been an in-patient at the

Lariboisière Hospital for several months, on account of her paralysis, which had occurred two years previous to my seeing her. She had formerly had acute articular rheumatism, so that I conjectured that her cerebral affection was the consequence of an embolon, which had come from one of the diseased cardiac valves. The hemiplegia had been sudden and complete; she had not lost consciousness; but the faculty of speech was abolished then as it was on admission. The paralysis of the arm had not been in the least modified, but she could perform certain movements with her right leg.

You remember how long I used to stand by her bed, in order to ascertain the state of her mind. When I asked her her name she could not tell it me. Whenever she got impatient, she exclaimed, "Oh, pity!" She answered pretty well by signs. When asked whether she remembered her name, she answered "No;" yet if another name than hers were mentioned, she shook her head, whilst if her own were mentioned, she laughed and nodded approvingly. She sometimes remembered her Christian name, *Adèle*, although she pronounced it very badly. She could never be made to say *fork*, *spoon*, *mirror*, *book*, whether the objects themselves were shown to her, without their names being mentioned, or whether their names alone were mentioned. I succeeded, however, in making her count up to twenty, by calling out the figures, one after the other, in her presence. But if, after she had gone through this exercise, I asked her to go over it again by herself, she got muddled before she had counted ten, and never could count up to twenty entirely by herself. Whenever she exerted herself trying to remember and to articulate words, she constantly exclaimed, "Oh, pity!" and complained of a painful sensation in the right side of her throat, just as the woman, Marie K——, whose history I have already related. She read almost all day a religious book which the sister had given her, but I discovered that she almost always read the same chapter and the same page; and yet she pretended that she understood well what she read.

As is the case with nearly all aphasic persons, she knew the use of the objects which she could not call by name. Thus, when I put a spoon in her left hand, she lifted it to her mouth, and when I showed her a mirror, she held it before her, and looked at her image in it, laughing. I need not add that the movements of her tongue and lips were normal. She knew *ecarté*, and I played several games with her. I will not say that she played well, but she never, at least, played the wrong color; she knew well her trumps, and when I pretended to cheat she found it out, and protested, laughing. After all, she spent a whole year in my ward, and although physicians and students, the sisters, the servants, and the other patients kindly tried to teach her, she went from here to the Salpêtrière in nearly the same state as when she was seized, three years previously.

The next case, which, is the analogue of the preceding, is more interesting, from the patient being a very intelligent and educated man.

Mr. T——, a Russian functionary, is nearly sixty years old. He has always enjoyed good health, in spite of fatiguing and assiduous office work. He has never had heart disease. Last year, in the spring, he was seized with paralysis on the *right side*, without loss of consciousnes, but with complete loss of speech. From the very beginning his friends were struck with this important fact, that his eyes expressed intelligence, that the gestures which he made with his left hand showed great lucidity of mind, and yet that the most direct and pressing questions could only elicit *yes* or *no* from him. For a few months he remained in the same condition, but after that time he could get up, and the paralysis of the arm and leg improved so much that he could walk pretty well, and use his right hand in dressing, but he did not recover his speech.

I saw him in the autumn of 1862, with Dr. Galinzowski, and again on July 2d, 1863, on his return from Nice. With the exception of attacks of hepatic colic, which were very severe, but fortunately rare, his general health was good; his aspect was good, he had got stout, and the movements of his arm and leg were embarrassed to a slight extent only. His tongue moved rapidly and freely, and could execute all the movements necessary for articulation, and yet he could not speak. It sometimes happened, however (as in all cases of aphasia), that he said a word very distinctly and to the point, but was unable to repeat it, when asked to do so, however pressingly.

A remarkable circumstance in Mr. T.'s case is this: He belongs to the highest circles in St. Petersburg, and speaks French like a Parisian; yet, since his illness, he does not speak a word of French. When I question him, he understands me perfectly, but he always answers with a Russian word. On my telling him, in fun, that he is not polite, because I don't understand Russian, he smiles, and says *da*, a Russian word which means *yes;* but he is unable also to construct even a part of a sentence in his own tongue. He, nevertheless, gives signs of intelligence which are rather curious. His ordinary medical attendant, Dr. Galinzowski, is a Pole, and one can understand that, whilst the present cruel war between Poland and Russia is carried on, the doctor and his patient's friends should not agree.

On one occasion, Dr. Galinzowski was speaking of a terrible engagement which had been fought, near a small village, in his own province in Poland, when Mr. T., who had seemed to take part in the discussion, as shown by his animated looks and his agitation, got up, and going to a map, pointed with his finger, after a prolonged search, to the locality which was being discussed; and it turned out that he was right.

He plays whist every day with his daughter, or those of his friends who come to visit him. He plays as well as he ever did; he counts his points well, questions by signs those of his adversaries, and if one of them counts too many honors, he notices it, and by a gesture insists on correcting the error.

These are, certainly, proofs of intelligence and memory; and yet Mr. T. has forgotten the names of things. When asked what a spoon is, he makes a movement showing the use of a spoon; but when asked its name, he does not know it either in Russian or French, and this does not proceed from inability to articulate, but is due to actual forgetfulness. There is no doubt on this point, because when asked if he remembers the name of a spoon which is shown to him, he shakes his head, although he says yes, and shrugs his shoulder, at the same time, in a manner expressive of the grief which he feels at his want of memory. When told that it is a pencil or a fork, he shakes his head, although he still says yes; but when the spoon is called by its proper name, he nods approvingly, thus affording the proof that he has forgotten the word, and only remembers it when it is mentioned in his presence.

In August, 1863, a lady came to consult me with her son, aged twenty-five. Four years previously, this young man had, for several days, complained of headache, when he suddenly called out to his mother one morning, "*Oh! I feel something extraordinary inside me.*" These were the last words he spoke: his right arm and leg became numb, and after a few hours, the hemiplegia was complete. After a short period he regained some power of moving first his leg, and then his arm; but when he came to me, he still walked with difficulty, and could only use his hand for very rough purposes. The aphasia, however, which had from the first day been complete, had not diminished. He could articulate two words only: *No*, and *mamma*. "What's your name?"—"Mamma." "What's your age?" —"Mamma, no." He yet knew that he did not answer as he ought. He had taught himself to write with the left hand, but had not got beyond signing his own name, Henri Guénier. He wrote it very legibly on a piece of paper which I gave him. "Since you write your name," I then told him, "say Guénier." He made an effort, and said "*Mamma*." "Say Henri." He replied, "*No, mamma*." "Well, write *mamma*." He wrote *Guénier*. "Write *no*;" he wrote again, *Guénier*. However much I pressed him, I could obtain nothing more. His mother informed me that he played a pretty good game at cards or dominoes. He used to be very fond of reading, and often took up books, which he seemed to read with intelligence; but his mother had noticed that he put the book away after a few minutes, as if he found no interest in it; and yet she took care that the books about him were of easy comprehension as well as amusing. His face looked intelligent, as it does in most cases of aphasia; but as he had perfect health and had no headache, and as his sight was excellent, his intellect must have been somehow impaired, since he found no charms in books which would formerly have amused him.

I observed another case of the kind, with Drs. Campbell and Blondeau. An English banker, a resident in Paris, aged 42, robust and stout, fond of good living, and of a lively, cheerful temperament, went out as usual in his carriage, on the 9th of April, 1863. He was returning home to breakfast, about eleven o'clock, when, on stepping out of his carriage, he dropped down without losing consciousness. His whole right side was paralyzed, and the paralysis had probably begun, without his perceiving it, during the latter portion of his drive. The porter carried him into his lodge, and Drs. Campbell and Blondeau were fetched, and arrived at the same moment with one of my colleagues in the Faculty. There was complete right hemiplegia, sensibility being almost completely abolished, whilst the most violent irritation could not excite any movement. The patient tried to speak, but could not articulate a single word, and scarcely succeeded in uttering a few grunts; yet his eyes were full of intelligence, and he seemed to understand the questions that were put to him. There had been no coma, no stertor.

Drs. Campbell and Blondeau refused to bleed the patient, as was advised by the third physician. They recommended that he should lie down with his head propped up, and merely prescribed acidulated and slightly laxative drinks, trusting more to hygienic means than to active treatment for warding off danger, although they were aware that the left side of the brain was irretrievably damaged. I saw the patient in the evening, in consultation with these two gentlemen, and thoroughly approved their prudence, feeling confident that the patient's life would have been gravely compromised by bleeding and violent purgation, by blisters and those numerous remedies which are used against a deep lesion, which has, as a rule, occurred when the physician is called upon to interfere.

On the two following days fever was lighted up, and symptoms of pulmonary congestion showed themselves, which gave us great anxiety; but a little calomel and musk soon got rid of these, and we could entertain the hope that the patient's life would be saved. The pulse became quiet again, and respiration natural; light food was taken well, and on the twelfth day the patient was made to get up and to sit in an easy chair. Since then, up to the present time, the amelioration has gradually increased, but the faculty of speech is almost *nil*, although the patient can walk by leaning on a friend's arm, and can use his hand to some extent. For more than three months he has only been able to say a few words, devoid of meaning, and always the same; on one occasion, however, a fortnight after the attack, he distinctly said "*My dear*" to his wife. But we never could make him repeat those two words. At present he can say a few words, but very few, and they are not always used rightly. Eight months after his seizure, he had in December, 1863, an attack of eclampsia, and another again in February, 1864, and these attacks will probably recur again.

I now pass on, gentlemen, to the case of a man who died in my wards, after having presented, during life, the most characteristic symptoms of aphasia, and whose brain was examined after death with the utmost care, in Dr. Broca's presence.

The patient was 60 years of age. He had been, at first, under the care of Dr. Vigla, who transferred him to me. His intellect seemed to be impaired; he had been paralyzed for several months, and although he looked as if he understood, when he was spoken to repeatedly, he never answered anything beyond "*Oh! mad.*" General sensibility was normal, and when he was pinched hard, he exclaimed in a more decided tone, "*Oh! mad,*" and shook his head to show that he was annoyed. He died a few weeks after admission. As this was an important case, which might confirm or upset the theory concerning the localization of intellectual faculties, I requested Dr. Broca to be present at the post-mortem examination. The brain was removed with care, and there was found on the left side yellow softening of the lower marginal convolution, of the lower portion of the transverse parietal convolution, and of the convolutions of the insula. At first sight, the frontal lobe seemed to have escaped; but on drawing away the edges of the Sylvian fissure, the softening was seen to extend from the convolutions of the insula to the lower portion of the transverse frontal convolution, and moreover, that the third frontal convolution was itself softened in its posterior portion, that is, in the part nearest the sulcus of Rolando.

The morbid specimen was exhibited by M. Dumontpallier, my then clinical assistant, to the members of the Biological Society, at their meeting on the 28th of March, 1863; and, at Dr. Broca's request, it was afterwards placed in the Dupuytren Museum, where it can now be seen, I believe, so that any one may ascertain for himself that the lesion was really seated in a portion of the sphenoido-temporal lobe, and in the third frontal convolution. This case, therefore, supports Dr. Broca's views. M. Dumontpallier showed also, at the meeting, that the left middle cerebral artery was obliterated by a fibrinous clot, which might have been ascribed to embolism (as the walls of the artery were healthy), if the examination of the heart had ascertained this point. None of the cerebral arteries were atheromatous, however, the right middle cerebral artery was pervious, and there was no trace of defective nutrition in the right hemisphere of the brain.

Now that I have related to you this interesting case, I will pass on to the anatomical questions bearing on aphasia, and discuss what has been written on the subject.

The physiological conditions of aphasia have long ago been observed. I need only mention the following passage of Pliny, in which the learned naturalist observes that nothing in Man is so fragile as Memory: "Illness, falls, a mere fright, impair it partially, or destroy it completely. A man, struck by a stone, forgot the letters of the alphabet; another, who had fallen from a very high roof, no longer recognized his mother or his friends; a third, after a severe illness, forgot that he possessed slaves; and Messala

Corvinus, the orator, forgot his own name."[*] Schenkius,[†] who lived at the end of the 16th century, noticed that, in some cerebral affections, although the tongue was not in the least paralyzed, the patients could not speak, because they had lost their memory: "Observatum a me est plurimos, post apoplexiam aut lethargum, aut similes magnos capitis morbos, etiam non præsente linguæ paralysi, loqui non posse, quod memoriæ facultate extinctâ, verba proferenda non occurrant."

In 1820, the illustrious Lordat,[‡] who became aphasic eight years afterwards, ascribed this affection, which he termed *alalia*, not to paralysis of the tongue, but to a defect of co-ordination of the muscles which are used in the act of speaking. But Lordat did not attempt to specify the part of the brain an injury to which might cause loss of speech. Gall conjectured that the faculty of articulate language was located in the anterior lobes of the brain. Dr. Bouillaud, who studied and adopted some of Gall's theories (within restricted limits, it is true), was led, by clinical observation, to locate this faculty in the frontal lobes. "The anterior lobes of the brain," said he, in the year 1825, at page 284 of his Treatise on Encephalitis, are the organs "for the formation and recollection of words, or the principal signs which represent our ideas." Whilst, in the next page, he emphatically declares that "the anterior portion of the brain is the organ of articulate language."

In 1836, Dr. Marc Dax, of Sommières (Gard.), read at the Medical Congress of Montpellier a very interesting and original essay, in which he attempted to specify, with greater precision than Dr. Bouillaud had done, the part of the brain which he regarded as the seat of the manifestations of thought by speech. So far back as the year 1800, he had noticed that patients suffering from aphasia, when paralyzed at the same time, were paralyzed on the *right* side, and that consequently the anatomical lesion was seated in the left hemisphere. When once his attention had been called to this curious point of pathological physiology, he ascertained clinically that, when there was loss of memory of words, the lesion was always seated on the left; and he added that he had never met with this affection in cases of cerebral disease exclusively limited to the right hemisphere. The title of his essay, besides, sums up his views: *Lesions of the left half of the brain coinciding with the loss of memory of the signs of thought.*

Thus, gentlemen, we see Dr. Bouillaud demonstrating by facts what Gall had obscurely seen; namely, that the material condition on which depends the memory of the principal signs of thought is the integrity of the anterior lobe of the brain; whilst Dr. Marc Dax localizes this faculty in the left hemisphere exclusively.

In the beginning of 1863, Dr. G. Dax, following in his father's steps, sent to the Academy of Medicine a memoir, in which he tries to prove that, in aphasia, the lesion is not only invariably seated in the left hemisphere, but in the anterior and outer portion of the *middle lobe* of that hemisphere. This statement, made in 1863, differs very little, as you may see, from what Dr. Broca has lately shown; for the spot in which Dr. G. Dax locates the lesion is evidently very close to the *insula of Reil*, and consequently to the posterior portion of the frontal lobe.

But Dr. Bouillaud, whilst inclining to the opinion that aphasia is, in most cases, due to a lesion of the anterior portion of the anterior lobes of the brain, admitted also that the same morbid phenomenon could be produced by disease of the posterior portion of these lobes. In the memoirs read by Dr. Bouillaud at the Academy of Medicine on the 22d of February and the 7th of March, 1848, these points are well established.

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[*] Pliny. Natural History, book vii, sec. 24.

[†] Joan Schenkii, Obs. Med., lib. vii, in fol., p. 180. Lugduni, 1585.

[‡] Rev. pér. de la Société de Méd. de Paris. Décembre, 1820, p. 317.

In 1856, Dr. Marcé tried to show that there is a co-ordinating principle for the acts of speaking and writing (Memoirs of the Biological Society); and that, in a certain number of cases, the faculty of articulate language can be lost independently of the faculty of language as expressed by writing, or *vice versâ;* but he denied that a special portion of the brain could be assigned as the seat of this co-ordinating principle.

In 1861, at the Anthropological Society of Paris, an important discussion arose concerning the localization of the cerebral functions, in which Drs. Gratiolet, Auburtin, and Broca took the most prominent part. Dr. Gratiolet maintained that all attempts at localization which had been made up to that time, had no basis. Dr. Auburtin affirmed the reverse proposition, citing Dr. Bouillaud's researches, several cases related in Dr. Rostan's works and in Lallemand's letters, and concluding that the anterior lobes of the brain were the seat of the co-ordinating faculty of speech.

The authority of Dr. Gratiolet, who had studied, specially and deeply, the anatomy and physiology of the nervous system, on the one hand, and the facts quoted by Dr. Auburtin from justly-esteemed works, on the other hand, *rendered the solution of the point at issue difficult.* Was the brain to be regarded as a great whole, all the parts of which, as well as their faculties, are mutually dependent? or should it be divided into departments, and the department of each faculty ascertained? M. Broca hesitated like the rest; and yet he was one of the first to bring forward cases lending great support to the theory of cerebral localizations. A few weeks after the discussion at the Anthropological Society, a man named Leborgne, 51 years old, and who had lost speech for twenty years, was transferred to his care, in Bicêtre. The case is related in detail in the Bulletins de la Société Anatomique, August, 1861; but the following is a summary written by M. Broca himself for the Proceedings of the Anthropological Society.

The patient was admitted into Bicêtre twenty-one years ago. Shortly before then he had lost the power of speech, and could utter one syllable only, which he usually repeated twice in succession. Whatever question was asked him, he always answered, *tan, tan*, accompanying his answers with very varied and expressive gestures. Throughout the asylum he was therefore known by the name of *Tan.*

At the time of his admission he was intelligent, and could use all his limbs perfectly. After ten years, he gradually lost the power of moving his right arm, and next his right leg; so that, for the last six or seven years, he has been constantly confined to his bed. For some time past, his sight has been noticed to grow weaker; and, lastly, those who were frequently about him observed that his intelligence had failed a good deal within the last few years. He was transferred to M. Broca's care, on account of a diffuse gangrenous inflammation of the cellular tissue of the whole lower extremity, on the right or paralyzed side, extending from the instep to the buttock. The case of this poor fellow, who was unable to speak and write, was somewhat difficult to study. It was ascertained, however, that common sensibility was nowhere impaired; that the left arm and leg moved in obedience to the will; that the muscles of the face and tongue were not paralyzed; and that this last organ moved very freely. There was no doubt, according to M. Broca, that the "*patient's intellect was deeply damaged,*" but that more of it was retained than is required for speech. Besides, Tan was perfectly intelligent for sixteen or seventeen years, although he had been unable to speak for twenty-one years. He died on the 17th of April, 1861.

At the post-mortem examination, the dura mater was found thickened and vascular, lined on its inner aspect by a thick pseudo-membrane; the pia mater was thickened and opaque over the anterior lobes, to which it was adherent, especially on the left side. The frontal lobe of the left hemisphere was softened in the greater part of its extent; the convolutions of the orbital lobule, although atrophied, had preserved their shape; most of the other frontal convolutions were destroyed. From this destruction of the cerebral tissue, there had resulted a large cavity, of the size of a hen's egg, filled with serosity. The softening extended backwards to the ascending portion of the parietal lobe, downwards to the marginal portion of the temporo-sphenoidal lobe, and inwards to the lobule of the insula and the extra-ventricular nucleus of the corpus striatum. To the disorganization of this last part must be ascribed the motor paralysis of the upper and lower limbs, on the right side. The oldest and most extensive lesions, however, were found in the middle portion of the frontal lobe of the left hemisphere. The neighboring portions had softened very gradually only, and it may be considered a certainty that, for a very long period, the convolutions of the frontal lobe were alone affected.

This period probably comprised the eleven years which preceded the paralysis of the right arm, and during which the patient's intellect was unimpaired, and speech alone was lost. It was, therefore, allowable to ascribe, in this case, the loss of speech to the disorganization of the frontal lobe, particularly when Professor Bouillaud's views were kept in mind.

M. Broca's second case, however, seemed to point to a very limited part as the seat of the faculty of articulate language. A man, aged eighty-four, had been eight years previously admitted into Bicêtre, on account of senile debility. He was not paralyzed at that time; his senses, his intellect, were perfect. In April, 1860—that is to say, when he was eighty-three years old—Lelong was seized with apoplexy, whilst going down a staircase. A few days afterwards he left the infirmary, having never been paralyzed, but having suddenly and completely lost the power of speech. He could only articulate a few words with difficulty; his gait was somewhat uncertain, but he was not lame; his intellect did not seem to have been appreciably impaired; he understood what was said to him, and his small stock of words, accompanied by an *expressive pantomime*, enabled him to make himself understood in his turn by the people who lived habitually with him.

On the 27th of October, 1861, Lelong was admitted into the infirmary, under M. Broca, on account of a fracture of the neck of the femur, on the left side. He was *not paralyzed* of motion or sensation; his tongue moved freely in all directions, he could swallow well, all his senses were normal, and his intellect was unimpaired. Yet, when questions were put to him, he only answered by signs, uttering at the same time one or two syllables suddenly and with a certain degree of effort. He could only say, *yes*, *no*, *three*, and *always;* when he was asked his name, he replied *Lelo* instead of Lelong which was his real name. He said *yes* and *no*, at proper times; but he made use of the word *three* in order to express any number, although he knew well that the word did not always convey his meaning, and corrected the mistake which he made in speaking by holding out the proper number of fingers. He could tell the time by a watch, and had retained the notion of units and tens. He had not lost his memory, and on one occasion only, according to M. Broca, was his memory at fault, when he was asked how long ago he had lost the power of speech.

M. Broca sums up the history of this case, which is published *in extenso* in the Bulletins de la Société Anatomique (November, 1861), by affirming—1. That Lelong understood all that was said to him; 2. That he used with judgment the four words of his vocabulary; 3. That he was of sane mind; 4. That he knew written numeration, and at least the value of the first two orders of units; 5. That he had lost neither the general faculty of language nor the power of moving the muscles which are used in the production of sound and in articulation, and that he had consequently lost the faculty of articulate language alone. He was, therefore, aphasic. M. Broca does not, however, in his conclusions, call attention to the fact that Lelong, who knew how to write, and whose hand was not paralyzed, could not *guide* his hand so as to form letters; although the fact itself is mentioned in the detailed and complete history of the case, as given by M. Broca.

The patient died on November 8th, 1861, twelve days only after his fall, from the consequences of the fracture of his femur, and without having suffered from any cerebral complication. The post-mortem examination was made with the greatest care. The right hemisphere was found healthy throughout, as well as the cerebellum, the pons Varolii, and the medulla oblongata. In the left hemisphere, the thalamus opticus, the fornix, corpus callosum, corpus striatum, the occipital and parietal lobes, the lobule of the insula, and the orbital convolutions which form the inferior layer of the frontal lobe, were healthy. It was thought, however, that at the point of union of the anterior extremity of the ventricular nucleus of the corpus striatum with the medullary substance of the frontal lobe, the consistency of the brain-substance was slightly diminished; but this lesion, M. Broca adds, if it can be regarded as such, was totally independent of the principal one, and separated from it by a considerable thickness of healthy tissue.

Before describing, however, the lesion which was found, in this case, to be perfectly limited to a portion of the posterior third of the second and third left frontal convolutions, it will be necessary to give a brief description of the arrangement and relations of the cerebral organs which shall have to be mentioned.

The sulcus of Rolando separates the frontal from the parietal lobe, running obliquely from above downwards along the outer surface of the hemisphere, and beginning at the median fissure between the two hemispheres, and ending in the Sylvian fissure. It is limited, anteriorly, by the transverse frontal convolution, posteriorly, by the transverse parietal convolution. The anterior or frontal lobe comprises, therefore, laterally, all that portion of the hemisphere which is situated in front of the sulcus of Rolando, and inferiorly, all that portion which is in front of the Sylvian fissure. The lower portion of the frontal lobe consists of the orbital convolutions, whilst its upper and lateral portions are constituted by the frontal convolutions properly so called. These are three in number: an upper or first frontal convolution, a middle or second convolution, and a lower or third frontal convolution. They are all directed from before backwards, and terminate, after a more or less tortuous course, in the transverse frontal convolution, of which they seem to be the ramifications. The third frontal convolution is free in its upper half, and separated from the temporo-sphenoidal lobe by the Sylvian fissure, of which it forms the upper margin. It is on account of this relation that the third frontal convolution is sometimes termed the *upper* marginal convolution, whilst the name *lower* marginal convolution is restricted to the first temporo-sphenoidal convolution. When the two marginal convolutions, the upper and lower, are drawn away from the Sylvian fissure, there is seen a large and slightly prominent eminence, from the summit of which proceed five small simple convolutions, or rather five rectilinear folds, radiating in a fan-like manner. This eminence is the *lobule of the insula*, which covers the extra ventricular nucleus of the corpus striatum, and which, rising from the bottom of the Sylvian fissure, is structurally continuous by its cortical layer with the deepest portion of the two marginal convolutions. The result of these structural relations is, that a lesion which extends by continuity from the frontal to the temporo-sphenoidal lobe or the reverse, must necessarily pass through the lobule of the insula and then affect the extra ventricular nucleus of the corpus striatum.

These anatomical details, which I chiefly borrow from M. Broca's memoir, easily account for certain very limited lesions, such as those found in Lelong's case. In his case, indeed, the posterior third of the second and third left frontal convolutions was alone destroyed, over a space of about 15 or 18 millimetres. The transverse frontal convolution was normal, whilst inferiorly, the lesion extended as far as the lobule of the insula, but without involving it. The result of this loss of substance was a cavity full of serosity and closed externally by the pia mater. The walls of this cavity were firm, and on them were small spots of an orange-yellow color, probably of blood origin, and afterwards proved to be so by the microscope. This was, therefore, an old hemorrhagic cyst, and the patient, as you know, had suddenly lost his speech since a fit of apoplexy which he had eighteen months before he died.

This case proves, therefore, that when there is no other brain lesion than a loss of substance of the posterior third of the second and third left frontal convolutions, there may solely exist, either as a coincidence, or as a consequence, loss of the faculty of articulate language.

Thus, gentlemen, we have Professor Bouillaud placing the organ of the manifestations of thought by speech in the two anterior lobes of the brain; Dr. Marc Dax locating it in the left hemisphere exclusively; and Dr. G. Dax, at the point of union of the middle with the frontal lobe of the left hemisphere; whilst M. Broca points to a more definite spot, and although he did not probably know of Dr. Marc Dax's essay, and certainly not of Dr. G. Dax's researches, he, like them, points to the posterior portion of the left frontal convolution as the seat of the faculty of speech.

If there were on record several cases exactly similar to the one which I have just related, we should be compelled to admit a relation of cause and effect between the seat of the anatomical lesion and the loss of speech. Dr. Charcot, in 1862 and 1863, exhibited to the members of the Biological Society several brains removed from old women who had died in the Salpêtrière, and who had, for a variable period during life, suffered from loss of speech. In most instances, the lesion was complex as in M. Broca's first case. Thus, there had generally been paralysis as well as aphasia, and the second and third frontal convolutions were not the only spots where there was softening or hemorrhage, but the lobule of the insula and the temporo-sphenoidal lobe were also the seats of an anatomical lesion which had probably occurred simultaneously everywhere, or which had resulted from the extension of the softening by continuity of tissue.

The softening was frequently of an amber-yellow color, and varied much in degree, both as regards depth and extent. A very remarkable fact, however, was, that in the first ten or twelve cases related by Dr. Charcot, the third frontal convolution was disorganized at its posterior extremity, thus lending considerable support to M. Broca's theory.

Subsequently, however, Dr. Charcot was the first to communicate to the members of the Biological Society a case of aphasia without damage to the third frontal convolution.

This case has been published in the "Gazette Hebdomadaire," and M. Broca, who assisted Dr. Charcot at the post-mortem examination, has fully acknowledged its value, and that it invalidated the anatomical law which he had laid down. The patient was forty-seven years of age, and had become hemiplegic and aphasic since an apoplectic fit which she had had eight months previously.

Intellect and memory seemed to be preserved, for, during her stay in the infirmary, she recognized patients whom she had formerly seen in the Salpêtrière. But her power of articulate language "was restricted to the utterance of the monosyllable *ta*, which she habitually repeated, with very great rapidity and distinctly, four or five times in succession (*ta*, *ta*, *ta*, *ta*), whenever she attempted to answer a question or to communicate her own ideas. The tongue moved freely and in all directions."

An examination of the brain after death, showed that there was softening: "1st, of the so-called lower marginal convolution in all its extent, and of a portion only of the second temporal convolution, of the temporal lobe; 2d, of the lower extremity and the whole of the two posterior convolutions of the insula of Reil. In depth, the softening extended in the direction of the corpus striatum; the whole of the extra-ventricular nucleus and the posterior half of the intra-ventricular nucleus were also softened. The thalamus opticus was normal."

The transverse parietal and transverse frontal convolutions, the three antero-posterior frontal convolutions, known by the names of first, second, and third frontal convolutions, were examined thoroughly, one after another, with the greatest care, in M. Broca's presence. To the naked eye, they did not seem to have undergone any change of size, color, and consistency. Lastly, thin sections of several portions of the third frontal convolution were examined under the microscope, and the nerve-tissue was found unchanged; here and there only, two or three granular bodies were seen on each microscopic preparation. But neither Dr. Charcot nor M. Broca himself regarded the presence, in such small numbers, of these granular bodies as a sign of organic lesion, as they can be met with in nerve-tissue without any coexisting functional disturbance. Let it be kept in mind, besides, that both Dr. Charcot and M. Broca were anxious to determine the presence of a pathological lesion; and if they declare that none was to be found, we must certainly conclude with them, that the faculty of articulate language may be destroyed without there necessarily being an appreciable organic alteration of the third left frontal convolution.

The following case, observed by Dr. Vulpian, equally proves, in my opinion, that aphasia may be caused, as Dr. Marc Dax had conjectured, by lesions of the left hemisphere independent of the frontal lobe.

"A woman, aged seventy-three, is transferred to the infirmary at the Salpêtrière on the 15th of December, 1863, because for a few days past she had been observed to grow markedly weaker.

"When first seen by Dr. Vulpian, she had no fever, and seemed to suffer from no thoracic or abdominal complaint; but she was not able to speak, and all the attempts to make her say a single word were fruitless. She seemed to understand what was said to her, tried to answer, but on rare occasions only succeeded in stuttering unintelligibly; in general, she uttered no sound at all. She had no paralysis of the limbs, face, or tongue; she squeezed pretty hard and equally well with both hands; she walked without help, but slowly, and taking short steps, without dragging either leg. She did nothing extravagant.

"She was watched day after day, and for the first ten days her condition did not change. Her intellect was evidently of a low grade although she correctly nodded or shook her head, according as she meant yes or no. 'One morning I found her in tears' (says Dr. Vulpian), 'and as soon as she saw me she went through a pretty expressive pantomime which suggested to me the idea that she might have been beaten; and I indeed learned that a neighbor of hers, who was delirious, had got up during the night and struck her repeatedly. I must add, that her pantomime, although expressive, was not so clear as would have been that of a perfectly intelligent person.'

"On one occasion only, she said, '*Yes yes, sir,*' but could not be made to repeat the same words on the following day. Ten days after her admission, after looking more prostrated than usual during the night, she was found one morning partially paralyzed on the right side; on the next day, the paralysis had become complete. A few days afterwards, the face deviated a little (the left commissure being pulled a little towards the ear); there was some tendency to contraction of the paralyzed arm. No appreciable change in the patient's intellectual condition was noticed with regard to speech, although she no longer uttered the sounds which she did before. A month after her admission, she had pneumonia on the right side, of which she died at the end of six days.

"The persons who had been in the same part of the asylum with her, declared that she was not able to speak when she was *first admitted into the Salpêtrière,* and one of them even went so far as to assert that the patient had lost her speech *three years before she was transferred to the infirmary.* I had felt no hesitation, therefore, in regarding this case as a typical one of aphasia, which was all the more remarkable from there having been no paralysis at first, and from right hemiplegia having occurred towards the close.

"Dissection, however, did not disclose the lesion which I fully expected. I found a broad patch of softening, of apparently recent date, in the posterior half of the white supra-ventricular nucleus of the left cerebral hemisphere, and no trace of disease in the frontal or other convolutions. Old lesions, slight in degree, lacunæ, were seen in the corpus striatum and the thalamus opticus on the same side, and an analogous lesion, of still smaller extent, but of as old a date, in the right corpus striatum.

"This case, therefore, was apparently an exceptional one, and by ascribing a somewhat old date to the softening (a central portion of it looked, certainly, a little older than the rest) the aphasia could be referred to this lesion. So that this would have been a case of aphasia produced by a lesion of the posterior part of the hemisphere. Luckily I found some notes about this patient who, six months previously, had been under my care for nine days. At that time she spoke, and could articulate any word. She could ask for what she wanted, and even talk a little with other patients. It was true that she spoke very little, with some difficulty in finding words. Articulation was slow when a sentence had to be spoken, whilst, on the contrary, the following words came out explosively, as it were: *Yes, sir, yes. No, sir, no.* She never said *yes* or *no* in any other way, except when repeatedly pressed. She had no paralysis of the face, eyes, tongue, or limbs, but there was already some weakness of the lower limbs. She told me at that time that her speech had become embarrassed three months previously, after repeated attacks of giddiness several days in succession. Since then, she occasionally felt giddy, and her speech became more embarrassed. These notes, therefore, to some extent modify the conclusions which might be drawn from the case.

"Both middle cerebral arteries were very atheromatous; but whilst the right artery was still pervious to the blood, the left one was completely or almost completely plugged up (the plugging seemed to be complete in one point at least) in two places, separated by an interval of about one centimetre from one another, owing partly to the atheromatous thickening of its walls and partly to an indurated fibrinous deposit of manifestly old date. This deposit seemed to have been the result of thrombosis rather than of embolism. It is probable that the plugging of the vessel was the cause of the first symptoms. Circulation was considerably impeded on several occasions, but was probably re-established, although incompletely, by collateral channels. I can thus account for the old and partial patches of softening, resulting in the lacunæ, which dissection disclosed in the two corpora striata and in the left optic thalamus, and the somewhat intermittent embarrassment of speech, as well as the weakness of the lower limbs and the failure of intellect. All these symptoms were due to insufficient nutrition of the brain. The right cerebral hemisphere must have been also imperfectly nourished, although to a less degree than the left hemisphere, since the walls of the left middle cerebral artery were atheromatous.

"When the patient was for a second time admitted under me, she was then suffering from one of the attacks to which she was liable, and during which the impediment in her speech was so exaggerated as to merge into aphasia. The cerebral softening set in next, doubtless owing to the persistent plugging of a portion of the arterial system, which, until then, had been more or less pervious to the blood."

M. Fernet, house-physician to the hospitals, communicated to the Biological Society, in March, 1863, a case of complete left hemiplegia, with softening of the right frontal lobe and thrombosis of the middle cerebral artery on the same side.

The patient, a woman aged forty-six, had not been aphasic. The whole of the frontal lobe was in a pulpy condition, and although it is not specified that the third frontal convolution was softened, it was no doubt affected, and M. Fernet has himself informed me that it was. The temporo-sphenoidal lobe and the convolutions of the insula were not involved.

Since this patient was not aphasic, we must conclude that the frontal lobe on the right side may be entirely disorganized, without aphasia following of necessity. Some very severe critics might not be disposed to set much value on this case, because the precise limits of the softening are not given in detail; but M. Fernet, it should be observed, did not draw attention to the question of aphasia, because as he said himself, he was not sufficiently acquainted with the subject. This case becomes of great importance, however, when it is placed by the side of the one published a few months later by Dr. Parrot in the Gazette Hebdomadaire (July 31, 1863), under the following heading: "Complete Atrophy of the Lobule of the Insula and of the third Convolution of the Frontal Lobe, with Retention of Intelligence and of the Faculty of Articulate Language." The pathological portion of this case does not admit of criticism, and it must be acknowledged that there was really softening of the posterior third of the frontal convolution,—of the very spot, in fact, where M. Broca had located the faculty of articulate language.

The following case, which Dr. Charcot has kindly communicated to me, is exactly like Dr. Parrot's.

"Egris-Valentine Thérèse, aged seventy-seven, is admitted into the Salpêtrière on the 21st of December, 1863, on being discharged from La Pitié, where she had been for three months under Dr. Marrotte's care.

"*Intelligence and memory seemed to be remarkably good.* The patient declared that, about three months ago, she was seized with complete paralysis of the left side; she fell down, lost her senses, and remained insensible for nine hours. She was carried to La Pitié, and her speech, which had been impeded at first, soon became natural again. Whilst she was in La Pitié Hospital, her lower limbs and her left arm swelled considerably; and they were still œdematous. The swelling was preceded by diarrhœa which had continued ever since. For the last month the patient had had no control over her bladder or rectum, and a patch of gangrene had formed over the sacrum. *There was no embarrassment of speech, no forgetfulness of, or mistake in, words when speaking.*

"The patient died of pneumonia on January the 3d, 1863; and at the post-mortem examination, the following lesions were found: The amount of subarachnoid fluid is considerable; there is very extensive yellow softening of the outer surface of the right frontal lobe, with nearly complete atrophy of the convolutions. The parts softened are the anterior marginal convolution, and the second and third frontal convolutions, which are completely destroyed, and the posterior part of the lobule of the insula. Microscopical examination shows, in the diseased parts, numerous granular corpuscles, a considerable amount of fatty granules in the intercellular tissue, and atheromatous degeneration of most of the bloodvessels.

"The central parts are healthy—namely, the corpora striata, thalami optici, and lateral ventricles. The right crus cerebri is markedly smaller than the left, and is of a grayish tint. In the interstices between its nervous elements, a certain number of granular corpuscles are found. The pons Varolii is flattened on the same side, as well as the anterior pyramid, which differs from the left one, both in respect of its smaller size and its grayish tint like that of the crus, and like it owing to the granular bodies. The upper portion of the spinal cord was alone examined, and its left half was smaller than the right, the diminution in size being chiefly due to that of the antero-lateral columns."

You see, gentlemen, that in this case, as in those reported by M. Fernet and Dr. Parrot, the third frontal convolution was seriously damaged; but the lesion was seated on the *right* side, whereas M. Broca maintains that disease of the third convolution of the *left* frontal lobe can alone produce aphasia.

The cases on which M. Broca's memoir is based—the more numerous ones which I have just quoted, others published by Drs. Vulpian, Charcot, and Perroud—seemed to establish incontestably, not that aphasia was of necessity produced by a lesion of the third left frontal convolution, since Dr. Charcot's case has done away with that opinion, but at least that it was only produced by a lesion of the *left cerebral hemisphere*, and never by a lesion of the *right*, as Dr. Marc Dax had shown. Indeed, there was not a single authentic case on record of aphasia with left hemiplegia.

The three cases published by MM. Fernet, Parrot, and Charcot prove that lesions, which on the left side produce aphasia, do not cause it when seated on the right side. But M. Broca believed that he was in a position to affirm (however bold this opinion might be in a physiological point of view) that the faculty of articulate language was structurally dependent on the integrity of the third left frontal convolution. He did not attempt to explain this strange localization, but simply noted the facts which seemed to declare in his favor.

You see, gentlemen, that I have kept back none of M. Broca's arguments, and that I have allowed them to be stretched almost to the limits of absurdity; for is it possible in physiology to admit that in an organ so exquisitely symmetrical as the brain, there may be in one of the hemispheres a portion discharging a function which does not appertain to the other hemisphere? Analogy and common sense would protest against such a conclusion, and although, in almost all the cases of aphasia which have come under my observation, the paralysis (when present) always affected the right side, and I was therefore obliged to admit a lesion of the left hemisphere, I could not accede to M. Broca's strange doctrine. You remember the case of Marcou which I have already related to you, and which proves that aphasia, in its most characteristic form, may accompany left hemiplegia, and consequently, a lesion of the right hemisphere. M. Broca's doctrine was, therefore, upset by such a case, although it is true that when aphasia is attended with paralysis, as it most frequently is, the lesion is nearly always on the left side of the brain, whilst the loss of motion is on the right side.

I know very well that certain objections may be raised about Marcou's case. It will not be said that he was not affected with aphasia, but it will be suggested that as there was no autopsy, two lesions might be admitted, one causing left hemiplegia and seated in the right hemisphere, the other producing aphasia without hemiplegia, and seated in the third left frontal convolution. I admit that in the absence of an anatomical demonstration to the contrary, this is not impossible; but I must call your attention to the fact that Marcou became aphasic at the very moment when he was seized with left hemiplegia. There must have been, then, two simultaneous lesions—one of the left frontal convolution, and the other of the right hemisphere. Now such cases do pretty frequently occur, and I have, on several occasions, shown you multiple apoplectic cysts in the brain of individuals who had died of cerebral hemorrhage. But such multiple cysts are only found in cases of severe apoplectic attacks, and very rarely in such mild seizures as Marcou's. They are pretty frequently met with, also, as a consequence of falls on the head. Thus, in a case observed by M. Ange Duval, surgeon to the Naval Hospital at Brest, and communicated by M. Broca to the Surgical Society, on the 24th of February, 1864, the patient became aphasic after a fall on the head, and there was found at the same time, in the *right anterior lobe*, a sanguineous cyst with superficial alteration of the orbital convolution, and on the *left* side laceration of the third frontal convolution, which was completely softened. For the present, however, I am justified in regarding Marcou's case as one of *aphasia with lesion of the right hemisphere;* and I think that the following conclusions may justly be adopted:

Aphasia is produced in nearly all cases by an injury to the frontal lobes, as Professor Bouillaud has shown.

The lesion, as Dr. Marc Dax has established, is almost exclusively confined to the left hemisphere; whilst its most frequent seat is the posterior part of the third left convolution, as M. Broca was the first to point out.

Now, gentlemen, let us examine the question in another point of view.

If it be easily admitted that aphasia, when accompanied by paralysis, is due to softening or hemorrhage, it is difficult to conceive the nature of the lesion when the aphasia lasts a few minutes or a few hours only, and is not accompanied or preceded either by headache or by paralysis, even of a transitory character. Yet it is still more difficult to deny the existence of a lesion. I grant that this lesion is neither softening nor hemorrhage, but there must have been some modification in a portion of the brain, and probably in the same part which is deeply damaged in cases of aphasia attended with paralysis, a modification which is perhaps the analogue of the transitory congestions which we observe in certain exposed parts, or of those deep disturbances of the capillary circulation which sometimes manifest themselves by hyperæmia, sometimes by anæmia, sometimes by the loss or by the exaltation of sensibility.

We are driven to conjectures, as you see, gentlemen. But I wish to call your attention to an important fact which has not passed unnoticed, but which has been too much lost sight of by practitioners—namely, to remains of old lesions which are found in the brain of individuals who, for several months, had had aphasia without paralysis, and who died of some acute cerebral affection or of some complaint independent of aphasia. Clinical experience proves, therefore, that there may exist in the brain lesions of sufficient gravity to cause persistent aphasia without producing paralysis; and it is not impossible that a small hemorrhage may cause aphasia of a few hours' duration, in the same manner as we see it produce, in some cases, paralysis of one, two, or three days' duration. How often are the remains of eight or ten successive hemorrhages found on dissection in the brain of individuals who have only had two or three paralytic strokes? I could not, therefore, affirm that cases of transitory aphasia (which are not infrequent) are not produced either by a small hemorrhage or by the softening of a very limited portion of the frontal lobes. This view derives support from the fact that aphasic individuals, who for several months have given no evidence of paralysis, pretty often die after a violent attack of hemorrhage into, or of softening of, the brain, as in the case of the woman Desteben, which I related to you at the commencement of this lecture.

The seat of these small hemorrhages or of this partial softening, which only cause temporary paralysis scarcely noticed by the patient or his friends, is of very great importance. For if lesions of the frontal lobe very often cause the loss of the faculty of manifesting thoughts by speech, writing, and gesture, they possess a very limited influence on the loss of sensation and motion. If we admit with Professor Bouillaud, as I am inclined to do, that this part of the brain is the seat of the faculty in virtue of which thoughts are manifested by speech, writing, and gesture, whilst other parts hold more especially under their dependence the faculties of motility and sensibility, we can better conceive how the frontal lobes may be slightly injured and yet produce no hemiplegia, and how the optic thalamus, corpus striatum, or centrum ovale of Vieussens may be slightly damaged, and yet cause only a small degree of hemiplegia without affecting the faculty of speaking and of writing.

Dr. Auburtin goes further, and proves by clinical cases, some of which came under his own observation, and others have been reported by esteemed authors, that the anterior lobes of the brain may often be very gravely damaged without there being any sign of paralysis. It cannot be denied that his opinion is based on facts which cannot be contested. One need only read with care M. Broca's first case, that of *Tan*, to be convinced that the anterior lobes of the brain may be really damaged, without paralysis resulting. This patient, indeed, was completely aphasic for a period of ten years, without having manifested the slightest sign of paralysis of the limbs or of the face.

After that time his right limbs became paralyzed, whilst, after his death, dissection disclosed extensive lesions in the neighborhood of the third frontal convolution, as well as in the vicinity of the corpus striatum and the insula of Reil. Is it not evident, especially when this case is compared with the next one, that the aphasia was due to the lesion of the frontal lobe, and the paralysis to that of the parts in the neighborhood of the corpus striatum?

The case which I am now going to relate, came under my observation in the first year of my medical career, and it made such a deep impression on me, that I have ever recollected it.

In the spring of the year 1825 two officers, garrisoned at Tours, fought a duel after a quarrel. One of them fired first, and the ball entered his adversary's head at one temple, passed through the brain, and then raised the temporal bone on the opposite side. Portions of the brain came out of the aperture of entrance of the ball, and some of them were found on the wounded man's hat. He was immediately brought to the Tours Hospital, in a state of stupor; and, although his breathing was easy, he gave no signs of consciousness. The left temporal muscle was divided, the piece of fractured bone raised with a spatula, and the ball extracted. As the operation was concluded, the patient made a sign with his hands, and expressed his thanks in a very low voice. This terrible wound progressed very favorably, and after a few days the patient could speak, and was not paralyzed in the least. At the end of a month he could get about, and during the five months which he spent in the hospital, living almost constantly in the company of the resident pupils, he amused them by his merry ways and his witty conversation, whilst he employed his leisure by writing comedies. Towards the close of the summer, he complained of violent headache, and stupor supervened, with signs of acute cerebral softening, and on dissection, after death, a splinter of bone was found in the track of the ball, which had caused the inflammation of the cerebral tissue. The ball had passed through the two frontal lobes in their middle portion, and from the very first day after the infliction of the wound there had been no signs of paralysis, the patient could speak, and had never suffered from the least hesitation in the expression of his thoughts until the cerebral softening, which caused death, supervened. Mark, gentlemen, that this autopsy was made in 1825, at a time when nothing was said about the effect of an injury to a special portion of the frontal lobes. This remarkable case proves, therefore, on the one hand, what Dr. Auburtin maintains—namely, that grave lesions of the frontal lobe may be present without inducing paralysis; whilst, on the other hand, it shows that if lesions of the frontal lobe bring on aphasia, the lesion must at all events involve a special portion, probably the one indicated by M. Broca. Professor Bouillaud's doctrine as to the frontal lobes being the seat of the manifestations of thought by speech, writing, and gesture, remains entire then, whilst M. Broca has the credit of having localized the faculty more exactly.

The following case, observed by M. Peter, at the military hospital of Gros-Caillou, may be placed by the side of the above. A drunken cavalry soldier fell from his horse, on the back of his head, and fractured his skull. Stupor set in at once, followed afterwards by the most violent agitation and delirium. The man kept constantly shouting the worst possible oaths, and held connected conversations with imaginary persons. He died at the end of thirty-six hours, without having recovered his reason. On dissection, a fracture of the roof and base of the skull was found in all its length. The most remarkable fact was, that, although the man had fallen on the back of his head, as was shown by the bruising of the soft parts and the starred fracture of the occiput, the brain was not injured at that part; whilst its anterior lobes were in a pulpy condition, through a most violent contusion evidently caused by the knocking of the cerebral mass against the anterior portion of the cranial vault. The whole thickness of the lobes was disorganized, and the alteration extended on each side as far as the anterior origin of the furrow of the olfactory nerves. This case again showed that the two frontal lobes may be destroyed in their anterior portion, without causing a loss of the faculty of speech.

It is important to inquire whether the *intellect is damaged* in aphasic individuals, and to what extent it is so; but it is difficult to estimate this. A remarkable fact is, that such patients have usually an intelligent look, and, by a few gestures, supply the absence of spoken expressions which they cannot command. In order to estimate their intelligence, then, we can only use as our guides the expression of their face, writing, and gesture. The face, as I have already told you, does not differ much from its usual aspect, and so far, it would seem that the intellect is unimpaired; but to this I must oppose this remark. It must have often occurred to all of you to speak to a dog, and to question him as it were. You must, certainly, have been struck then with the bright look, the vivacity, and the singularly intelligent expression of the animal; with the manner in which he moves his head, and often also with the low cries, the emphatic grunts, with which he accompanies this pantomime. You must often have talked to him, and often exclaimed, "He only lacks speech." Well, gentlemen, apply this remark to an aphasic individual, and you will be convinced that there is less expression in his face than in that of a dog, and you will then admit that some other signs are required for judging of a man's intelligence.

Writing can help us; but most aphasic individuals are paralyzed on the right side, and cannot write. If they learn to write with the left hand, however, it can be easily seen that they cannot write a greater number of words than they are able to articulate. You have seen the many trials to which I subjected my patients. The young man Henri Guénier could sign his name with his left hand, when asked to do so; Paquet could do the same; but you may remember that Guénier's vocabulary consisted of two words, *yes* and *mamma*, and when I asked him to write down *yes*, or *mamma*, he always wrote his name. He had, with great pains, been taught to sign his name, and the motor muscles of his hand had got used to it, in a sort of automatic manner, and continued to act in the same way when he was asked something else. Paquet, also, signed his name well with his left hand, and when he was asked to write down *fork*, he still signed *Paquet*. I made him copy the word *ward*, which was printed on his card, and he did so; with some hesitation, it is true, but still he managed to do it. But when I took away the card, and then asked him to write down *ward* again, he wrote *Paquet*.

You will agree with me that such limited manifestations indicate great weakness of intellect. You may also remember the patient whom Dr. Lancereaux brought us. This man boasted of possessing an unimpaired memory, of being still able to read, or, at least, of understanding perfectly all that he read, and to be in full possession of his intellect, speech alone failing him according to his account. I asked him to read a letter which began in these words: "My dear master," and he read without hesitation, "Sir," and then stopped short. He mumbled a few incoherent words, as if he endeavored to decipher characters which had no meaning for him; then seeing by chance the word "miss," he read "madam." It was evident that he could not read. I asked him to write the word "sir," which he had just read by mistake in the letter, and he slowly wrote his own name. I next tried to make him read the preface of a "History of St. Geneviève;" and instead of "preface," he at first said "fasts," and then was unable to decipher the first sentence: "Four centuries have elapsed since a humble shepherdess." He pronounced the word "centuries" well, said "three" instead of "four," and singularly enough, whilst he said "three," he held out four fingers, so as to help, by a gesture, his impotent speech. On my reading myself the sentence aloud, he listened with a certain degree of attention, and at the word "shepherdess," he exclaimed, with a fatuous smile: "Oh! shepherdess, know well what it is: love well shepherdess; draw well shepherdess," always leaving out the pronoun "I," which he could not articulate.

As he is a painter, a pupil of Coignet, and boasts of still drawing very well, I asked him to draw a shepherdess. After three or four minutes spent in efforts which brought big drops of perspiration on his face, he only managed to draw with a pencil unformed features, which had no resemblance to anything whatever. He succeeded, however, in drawing passably a man's head, but such as would have been drawn by a child of eight years of age, who had not learnt to draw. Here is an individual, then, who pretends that his intellect is unimpaired, who says that he can read, write, and draw well, and who cannot, in reality, decipher anything, can only sign his name, and sketch a grotesque human head. In other words, the truth is that his fingers obey an automatic impulse, to which thought remains a complete stranger.

This man's intelligence presents strange gaps; he knows, for instance, what is meant by the word "strength," and is absolutely ignorant of what is meant by "weakness," which is, however, the correlative of strength. When I asked him whether he felt weak, he did not understand me; but on putting to him the same question indirectly, by asking whether he felt less strong, he understood. As to the word "weakness," he was not only unable to articulate it, but he had completely forgotten both the word and its meaning. His sentences were, besides, of the most primitive kind, in the following style: "Me always worked,—much worked;"—"me always first,—first—first." He had forgotten a great many parts of speech. I shall revert to certain particulars of this case by and by, when I speak of the psychology of aphasia.

Memory, which is so important a faculty of the understanding, is deeply injured, as may be easily ascertained. Most aphasic patients answer very well by signs, and I have repeatedly made the following experiment in your presence. I show them a spoon, and ask them what it is. They make no answer. Is it a knife? They make a sign of denial. Is it a fork? A sign of denial again. Do you remember the name of the object which I am showing you? Denial. Is it a spoon? A very earnest sign of approval. And so it is with nearly all aphasics. This is singular, however, that although they do not remember the name of the object, they perfectly remember the use of it. When shown a spoon, and asked what it is meant for, they take and raise it to their mouth, in order to indicate its use.

M. Lordat, who holds spiritualistic doctrines, and believes in the absolute independence of thought and speech, and *à fortiori*, in the independence of thought and the organs of speech, furnishes in himself the proof of their mutual dependence. Before he was attacked with aphasia in 1828, he improvised his lectures admirably; but after his perfect recovery, he became incapable, not of improvising only, but even of reciting from memory, lectures which he had written beforehand, and he was obliged to read them.

There is no doubt, therefore, that the intellect is deeply injured in aphasia; and when the affection gets well under our eyes, as it pretty frequently happens, we witness day after day the resurrection of the faculties, and we see them progress exactly as in the convalescence of a grave disease we see physical aptitudes return day after day.

But when the aphasia is temporary, the testimony of the patients becomes very precious. Those very individuals whose intellect seems to be least impaired, have still lost some of it. Recall to mind the case of my colleague, who was aphasic for a few hours only, and who remembered so well the curious phases through which his mind has passed. He was seized while reading one of Lamartine's Literary Conversations. This is not fatiguing reading, which requires great attention, and yet he noticed that *he did not understand* well what he was reading. He put his book down for awhile, but on taking it up again, he made the same remark. On then trying to speak, he could not utter a single word, and on attempting to write, could not manage it either; yet, alarmed by these symptoms, he moved his arms, his tongue, and thus ascertained that he was not paralyzed. He even collected his thoughts, and asked himself what portion of his brain could be, at that moment, injured. His intellect, therefore, was of a greater range than that of many men, and yet it was impaired, as proved by the difficulty which he had in understanding a page of Lamartine.

You recollect the woman Keller. She seemed to have recovered her intellect, answered with ease the simple questions which I put to her, and read part of the day. But when she was thoroughly well, I asked her to appreciate herself the state of her mind during her illness. She confessed that her memory was not so good, that she did not understand so well what she was told, that she had lost much, and that she *read with her eyes* well, but not *with her stomach*, a *naïve* and singular expression, by which she meant to designate her intellectual failure, whereas the organs of the senses acted to perfection.

Adèle Ancelin, also, read the whole day, and so did Paquet; so that those among you who are the champions of the intellectual aptitudes of aphasics, bring forward, as a powerful argument, the fact that the patients read attentively. Adèle Ancelin had, for a year, the same book in her hands, a religious book, the "Month of Mary." The poor girl almost always read the same page, a fact which showed that she could not understand what she read. On several occasions, as you may remember, I took her book, and read aloud the very page which she had constantly under her eyes; and when I asked her if she understood what I read, she shrugged her shoulders to express that she did not. Paquet had received a pretty good education, since, as I have told you, he was going to be ordained when he left the seminary. He sometimes reads a whole day, and I must confess that he follows pretty well the lines in the book, that he turns the pages correctly, and seems to understand perfectly. But the following experiment proved categorically that he understood much less than he seemed to do. I took up his book, and read aloud a few lines at the bottom of a page, asking him to follow me with his eyes, and to turn the page when I came to the last line, but he never could do it correctly. Now, a child of five years of age, who can read, will turn a page properly, however limited his intelligence may be. Another circumstance shows, besides, that if Paquet understands perfectly what he reads, he has no recollection of it, and it will be easily admitted that memory is one of the most important faculties of the understanding, and that animals themselves possess it in an eminent degree. Paquet has collections of tales on the table by his bedside, which he reads over and over again, and always with the same degree of attention. Now, as a rule, one cares very little for a tale which he has already read, and it would be an unbearable torture to be condemned to read the same tale thirty times a day. So that Paquet either does not understand what he reads, or does not recollect what he has just read, both hypotheses indicating a notable impairment of the intellect. He yet plays draughts and dominoes, and pretty well too; he is even guilty of cheating, an act which requires some degree of cunning; and when his adversary finds it out and compels him to begin again, he either grows impatient or laughs as if in joke. This very man, however, who plays these games, and makes pretty learned combinations, is incapable of counting his age on his fingers.

Insane individuals possess the same aptitudes. I have always been struck with the specialty of each man's intellectual aptitudes; but I have never understood the signs of extraordinary intelligence often exhibited by individuals who are completely demented.

When I was a resident pupil at Charenton, in 1825 and 1826, I often went to the drawing-room in the evening, and played some game with the insane patients. I have always been a bad player at draughts and chess, but I was vexed to be easily beaten by persons who could not put two ideas together. I was a better hand at backgammon, but I had not better luck when I played with individuals who had been formerly very good players. I was surprised at it, and even now, when I think of it, after an interval of nearly forty years, I cannot conceive how, in a brain so deeply disorganized as that of a demented person, combinations can be formed superior to those of a man of sound mind. I should not be astonished in the case of a monomaniac, because he is delirious on a pretty limited point only, so that he may retain all the aptitude which he may have previously possessed for certain games or for calculations; but I cannot understand how the very varied combinations of a game of cards or backgammon can be made in the mind of a maniac, who seems incapable of putting two ideas together.

To sum up, then, I maintain that aphasics are, as regards intelligence, considerably beneath the generality of men, and especially considerably beneath their former selves, when the comparison can be instituted. There is, however, a form of aphasia in which the intellect is unaltered. Memory is good, the patient writes easily, and expresses his thoughts correctly in writing, as educated deaf-mutes do. This form is very rare, and it has seemed to me to differ so widely from the other, that I have thought myself warranted in regarding it as a distinct variety, particularly as in all the cases of the other form of the disease, the inability to write is proportionate to the inability to speak. The following case struck me the most.

I received one day, in my consulting-room, a carrier of the Paris Halles, very young, and having the appearance of a man enjoying excellent health. He made signs that he could not speak, and handed to me a note, in which the history of his illness was detailed. He had written the note himself, with a very steady hand, and had worded it well. A few days previously he had suddenly lost his senses, and had been unconscious for nearly an hour. When he came round, he exhibited no symptom of paralysis, but could not articulate a single word. He moved his tongue perfectly—he swallowed with ease, but, however much he tried, he could not utter a word. Thinking that faradization might be of some use to him, I transferred him to my friend Dr. Duchenne (de Boulogne). He was ineffectually galvanized for a fortnight; but, without any special treatment, he completely recovered his speech five or six weeks after the invasion of the complaint. It is very remarkable, however, that during the whole course of this singular affection, he could manage all his affairs, continue them even in a certain measure, by substituting writing for speech.

The following interesting case is something like the above. A lady residing at Boulogne-sur-Mer, was for ten or twelve years an object of curiosity in the town and the subject of every conversation. She had a very peevish temper, and the country people said that she was bewitched, on account of her ill nature. After an accident (the nature of which I have not been able to ascertain), she lost her speech, and only retained the faculty of uttering the oath, "Sacré nom de Dieu," by which she expressed all her thoughts, whether sad or gay. The curious point about her was, that for a great many years she was able to manage some rather important business, and keep her house in very good order. She went to market herself, made her own purchases, bargaining for them by signs, and occasionally exclaiming, "Sacré nom de Dieu." It never occurred to any one that she was insane, and her friends never tried to obtain her interdiction, although they may have perhaps desired it. She was not paralyzed. I do not know whether, like the carrier whose case I have just related to you, she could write, and thus express her thoughts in writing.

There is another form of aphasia which is sometimes met with after acute diseases, and which is characterized by the complete forgetfulness of words. The following is a remarkable instance of this:

Madame M——, who usually enjoyed excellent health, and was endowed with a very remarkable intelligence, was attacked at the age of fifty-six with erysipelas of the face and scalp. For a few days somewhat grave cerebral symptoms manifested themselves, and when the fever disappeared she had completely forgotten all words. For several days she remained in a sort of automatic condition, accepting food and drink without asking for them, and giving expression to no thoughts. After a few days she could repeat, using them in their proper sense, words which were told her. Shortly afterwards she began to put a few words together, so as to construct parts of sentences or very short sentences: at that time she had completely recovered her physical health. At first she merely repeated the words which she was told, but after a time, her memory, by degrees, suggested others to her. She asked for paper, pen, and ink, and spent several hours every day, for a period of three months, writing down all the words which she could remember. I have seen her manuscripts, and it was curious to observe by what process one word reminded her of another; sometimes the first, sometimes the second, syllable gave her the key to the next word. The rhyme often suggested words to her, and sometimes a very distant meaning. The following examples will illustrate this: "Cat, hat; hand, sleeve; gown, petticoat; rose, nose, nosegay; beer, froth; rope, well, ditch," &c., &c. She thus covered nearly five hundred pages with small text.

My colleague, Dr. Boucher, Professor of Medicine in the Dijon Preparatory School of Medicine, has since observed two cases of this kind, in the course of an epidemic of typhoid fever which desolated the town in 1863.

The son of the porter of the Imperial Lyceum at Dijon, aged thirteen, of a delicate constitution, was seized with fever in the month of September. His life was in danger for some time, but the symptoms became more favorable at last, and all was going on well, when complete aphasia supervened one morning. It was a sad, though curious sight, to see the supreme efforts of the child to articulate the simple word "no." Dr. Boucher tested the urine for albumen, and detected its presence in a small quantity. As the general symptoms, however, continued to be good, tonics and proper nourishment were insisted on. After four or five days the words returned by degrees, although enunciated with remarkable slowness; but at length complete recovery took place, and the boy, after a somewhat lengthened convalescence, resumed his studies at the school.

The second case observed by Dr. Boucher was that of a child aged three years, who had presented very grave nervous symptoms during an attack of typhoid fever, and who likewise passed albuminous urine. Speech was also lost suddenly, when the fever ceased to be dangerous: convalescence was very much prolonged.

You probably remember the case of a woman who was in my wards in the year 1863, and in whom we observed, after she had had a serious attack of typhoid fever, symptoms exactly like those described by Dr. Boucher.

It is not a rare thing to meet with paralytics who cannot distinctly articulate, who sputter out their words, and whose tongue is so embarrassed as to render them incapable of expressing the few thoughts which they have. With a little attention, however, it is easy to see that a particular intonation corresponds to each thought, so that the persons about such patients understand pretty well, after a time, these imperfect grunts. The patients use correct words for answering questions, but the paralysis of the organs of speech prevents them from articulating distinctly. The same thing occurs in glosso-laryngeal paralysis. In ordinary paralytics the intellect is deeply damaged, but in glosso-laryngeal paralysis the intellect is unaltered, the patient can read and write, and it can be easily seen that, when they try to speak, their eyes and gestures make up for the incompleteness of their speech. They have, therefore, at the command of their intellect, all the manifestations which a healthy man can dispose of, save speech, the impediment in which is proportionate to the degree of paralysis of the organs which are used for articulate language.

I can well imagine that all these distinctions appear somewhat subtle to persons who read books without seeing patients; but when they are studied at the bedside, they are so strikingly manifest, that even those among you who have just begun their medical studies catch at once the shades which separate affections which at first sight appear to be identical. There is another curious character by which the ordinary paralysis of aphasia may be distinguished from glosso-laryngeal paralysis, and the loss of speech which is a consequence of the total loss of memory; namely, the excessive emotional sensibility which is usually found in true paralytics. A person who has become hemiplegic after an attack of cerebral hemorrhage is constantly shedding tears, for the least thing; and this is a symptom which has been noted by all observers. But in aphasia this symptom is in most cases absent, and I confess that I cannot conceive the reason why. This proneness to shed tears is commonly ascribed to the grave impairment of the intellect, which usually accompanies paralysis; but in cases of aphasia, there is as deep an impairment of the intellect, and yet there is more rarely found this tendency to whining. I lately saw, at the Maison Municipale de Santé, with my colleague Dr. Bourdon, a merchant suffering from complete aphasia, and who was easily moved to tears. There were two things in his case, however; for he had had two successive attacks of paralysis, one on the left, and the other on the right side. He had become aphasic after the second attack, and since the first seizure, which had presented the same symptoms as the generality of cases of cerebral hemorrhage, he had been prone to shed tears on all occasions, and this had not disappeared since he had become aphasic.

In aphasia, the inability to speak depends on very various causes, which it is very difficult to analyze well. There is, in the first place, *amnesia*, as can be seen at a glance, and, in most cases, this is even the most prominent symptom. The patient does not speak, because he does not remember the words which express ideas. You recollect the experiment which I often repeated at Marcou's bedside. I placed his nightcap on his bed, and asked him what it was. But, after looking at it attentively, he could not say; and exclaimed, "And yet I know well what it is, but I cannot recollect." When told that it was a nightcap, he replied, "Oh! yes, it is a nightcap." The same scene was repeated when various other objects were shown to him. Some things, however, he named well, such as his pipe. He was, as you know, a navvy; and, therefore, worked chiefly with the shovel and the pickaxe, so that these are objects the names of which a navvy should not forget. But Marcou could never tell us what tools he worked with, and after he had been vainly trying to remember, when I told him it was with the shovel and the pickaxe, "Oh! yes, it is," he would reply, and two minutes afterwards he was again as incapable of naming them as before. There exists, therefore, a loss of memory sufficiently great to prevent the patient from being able, of his own accord, to designate an object by its proper name, but not sufficient to prevent his remembering the name when uttered in his presence. Thus, this same patient, Marcou, who could never spontaneously tell us the name of his nightcap, recognized its name perfectly, however, when mentioned in his presence. When it was shown him, and was called a pipe, or trousers, he exclaimed, "Oh! no," whilst he replied "Yes" when asked whether it was his nightcap. The condition of such patients is, therefore, analogous to that of a schoolboy whose memory fails him when reciting a lesson. If the master prompt him, he begins again, and continues without a mistake, provided that he is endowed with a certain degree of memory; but if he has no memory at all, every word has to be told him. On this condition alone can he recognize and repeat the words which he has learned, or tried to learn. In some cases, memory is so impaired that the patient does not feel positive as to the name of an object which is shown to him. Paquet, as you know, was very deficient in this respect. When told that he was wrong, after he had nodded approvingly on an object shown to him being called by its correct name, he hesitated, and looked perplexed. Marcou, however, was never shaken in his confidence.

It is undeniable, therefore, that there is a loss of memory, and this symptom sometimes constitutes the only morbid phenomenon. In the month of January, 1864, one of my colleagues in the Academy of Medicine was seized, on returning home, with slight vertigo. He tried to speak, and found that he could not remember a great many nouns! Drs. Pidoux and Const. Paul, who were sent for, easily recognized this singular disturbance of the intellect. The patient suddenly stopped in the middle of a sentence, unable to express a noun; he hesitated, manifested impatience, and if the word he wanted was pronounced by another, he exclaimed, "That's it," and repeating the word very distinctly, he continued his sentence. In this case, then, most of the parts of speech were perfectly remembered, but nouns were completely forgotten. Let us note, however, for it is an important fact, that the patient caught the word at once, as soon as it was pronounced, and articulated it with extreme facility.

But there is, in aphasia, another very strange phenomenon, which is, perhaps, another form only of amnesia; it is the inability to articulate words, however hard the attempt. Paquet, for instance, can say *cousisi*, and it seems, therefore, that he might easily say *cou-cou*, or *sisi*. But you saw that for several days in succession, I stopped a good while by his bedside, and that it was only after a few days that I could succeed in making him say *cou-cou;* whilst he never could say *sisi* alone. The same remark has already been made by Dr. Perroud, physician to the Hôtel-Dieu at Lyons, who had under his care an aphasic woman who could say very well, "Bonjour, monsieur," but could never be made to say *bonbon*, a word which is, after all, the repetition merely of the first syllable in *bonjour*. You saw, also, what pains I took to make Paquet repeat a few syllables. He could say *a* pretty easily, but could never say *pa*. I told him to imitate the movements of my lips as I pronounced the letter *p*, but he could not succeed in doing so. I tried again to make him say at first *peu a*, in hopes, after a time, to make him by elision contract the two sounds into the single one *pa;* but I failed completely.

It would seem, therefore, that there is, in these cases, an inability to co-ordinate the movements which are needed in phonation; for the patient has fully retained the power of moving his tongue and lips in every direction, as may be easily ascertained, although he is incapable of making combined movements in order to enunciate a word. I have asked myself whether there was not merely forgetfulness of the instinctive and harmonious combination of movements which we all learn in infancy, and which constitutes articulate language; and whether an aphasic patient was not, consequently, in the same condition as a child who is taught to speak for the first time, or of a deaf-mute, who, on being suddenly cured of his deafness, tries to imitate the speech of the persons whom he hears for the first time. There would then be this difference between an aphasic and a deaf-mute, that the one has forgotten what he has learnt, whilst the other does not yet know. I am the more inclined to believe that this opinion is well founded, because there is, in nearly all cases, loss of memory of writing as well as of speaking. In general an aphasic individual is not more apt to express his thoughts in writing than in speaking; and although he has retained the power of moving his hands as nimbly as before, he is yet as unable to compose a word with his pen as he is to articulate it. Now, in such a case, it is impossible to admit a defect of co-ordination, whereas loss of memory explains everything.

Another mode of expression of one's thoughts, namely, by gestures, is as deeply modified, in many cases of aphasia, as speech itself. Now, when an individual moves about with the greatest ease, when his face is variously agitated according as he is under the influence of joy, surprise, or pain, it is strange that his face should be incapable of feigning the expression of these same feelings when they are not really felt. Thus, an aphasic cannot, when asked to do so, put on the face of a person who is crying, and his inability cannot be due to a defect of muscular co-ordination, since, when he feels real grief, the expression of his face clearly shows it. You saw me make the following experiment with Paquet: I held out my two arms and hands, and moved my fingers, like a man who is playing the clarinent, and I asked him to imitate me. He immediately executed the same movements with perfect precision. When asked whether he knew that the attitude was that of a man who played the clarinet, he replied affirmatively, by nodding his head. A few minutes afterwards, however, when asked to put himself in the attitude of a man who is playing the clarinet, he seemed to think, and, in most cases, was unable to reproduce this easy pantomime. There was amnesia, then, in his case, for he did not recollect.

The illustrious Professor Lordat, who has himself suffered from aphasia, gave, after his recovery, an account of the inward sensations which he felt during his illness, which perfectly indicates the part played by memory. He could think, he could co-ordinate a lecture, or change its arrangement in his own mind, but he was unable, although he was not paralyzed, to express his thoughts in speaking or writing. "I thought," says he, "of the Christian doxology: 'Glory be to the Father, the Son, and the Holy Ghost,' and I was not able to *recollect a single word of it.*" Thoughts seemed to arise freely, but the mode of expressing them in sounds, the receptacle of these thoughts, was forgotten. In the experiment which I related to you just now, I evidently awakened Paquet's recollections, but he could not express them by gestures. I am anxious to give prominence to the fact, that in aphasia loss of memory plays the principal part; the patient forgets, wholly or partially, the various modes of expressing thoughts, and is in the condition of a deaf-mute, who, on suddenly gaining the faculty of hearing, does not yet know how to use the organs of phonation.

But does it follow that *aphasia* and *amnesia* are, in my opinion, synonymous? Certainly not. The aphasic patient, who has forgotten how to express his thoughts in speaking, in writing, and by gestures, often remains apt to form the difficult combinations needed in the games which tax memory much. They remember perfectly circumstances which occurred long ago; and you have heard how Professor Lordat, and that colleague of mine whose case I related to you, recapitulated, mentally, series of very complex ideas, although, according to all appearances, their intellect was perhaps not so clear as it had previously and as it has since been. Yet the fact of broad conceptions cannot be contested, of conceptions of a higher order, certainly, than those of uneducated and ignorant men, who, nevertheless, express themselves with readiness. It is assuredly very strange, that men who are evidently endued with a pretty extensive memory on certain subjects, should be completely devoid of all memory when they have to express their thoughts in speaking and writing, and by gestures.

You remember the stratagem practiced by the hospital employés for finding out Marcou's name and address; you remember how he walked back to the stone-yard where he usually worked, so that he must have recollected localities. Again, you have seen Paquet play dominoes and draughts, remembering pretty difficult combinations, although, for the last eight months he has only been able to say *cousisi*. Dr. Lasègue knew a musician, who was completely aphasic, and who could neither read nor write, and yet could note down a musical phrase sung in his presence. Whatever be the share, therefore, which I am disposed to ascribe to loss of memory in aphasia, I am compelled to admit that certain special kinds of memory are untouched. This statement may appear strange, for it might seem that there is only one kind of memory. But it is not so.

I knew a medical student, a good musician, but of ordinary intelligence in other respects, and not endowed with a retentive memory, who could, on his return home from the opera, play on the violin all the airs of an opera which he had heard for the first time. You may have heard of Mondheux, the young shepherd in Touraine, who had such a wonderful memory for figures and calculations, that, if he were unexpectedly asked how many hours had lived a man aged forty-five years, four months, and five days, he could, in less than two minutes, give the correct answer, without having recourse to a pencil or a pen. His memory was, in other respects, of very ordinary range, and when he was a little older he evinced no aptitude for mathematics. One man has a good memory for localities, another for names, a third for dates or for figures, and each of these *varieties* of memory, if I may be allowed the expression, is independent of the rest. So that, if it were admitted that an aphasic patient suffers, after all, from loss of memory, it should be added that he has lost all recollection of the mode of expressing thoughts in writing, in speaking, and by gestures.

Now, I do not know how to classify those cases of aphasia in which the patient is able to express himself in writing, though not in speaking, as in the case of the carrier which I have related to you. We must, in such instances, suppose that the patient no longer remembers the movements which are needed for producing and modifying vocal sounds. Thus, the man whom I saw with Dr. Duchenne, and who could give in writing proofs that his intelligence was still of a good order, could not even articulate the syllable *ba*. He moved his tongue and lips perfectly, he swallowed as well as before, and yet, when we stood before him, and asked him to say *bon*, to close his lips as we did, and then to utter the sound *a*, as we opened our lips, he made the most curious grimaces, but could not succeed in saying *ba*. Now, this very man, when eating or drinking, approximated or separated his lips with perfect regularity. There was, consequently neither loss of power nor disorder; this occurred only when he attempted to execute a determinate movement, as in the act of speaking; just as there are loss of power and disorder in cases of what Dr. Duchenne has called *functional spasm*.

Such patients, indeed, use their right hand very well to shave themselves, to play the piano, to sew, to pick up the most minute objects, but as soon as they try to write, the muscles of their hand instantly become affected with spasm, and their pen can only trace illegible characters. Again, a violin-player may write perfectly, but he either cannot hold his bow, or the hand which holds the violin is the seat of spasmodic contractions. Is this the case in this particular form of aphasia? It may be objected that there is no spasm of the vocal organs in aphasia, but loss of the aptitude in virtue of which the numerous organs concerned in the production of voice (the lips, tongue, soft palate, glottis, and the various parts of the larynx) act in harmony, in order to produce determinate sounds. It may be objected that there is loss of this complex co-ordination, which seems to us natural and easy only because we have forgotten the time and trouble which it has cost us; in other words, that there is what Dr. Lordat so justly terms *verbal incoördination*. There is not incoördination only, but *verbal amnesia* also; for the patient has lost the memory of words. Yet, has he forgotten words alone, apart from the ideas which they express? This question involves one of the most intricate problems in metaphysics; namely, whether ideas can exist independently of the words which represent them. I will not presume to setttle definitively this problem, which has been solved in two opposite ways by spiritualists and sensualists, but will merely confess that I incline to the opinion advocated by Condillac and Warburton, that words are necessary, nay, indispensable instruments of thought.

I cannot, therefore, indorse Professor Lordat's view, that thought is absolutely independent of speech, and that a subject may be limited, may be developed and divided into elementary ideas, which, in their turn, may be subdivided into simpler ones, although all recollection has been lost of the sounds which are used as signs, that is to say, when all memory is lost of the words composing language.

In support of his theory, Dr. Lordat quotes his own case. Although, seized with aphasia, he could, says he, combine abstract ideas, distinguish them accurately, *although he could not command a single word to express them, and without in the least thinking of this mode of expression.* "He was conscious of no impediment in the act of thinking. Accustomed to teach for a great many years, he congratulated himself on being able to dispose in his mind the chief points of a lecture, and on having no difficulty in introducing any changes which he liked, in the arrangement of his ideas."

But the celebrated professor will allow me to ask him whether he did not deceive himself? and whether he was not in the same condition as that patient of Dr. Lancereaux's whom I mentioned a short time ago, and who also believed that he was in full possession of his intellect? Yet, on putting it to the test, his intellect was clearly shown to be impaired; thus proving that he had not merely lost the physical faculty of the material transmission of his thoughts.

Doubtless the mind may wander a little at random, without being obliged to give a corporal form to its ideas; but as soon as it tries to make them concrete (a condition which I regard as *indispensable for their co-ordination*), it seems impossible to me, as least in my own case, not to clothe them in their material dress,—namely, words.

It will, doubtless, be objected that a deaf-mute evidently thinks, before he is taught the gestures by means of which he will henceforth hold communion with his fellow-men and improve his mind. But is it clearly proved that an untaught deaf-mute is apt to form conceptions of a very high order? Is it not probable that he makes use, even for the *elementary* ideas of which he is capable, of the material images of things instead of words, the images of ideas, as we do? Is it not probable that, when he thinks of a tree, for instance, he pictures to himself a tree by the image of the tree itself, instead of thinking, as we do, of the word *tree?* See, then how inferior is the memory of an untaught deaf-mute, when compared with ours. Like him, we can remember a tree by the image of a tree, but we are also reminded of it by the word *tree*, which pictures itself to our mind, because we have read and written it. One may imagine how fettered thought must be in such cases, and how rudimentary intellectual conceptions must therefore be. A great thinker, as well as a great mathematician, cannot devote himself to transcendental speculations, unless he uses formulæ, and a thousand material accessories which aid his mind, relieve his memory, and impart greater strength to thought, by giving it greater precision. Now an aphasic individual suffers from verbal amnesia, so that he has lost the formulæ of thought.

I believe that the same thing obtains in metaphysics as in geometry. In the latter case a man may vaguely conceive space and infinity without any precision or measure, but if he wishes to think of the properties of space, and more particularly of the special properties of the figures which bound space, as, say, conic sections, it is impossible that his mind does not immediately see the curves proper to a parabola, an hyperbola, and an ellipse. In metaphysics, on the other hand, I believe that a man cannot think of the special properties of beauty, justice, and truth, for instance, without immediately giving a material form, as it were, to his thoughts, by using concrete examples, and without associating words together—words which represent concrete ideas, and which then stand in the same relation to particular metaphysical ideas as figures do to determinate geometric ideas.

In aphasia, therefore, there is not merely loss of speech, but there is also impairment of the understanding. The patient has lost simultaneously, in a greater or less degree, the *memory of words, the memory of the acts by means of which words are articulated*, and *intelligence*. But he has not lost all these faculties in an equal degree, for the understanding is less injured than the memory of the acts for producing sounds, and this latter faculty less impaired than that of remembering words.

To sum up, aphasia consists in loss of the faculty of expressing one's thoughts by speech, and in most cases, also, by writing and by gestures.

As every distinct faculty presupposes a special organ, the advocates of localization made out that the seat of this faculty in the brain is the posterior portion of the third frontal convolution, chiefly on the left side. But the most varied lesions of this spot, and I will add of neighboring parts, more deeply situated, such as the insula of Reil and the corpus striatum, can bring on aphasia. Hence the same *prognosis* does not apply to aphasia occurring during convalescence from a grave fever, and to aphasia due to softening of, or hemorrhage into, the brain. In the former case we can scarcely conceive the nature of the lesion, and it must be confessed that this form, which is essentially transitory, differs greatly as to its progress and termination from the persistent affection of which I have given you illustrations. In those cases where the affection is transitory, or is more or less prolonged, but is *unaccompanied by hemiplegia*, there may perhaps have been mere congestion. But however this may be, these two forms must be carefully distinguished from a third, in which there is concomitant hemiplegia, for this last is, in most cases, absolutely incurable, or at best admits of very slight amelioration only. Another fact which should be made prominent, is the frequent termination of aphasia in rapidly fatal apoplexy. This occurred in the instance of the woman Desteben, in that of the gentleman whom I went to see in the department of Landes, and in that of another patient whose case was mentioned to me.

It is undeniable that bloodletting has been immediately followed by happy results, but only in cases of aphasia without hemiplegia, that is to say, when the lesion was not probably very deep. In cases of aphasia with hemiplegia, unless dependent on syphilis, as in Marie K——, I must confess that we are almost completely powerless. We can no more cure the aphasia than we can the paralysis which accompanies it. Nature alone, or nearly alone, brings on improvement, which is in all cases merely partial. The intellect of an aphasic patient is forever damaged, just as the motility of one half of his body is impaired, and he will always be lame mentally.

LECTURE LXII.

SPERMATORRHŒA.

Local Phenomena.—General Symptoms.—Symptoms which may be mistaken for Manifestations of very different diseases from those to which they belong.—Spermatorrhœa depends on Different Causes.—Spermatorrhœa consequent upon Chronic Irritation of the Urinary Passages and Rectum.—Spermatorrhœa from Excessive Contractility of the Vesiculæ Seminales.—Spermatorrhœa from Atony of the Ejaculatory Canals.—Treatment must vary according to the Nature of the Cause.—Treatment of the two last-mentioned forms by Compression and the Topical Application of Heat and Cold according to the Indications.

GENTLEMEN: By *involuntary seminal emissions* or *spermatorrhœa*, we understand those losses or evacuations of seminal fluid which either take place without any, or with inadequate erotic excitement. In the normal state of a properly constituted person, the emission of seminal fluid requires not only that the venereal orgasm should be carried to a very high degree, but also that a series of acts be repeated for a longer or shorter time: there is required the mechanical act of copulation, or the use of some other analogous means. Amorous desires, be they ever so keen, occurring even in the strongest and most continent persons, do not in general cause spontaneous ejaculation of semen: nor is ejaculation brought about by mere contact with the object of desire. When ejaculation occurs independent of the erotic excitement generally required, there is an involuntary loss of semen. Bear in mind, however, that, using the term in a wide acceptation, I for the moment place in one general group very different degrees of the affection, beginning with nocturnal pollution properly so called, an occurrence which in many cases is not morbid, and culminating in spermatorrhœa, the malady upon which I propose now to address you.

The pollutions which spontaneously occur in persons who are too continent, supervene during sleep under the influence of lascivious dreams accompanied by erection and a high degree of venereal orgasm, do not generally come within the province of the physician, inasmuch as they generally indicate an excess of health and vigor, rather than a state of feebleness and disease. The individuals who have these emissions usually experience on awaking a general feeling of comfort succeeding to the uneasy sensations by which they had often been previously tormented; they feel themselves freer, more buoyant in spirits and more at their ease; they are in the position of persons who have satisfied a physical want. I ought, however, to remark, that nocturnal pollutions in chaste persons in good health occur much less frequently than is commonly believed. A very vigorous man may entirely abstain from women for months without having nocturnal pollutions; and, speaking generally, they ought to be of rare occurrence, excepting perhaps during the first years of manhood. Should they take place every month, still more, should they occur every fortnight, or every week, should the loss of semen happen in this way even less frequently than in those who resort in moderation to coitus, there will nevertheless be some bad effect produced: and although in the morning after the pollution, there is a feeling of wellbeing, the individual is on a decline which may lead to bad health. In fact, the seminal emission, under the influence of different causes, and even from mere habit, soon becomes a pathological occurrence: it recurs at intervals less and less distant, it ceases to be active and becomes passive, that is to say, happens without dreams, without erection, and without any erotic sensations being experienced. Ere long, the individual will cease to be conscious of what took place during the night, and would remain quite unaware of the occurrence, were it not for the visible traces of the semen. These are the pollutions which constitute the first degree of the malady we are now studying.

Almost always, if not always, spermatorrhœa properly so called begins with nocturnal pollutions. They have in the first instance been the result of erotic dreams: they recur frequently: through habit, the frequency increases to such an extent that they take place not only once every night, but several times in the same night. At a more advanced stage of the disease, the seminal emission occurs without erethism, and also without erection, by which, in the first instance, it used to be preceded and accompanied. At all events, the patients are quite unconscious of having experienced any voluptuous sensations, and it is only on awaking that they become aware of what has happened to them when asleep.

Then also, in place of experiencing a feeling of wellbeing, they feel out of condition and weary: they complain of heaviness of the head, and a certain amount of mental wandering and incoherency of ideas: they are not in a fit state to work either with mind or body.

After the lapse of a certain time, there are diurnal as well as nocturnal pollutions. At first, there is still the necessity of a certain degree of orgasm, but an imperfect erection lasting only for a short duration will suffice to cause emission. If the patients copulate, ejaculation takes place immediately: it sometimes happens that the introduction of the penis has hardly began, when the venereal act ends, and the erection suddenly ceases. At a later period, still less will be required: mere touching or rubbing, such as occurs in riding on horseback, or in the movements of a carriage or swing, and at other times, the mere sight of objects suggestive of lascivious ideas will be the immediate determining causes of a greater or less flow of semen.

Under all circumstances, even in those cases in which the nocturnal pollutions are compatible with perfect health, involuntary seminal emissions take place under the influence of a sort of erotic excitement, but an inadequate erotic excitement, as compared to that which there ought to be, were the ejaculation accomplished in a normal manner. But when the spermatorrhœa reaches an advanced stage, the seminal emissions take place without there having been the least previous excitement.

This kind of seminal emission may occur, however, irrespective of the disease of which I am now speaking, but it is then only a transient and unimportant symptom. For example, an individual, generally in good health, becomes affected with obstinate constipation. When at stool, he has a seminal emission: it is an unimportant mechanical phenomenon; for in this case, the seminal emission is the result of pressure upon the seminal vesicles by the excrement which the energetic intestinal contractions are expelling.

This is a fact which we do not require to stop to consider: but it is otherwise in respect of spermatorrhœal patients having habitual seminal emissions not only in the act of defecation, but also during micturition. In the first referred to class of cases, the seminal flux only takes place in small quantity, and under the influence of a violent effort, while in the latter it supervenes when there is no straining—when the motions are diarrhœal—as well as when they are hard and solid.

The emission of urine will excite the emission of semen, which will come sometimes with the first, but more generally with the last portion of urine. Lallemand (of Montpellier), to whom, as you know, we are indebted for the most complete inquiry which has been made into this subject, says that seminal emissions supervening during micturition are the most serious in their character, and the most rebellious under treatment: they are also the most difficult to recognize, in consequence of the change produced on the semen by its mixture with the urine.*

Lallemand, however, has pointed out certain physical and microscopical characters which may be of use in the diagnosis of such cases. Sometimes, the patients themselves perceive that there is a change in the appearance of their urine: they observe that the last drops passed are thick, glutinous, and viscid, liable to form small curdled clots, which stopping at the entrance of the meatus, acquire a sticky starchy consistence, and leave a mark on the linen like that caused by the starch used in the laundry.

If the urine in the vessel is examined as soon as it is passed, small bodies, may be seen rolling at the bottom of the fluid, which are variable in size, semi-transparent, irregularly spherical, and in appearance very much resembling grains of semolina. These bodies are soft, and do not adhere to the sides of the vessel: they appear in the urine before it has cooled, and in urine which is quite clear—a circumstance which prevents the semen being mistaken for a deposit of urinary salts.

When the malady has advanced still farther, the characters now indicated are absent. The urine does not deposit granular bodies sufficiently large to collect at the bottom of the vessel, but it contains a thick, homogeneous, yellowish cloudiness studded with small brilliant points occupying the inferior strata, and resembles the deposit which forms in a concentrated decoction of barley or rice. Lallemand says that the presence in the urine of these granular bodies leaves no doubt as to the nature of the cloudiness in which they are observed. Certain precautions have to be taken to enable us to lay hold of the characteristic appearance of which I am now speaking. Urine passed at different periods of the day does not always present the same appearance, so that what is passed at each micturition ought to be kept in a separate vessel. Generally speaking, the urine voided in the morning, particularly when the patient has passed a bad night, are the most loaded: at other times, the urine most loaded is that voided after venereal excitement, violent mental emotions, or when digestion has been difficult. Sudden chilling of the body may produce the same effects. During the day, the urine is generally clear.

By the assistance of the microscope, Lallemand ascertained that the cloudiness of which I have been speaking is in a great measure due to the presence of semen mixed with the secretion from the mucous membrane of the urinary passages, and that the shining points in the cloudiness consist of the matter excreted by the seminal vesicles. A fact still more interesting has been elicited by microscopical inquiry, viz., the altered state of the spermatozoa in patients affected with spermatorrhœa. The animalcules decrease in number and volume, and in very severe cases, assume a spherical form: moreover, their vitality diminishes as the disease advances.

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* LALLEMAND: Des Pertes Séminales Involontaires. Paris, 1836-1842. Three volumes, 8vo.: published in five parts.

Gentlemen, I have told you that the patients themselves sometimes perceive a physical change in the character of the urine: I would now add that some of them complain of experiencing at the time of micturition certain sensations which announce the occurrence of the pollutions. There is felt a peculiar grazing sensation caused by the passage of the urine, and arising from its unusual density: there are spasmodic contractions: there is pain extending from the neck of the bladder to the glans penis and margin of the anus: there is also a shiver accompanied by a general feeling of discomfort. Lallemand states that those who are accustomed to these peculiar coincidences know that they will find in their urine a flocculent deposit containing the granulations of which I have been speaking. So complete is their conviction on this point, that they immediately experience a cold sweat accompanied by faintness.

Irrespective of the changes in the urine due to the presence of semen, there are also others which are pretty frequently met with. They are connected with certain complications, which often accompany spermatorrhœa. These complications are acute and chronic cystitis; inflammatory affections of the prostate gland, ejaculatory canals, and even of the seminal vesicles—inflammations leading to the formation of morbid mucous secretions, which explain the presence of mucus, purulent mucosity, and pure pus, all contributing to produce the cloudiness of which I have spoken.

These coincident inflammations also explain a certain number of the symptoms complained of by the patients, such as discomfort and weight in the hypogastric, perineal, and anal regions—pain accompanying any slight overexertion, walking, and riding, which are acutely felt even after sitting a long time.

We have now reached the question: What are the symptoms which usually bring in their train involuntary seminal emissions?

Lallemand, though he possibly exaggerated the disastrous consequences of this disease, is certainly entitled to the merit of having in a special manner called the attention of the profession to this subject; and no one more than the illustrious physician of Montpellier has diffused information on this point, so important both in the science and art of medicine. He thought, and correctly thought, as you will see immediately, that spermatorrhœa was the cause of certain diseases of the nervous system and affections of the moral and intellectual powers [*de vésanies*]; but unquestionably he exaggerated, attributing to involuntary seminal emissions some affections not in any way originating in this cause. He has not sufficiently recognized the fact, that spermatorrhœa is not necessarily the cause of the different neuroses described in his book, but is in many cases only the expression of a nervous disorder which first shows itself as spermatorrhœa, and then assumes forms of a much more serious character.

Before entering into this part of our subject, let us recall what takes place after the act of copulation. Coitus is succeeded by a state of depression and weariness: if inordinately repeated, there is a still greater state of bodily prostration, the intellectual powers are blunted, and the mind temporarily loses its accustomed energy. Cessation of erection is, however, the immediate result of the accomplishment of the venereal act. Whenever ejaculation has taken place, erection ceases more or less quickly in most animals as well as in man: a certain time is required to recruit the strength; and to restore the erection, new excitement is required. In a word, frigidity [*frigidité*] is the immediate result of ejaculation, a frigidity, of course which is relative, and, under normal conditions, temporary. Thus it becomes easy to understand how it is that involuntary seminal emissions recurring at short intervals have a principal tendency to lead to absolute frigidity, and finally to impotence.

Fully understand, gentlemen, that I attribute this impotence to the state of feebleness which is associated with habitual loss of semen, and not as do many physicians to the shaking of the nervous system accompanying the venereal act. In support of my opinion, allow me to enter into some explanatory details.

It has been alleged, I said, that the feebleness and prostration which succeed ejaculation, or the last stage of the venereal act, to speak more correctly, depended upon the shaking of the nervous system, experienced during the accomplishment of that act. In my opinion, however, that is an element which only to a small extent enters into the causation of the exhaustion.

Reflect on what takes place in the woman. In her, the excitement of the nervous system, what is called the cynic spasm, is quite as energetic as in the man: often, it is even much stronger, and nevertheless a woman can, generally speaking, engage in copulation, and repeatedly accomplish a complete venereal act within a very short space of time, at much shorter intervals than a man could, and yet without her experiencing extreme fatigue or much exhaustion of strength. It follows, therefore, that the cynic spasm, the shaking of the nervous system, which accompanies coitus, cannot be regarded as the principal cause of the debility and exhausted state induced by it; and consequently, cannot be looked upon as the principal cause of the frigidity and impotence, which must be attributed to the loss of seminal fluid. If we turn to pathological data, we shall there again find the proof we are in quest of; for the debility consecutive upon passive pollutions, that is to say, pollutions irrespective of erotic dreams, without erections and without voluptuous sensations, is much greater than when the pollutions have been active and accompanied by a certain degree of cynic spasm.

Be that as it may, impotence is one of the first consequences of involuntary seminal emissions—I say one of the first consequences, as the malady need not have long existed for this symptom to show itself.

Loss of procreative power is the usual result. This condition, however, must not be confounded with impotence. An impotent man may be fit to generate, while a man in the full vigor of virile power may be unfruitful. The infecundity of the latter may depend on different causes. Thus, with every appearance of virility, the man may be destitute of procreative power, because his semen has not the necessary qualities, is destitute of spermatozoa, or if it do contain these little animalcules, they are altered and misshapen. Men in whom the testicles have not descended into the scrotum, are unfruitful, but not impotent. Cryptorchidous men and mongrel animals, while they are perfectly capable of discharging into the female, and are even exceedingly lascivious, are unfruitful. Infecundity in the man may also depend upon imperfection or morbid alteration of the external genital organs. The penis may be deficient in length, either by natural conformation, or by having been accidentally shortened, as, for example, when it is, so to speak, obliterated by a tumor in its vicinity, a hydrocele, or a scrotal hernia; on the other hand, it may be excessive in length or thickness, or its direction may be vicious; again, there may be epispadias, hypospadias, or phimosis—anomalous conformations which may prevent the ejaculated semen from being deposited in the manner in which it ought in the sexual organs of the woman: once more, stricture of the urethra may be a cause of infecundity by embarrassing the passage of the semen. Cases are on record in which stricture was produced during coitus by the erection being too vigorous, so causing the semen to regurgitate into the bladder, or preventing its being ejaculated till the turgescence of the penis had ceased.

Infecundity, therefore, does not necessarily imply impotence; and as I have said, the latter may exist—only, of course, to a limited extent—without the individual having lost his procreative power. To enable such a person to engender, it would only be necessary to introduce the penis completely within the vulvar passage, for then, even without erection, fecundation might take place.

In the subjects of spermatorrhœa, infecundity may depend upon ejaculation occurring before introduction of the penis into the sexual parts of the woman; or it may arise, notwithstanding introduction, from the ejaculation being too feeble to send the semen far enough; in both these cases, the uterus may not be sufficiently excited. The principal cause, however, of infecundity in spermatorrhœa is the alteration of the semen, which either does not contain spermatozoa, or only contains spermatozoa which are malformed or essentially changed in their nature.

Impotence and infecundity are far from being the only effects of involuntary seminal emissions; they may also originate various nervous diseases. Under this class of affections may be placed various disturbances of the great functions of organic life, the origin of which is often misunderstood.

During the early period of the disease, the appetite may not only remain, but may even be increased; but the sensation of hunger is not that of ordinary hunger; there are twinging pains, a feeling of discomfort, and a sinking sensation, temporarily relieved by taking a small quantity of food; soon, however, a loathing of food sets in, and to satisfy nature's demand for nourishment, the patients endeavor to stimulate the appetite by strongly spiced meats and exciting drinks. The results of this kind of feeding are irritation of the stomach and difficult digestion. The exciting food and surcharged state of the alimentary canal produce a notable increase in the involuntary emissions. Gastric and intestinal symptoms, varying according to the individual, and varying from day to day in the same individual, set in; constipation is succeeded by diarrhœa, which finally establishes itself in a chronic form, becoming a permanent and direct cause of spermatorrhœa.

The exhaustion and general debility produced by habitually involuntary loss of semen necessarily increase under the influence of disturbed nutritive functions. The victim of spermatorrhœa falls into a state of extreme wasting. He loses color, the complexion becomes pale, and the skin acquires a yellowish leaden hue; his eyes become encircled with a blue ring, hollow, dull, and expressionless. He is easily injured by reduction of the external temperature; and he progressively loses his moral and physical energy. His muscular activity is more and more impaired; he becomes unable to sustain bodily exercise, even for a short time, without complaining of panting and dyspnœa; and in proportion to the advance of the disease, so increases the difficulty of performing muscular movements. It is an extraordinary fact, and one which, according to Lallemand, is a pathognomonic phenomenon of spermatorrhœa, that in conjunction with this feebleness—even when it exists to an extreme degree—the patient has an unconquerable desire to move, and, even when hardly able to stir, is impelled by physical restlessness to seek constantly to go from place to place.

Palpitation of the heart, and an accelerated, small, feeble pulse, give evidence of disordered sanguinification, and anæmia is not unfrequently manifested by a vascular bellows-murmur. I have referred to the shortness of breath complained of by the patients on taking slightly prolonged exercise; at a later period, this oppressed breathing is constant, rest not causing it to cease; the respirations are slowly drawn, few in number, and not deep. Certain patients are tormented by a constant dry cough, and suffer from neuralgic pains in some part of the chest; auscultation reveals a feebleness of the respiratory murmur depending upon general debility.

The sensorial functions are variously modified. A form of anæsthesia exists, which from its mobility may be compared to that observed in hysterical and hypochondriacal patients: sometimes in the hands, sometimes in the chest, sometimes in the abdomen, and sometimes in the integument of other parts of the body, the tactile sensibility is obtuse in a surface more or less extensive, and for a longer or shorter period. The individuals complain of very transitory sensations of heat, burning, or cold; they compare them to sensations caused by a current of electricity, by cold air, or by tepid water; they also complain of pains similar to those produced by a violent squeeze, by a contusion, or by tingling, of which the back and shins are the principal seat.

At last, the special senses participate in the general disorder of the system. *Disorders of the sense of sight* arise. Complete amaurosis may occur, beginning with amblyopia or diplopia, though cases of this description are rare. The impaired vision is accompanied by extreme sensibility to light, and a more or less remarkable dilatation of the pupils. The *sense of hearing* loses its delicacy and precision: it becomes exceedingly sensitive; there is buzzing, ringing, and singing in the ears, symptoms which sometimes proceed to such a length as to constitute complete deafness. The *senses of taste and smell* may also be perverted.

Pains in the head and vertigo, symptoms which constitute part of the concomitant train of phenomena of spermatorrhœa, are most palpable when the patients have difficult digestion, when they have attempted somewhat sustained mental exertion, or when they have passed sleepless nights. Their sleep is generally light, and but little restorative: as the involuntary seminal emissions take place most frequently during the night, they are more exhausted than before they fell asleep. At an advanced stage of the disease, there may be complete insomnia: when it is so, the patients pass the night in a state of great excitement, covering and uncovering themselves, getting up and lying down, changing their position every moment without ever finding one more convenient. When sleep does come at last, it is troubled by painful nightmare. These distressing nights leave behind them extreme fatigue: and during the whole day, the patients remain in a sort of brutish stupidity of which they are conscious, a circumstance which explains the mental depression, hopelessness, and melancholy which make them seek to fly from every kind of society.

The patients undergo a great moral change. Wholly engrossed with their own state of health, they are indifferent to the circumstances by which those around them may be affected: they are exceedingly pusillanimous, irascible, and as insupportable to others as to themselves. Their memory becomes weak; and this enfeebling of the memory, combined with a certain degree of paralysis of the tongue, combined also with feebleness of voice and hesitating speech, makes it difficult for them to express their ideas, the elaboration of which, moreover, is less active and less precise.

Finally, the disturbance of the intellectual faculties may proceed to such an extent as to constitute insanity. The insanity may be temporary, and, remaining entirely subordinate to its cause, may be recovered from, when recovery from the spermatorrhœa takes place: but it may also be persistent, continuing long after the complete cessation of the spermatorrhœa which caused its evolution. Lallemand clearly indicated this capital fact, and observed that the most common forms of insanity which occur as consequences of spermatorrhœa are hypochondriasis, melancholia, and lypomania, complicated sometimes with a tendency to commit suicide.

This long programme of symptoms, upon which Lallemand has dilated with so much care—with perhaps too much care—recalls the characteristic features of confirmed hypochondriasis.

In general, it is not usual to find a young man suffering from severe hypochondriasis who has not spermatorrhœa. There are, however, numerous cases of hypochondriasis occurring irrespective of involuntary seminal emissions. But before I enter upon the doctrinal question touching the relation of spermatorrhœa to the state of the nervous system, I wish to bespeak your attention to certain material causes of spermatorrhœa.

It has been observed that spermatorrhœa is rather common among persons who have an unusually long prepuce. In such cases, the sebaceous secretion accumulates around the gland, which it irritates; and it can be understood that, when these conditions exist in persons predisposed to spermatorrhœa, ejaculations may occur as readily as they occur in the same persons when they have the least contact with a woman. Lallemand has frequently seen, that in such cases circumcision, if it be not a means of cure, has at least an extraordinary influence in diminishing the extent of the spermatorrhœa.

But disproportionate length of the prepuce has relation to another cause, which did not escape the sagacity of Lallemand—I refer to the imperfect development of the corpora cavernosa. In this case, we must look to the nervous system for the original cause of the affection. Individuals affected in this way are the children of parents nearly related by blood to each other, of insane and epileptic persons; or at least some members of their family are insane, epileptic, or the subjects of hare-lip. They have a preexisting lesion of the nervous system, whence follows a complete series of consequences, and first of all an imperfect evolution of certain organs—on the other hand, a constant tendency to neuroses in general, and to spermatorrhœa in particular, the latter being in reality a neurosis of the organs of generation.

A similar explanation may be given of the pollutions which occur in monorchidous and cryptorchidous persons, and in individuals affected with hypospadias or epispadias: in these subjects, there exist both arrested development of the organ, and disorder of its function, consequent upon a bad congenital condition of the nervous system: the congenital material lesion, as well as the disorder of the function with which the organ is charged, express one and the same thing, that is to say, a vice in organic evolution.

To return to the very various nervous symptoms which I have already enumerated in detail, let me remark, that you will meet with those which signalize the beginning of locomotor ataxy, such as diplopia, amblyopia, the darting pains in the limbs; and let me remind you that, as a corollary, I have told you that locomotor ataxy is often preceded by seminal emissions.

Gentlemen, I have already stated my opinion regarding the extraordinary influence which seminal losses have upon the entire economy, but particularly upon the nervous system. I am convinced that Lallemand has shaded his picture much too darkly, and that he has made a great mistake in attributing perturbations of the nervous system to exhaustion caused by excessive and too frequently repeated discharge of semen, when the *nervous disorders* might more justly be regarded as the *cause* of the spermatorrhœa.

Permit me, gentlemen, to expatiate a little upon this opinion. First of all, we know by experience, that a large proportion of the young men who have spermatorrhœa have had *nocturnal incontinence of urine* in infancy: this is a very serious nervous symptom, and it often happens that the same subjects show eccentricities of character, irritability, and unequivocal signs of hypochondria at an age when hypochondria is very rare. Could we penetrate into family secrets, we should often find that among the predecessors of our patients there were brothers or sisters who had had serious diseases of the nervous system, such as hypochondria, insanity, epilepsy, and locomotor ataxy. We should thus find as an explanation of the spermatorrhœa, as an explanation of the nocturnal incontinence of urine, hereditary and personal predisposing causes; and should, therefore, have no right to attribute the nervous symptoms to the spermatorrhœa. It is far more reasonable to think and say that the nocturnal incontinence of urine and the spermatorrhœa are consequences of an unhealthy state of the encephalon, and particularly of the spinal marrow, a state the nature of which it is not easy to specify.

To illustrate this point, let me give you some examples which occurred within a short period in my private practice.

On 10th April, 1866, I was consulted by M. C——, who had married his cousin-german. He had by this marriage two sons, whom I have seen several times. The eldest, 9 years of age, is a delicate boy, and affected with nocturnal incontinence of urine: the youngest, 8 years of age, has from his birth been an epileptic idiot.

On 13th April, I was consulted in the case of a young lady of 19, who was beautiful and healthy looking, but had been an epileptic for two years. She had not had nocturnal incontinence of urine in childhood, but her father had suffered from it till he was 7 years old.

On 16th April, I was consulted by a lady of 34 years of age: she had had puerperal mania. Of her two daughters, one was affected with nocturnal incontinence of urine till she was 12 years old: the other has hysterical paralysis. Father and mother are first cousins.

On 19th April, I was consulted on behalf of two young men whose father died insane: one was 24, and the other 21 years of age: both had frequent seminal emissions, and both led very loose lives.

On 20th April, I was consulted by a gentleman who had an insane brother. He himself had never had nocturnal incontinence of urine, but had long suffered from very abundant and very frequent spermatorrhœa. For two years he has had hypochondria almost proceeding to nosomania.

On 3d May, I was consulted by a young man of 21, who had had incontinence of urine till he was 14 years old. From infancy, he had been a sufferer from epilepsy, and had had both the "grand-mal" and the "petit mal." I was told that nervous diseases had never shown themselves in the family.

On 10th May, I received in my consulting-room a young lady from Copenhagen, who up to the age of 5 years had had nocturnal incontinence of urine. From that time, she had had epileptic attacks, particularly in the vertiginous form.

On the same day, I saw at Auteuil, at the house of M. Beni-Barde, a gentleman aged 30: his mother was very nervous and his grandmother had been insane: he himself had had nocturnal incontinence of urine till he was 12 years old: from 16 onwards till now, he has had spermatorrhœa: at present, he has hypochondriacal symptoms, the most curious nervous disorders, and apparently there is also imminent insanity.

On 11th May, I saw a young lady of Mezidon, aged 20. Her father was a lunatic, and her grandmother an hysterical subject. Till the age of 12, she had suffered from nocturnal incontinence of urine, and subsequently from epilepsy.

On 21st May, I was consulted in the case of a young lady of 13, the child of parents who were nearly related by blood. Till she was 5 or 6 years old, she had nocturnal incontinence of urine: from that date she had had vertiginous epilepsy, and sometimes the "grand mal."

On 22d May, I was consulted by a young man, aged 22, who had nocturnal incontinence of urine till he was 6 or 7 years old. From the age of puberty onwards, he had had spermatorrhœa. A brother was paraplegic.

On 26th May, a gentleman of 45 consulted me. For two years he had had epilepsy. His son, 14 years of age, had nocturnal incontinence of urine.

On 3d July, a Spaniard, aged 34, came to consult me. Till he was 10 years old, he had had incontinence of urine: from that age and onwards, he had had spermatorrhœa: he is now impotent: for the last three months, he has been subject to epileptic attacks.

On 5th July, I saw a young man of 22, who had suffered from incontinence of urine and fæces since he was 9 years of age. From puberty onwards, he had had spermatorrhœa. His mother and a brother were epileptics.

On 9th October, I saw an epileptic young man of eighteen. He had not had nocturnal incontinence of urine in childhood: but he had nocturnal spermatorrhœa about three times a week. The symptoms were not hereditary.

On 11th October, I saw a young man, a twin, of 25 years of age. In childhood, he had had incontinence of urine; he was, when I saw him, suffering from nocturnal and diurnal spermatorrhœa. He felt much more tired after sexual intercourse than after a nocturnal pollution. He was the subject of very decided anaphrodisia, and the seminal emission took place almost immediately.

I do not lengthen this catalogue of cases, though, without any difficulty, I could largely do so. You will perceive that the relatively considerable number of almost identical facts occurring within a very limited space of time is sufficient to prove, in the first instance, the relation between the genito-urinary and nervous symptoms of an individual, and also the influence on children of the consanguinity of their parents, and of the state of their nervous system; then, on the other hand, it establishes the existence of a chain of morbid phenomena, almost always the same in character, which begins with nocturnal incontinence of urine, continues with spermatorrhœa, and culminates in hypochondria, epilepsy, and insanity. This shows you that all the symptoms originate in one and the same cause, and that that cause is a primary morbid cause pertaining to the nervous system.

There is yet another consideration to which I wish to direct your attention. I am frequently consulted by men who have seminal emissions, and also suffer from the series of nervous symptoms of which Lallemand has so complaisantly drawn the picture. Upon entering into details, it is found that the nocturnal pollutions do not recur more than four times a week:

microscopic examination of the urine affords convincing proof that there are no other emissions than those which occur during the night. Now, inasmuch as a young man may without any bad consequence accomplish coitus three or four times a week, it may be assumed that if another young man who has involuntary seminal emissions three or four times a week has also very serious nervous symptoms, the latter are not caused by the loss of semen.

Another fact has to be stated. Involuntary seminal emissions are much less abundant than emissions preceded by the ordinary venereal excitement. On the other hand, microscopical examination shows, that in the seminal fluid of persons affected with spermatorrhœa, there are far fewer spermatozoa than in semen discharged *in coitu;* and physiologically, this ought to be the case, as the erotic excitement not only induces a more profuse secretion from the vesiculæ seminales, but likewise from the testicle itself.

Moreover, the intense nervous excitement which precedes, accompanies, and in a special degree terminates the act of copulation, leaves as its immediate consequence an amount of prostration which testifies to the great effect which it has produced on the nervous system: but nothing of a similar description is observed after involuntary seminal emissions, which frequently take place without erotic dreams, or after excitement so transient and little felt as sometimes not to be perceived by the patients except by the soiling of their bed or clothes.

Gentlemen, I do not know whether these considerations strike you as they strike me: but I am forcibly led to the conclusion that the nervous disorders observed in spermatorrhœal patients do not depend upon loss of seminal fluid.

By this statement, however, I do not mean to imply, that seminal emissions are without any influence on the system. I mean that in a hale young man, whose nervous system is in a state of harmonious action, coitus, terminating in seminal emission, may take place twice a week without any detriment to health; but if it be granted that in the spermatorrhœal patient, the brain and spinal marrow are in a state of debility, a smaller seminal loss, even when unaccompanied by sufficient erotic excitement, will become a powerful cause of nervous disorder; and this disorder will almost inevitably become excessive, if the seminal emissions recur very frequently, as is too often the case. Thus it is, that a bad state of the nervous system predisposes to spermatorrhœa, and that spermatorrhœa peculiarly aggravates the nervous affection which is the primary source of the evil.

Gentlemen, the symptoms which may bring in their train involuntary seminal emissions, by no means all show themselves in the same individual, neither do they develop themselves in the same order of succession, nor do they always reach the same extreme degree of severity. One or other phenomenon will in general predominate over the others, and so great will be its predominance, that the patient's attention will be directed to it exclusively. This may lead the physician, unless he be specially on his guard, to form an erroneous diagnosis, and to believe that there exists an affection very different from the real one. I cannot too frequently repeat, that the disease may present in its aspect, and in its progress, an infinity of varieties; and that its different forms are subordinate to individual peculiarities, and many special circumstances difficult to foresee. They are also subordinate to intercurrent complications.

In a young man, twenty-six years of age, whom we had in bed 18 of St. Agnes Ward, the disease was only of four months' duration, and seemed to have had a chronic gonorrhœa as its starting-point. The individual to whom I refer told us, that the gonorrhœa, of which he had only got rid nine or ten months previously, had lasted three years. The spermatorrhœa declared itself five or six months later, or at least only at the time when the patient first observed that he had involuntary seminal emissions on going to stool. Three weeks before the symptoms set in, he was troubled with obstinate constipation; but although the constipation ceased, and although the alvine evacuations became regular, and regained their natural consistence, the pollutions continued without abatement. From that time also, nocturnal pollutions supervened, an occurrence not in accordance with the general rule; for it is usual for the nocturnal to precede the diurnal pollutions. The nocturnal pollutions always occurred in connection with erotic dreams: the diurnal pollutions, which, as the patient stated, first took place when the bowels were being moved, and then recurred more and more frequently. If he attempted coitus, ejaculation occurred almost immediately, not only before copulation, but before there was complete erection: ultimately, even before there was any erection at all. This young man told me that one day when looking at the picture of an amorous couple exhibited in a shop-window, a pollution without previous erection was caused by the lascivious representation.

The general health of the patient was very much out of order. He complained of excessive debility; and was fatigued by the least exertion. Palpitation of the heart would be brought on by a rather long walk: his appetite became impaired, and he soon had a feeling of disgust for the different kinds of food which he tried to take: he preferred spiced alimentary substances and those prepared with oil or vinegar. Eating, however, temporarily calmed the gastric pain which preceded a repast, and caused a cessation of the eructations, which were of sickly odor and accompanied the pain; but in two hours both pain and eructations always returned.

Tonic treatment, sulphurous baths, cold hip-baths, and preparations of quinine (prescribed in one of the services of this hospital) produced some improvement. His disease, however, having soon become as bad as it was before, he returned to the Hôtel-Dieu.

The existence of gonorrhœa of old standing having suggested the idea that the spermatorrhœa might depend on a chronic affection of the urethra, an instrument was passed to ascertain whether there was stricture. The patient stated that he had experienced some difficulty in making water for the preceding three or four months—that the stream, which was long in coming, was flat and spiral—that it was sometimes interrupted—and that some drops of urine escaped after he believed that he had completed micturition.

On introducing the sound, I encountered one obstacle at the entrance of the urethra, a second about the middle of its cavernous portion, and a third obstacle in its prostatic portion. The treatment indicated by Lallemand seemed suitable; but it was not tried, as the patient was dismissed, at his own request, after a residence of three days.

Gentlemen, I shall now recapitulate some of the conditions under the influence of which spermatorrhœa is produced—conditions with which it is essential to be acquainted, that we may be able to institute a rational treatment of the disease.

Here, I find it necessary to make a short digression into the domain of physiology.

To the question, *By what organ is the semen secreted?* it might be supposed that no other reply could be given than—"*By the testicle.*" Nevertheless, this is not the correct answer; as is proved by experiments on the lower animals, as well as by the facts observed in the human subject.

Take an animal—a young dog, for example—tie the deferent ducts in two places,—and cut them through between the ligatures. In this way the testicles will be completely isolated from the urethra. Still, the animal will be able to copulate with energy, and the seminal ejaculation will be nearly as abundant as before the operation. A somewhat similar phenomenon is observed in the human subject. An individual contracts a gonorrhœa: this, to use a vulgar expression, falls into the scrotum [*tombe dans les bourses*], or, to speak more in the language of science, there supervene inflammation and consecutive induration sufficient to cause obliteration of the excretory ducts of the epididymis; but this does not deprive the individual of venereal aptitude: he will be quite as able to copulate as before, and ejaculation will lose nothing in energy or power. This arises, gentlemen, from the seminal fluid being furnished by two sources: the larger portion comes from the vesiculæ seminales and the other, and smaller portion, from the testicles. The portion, however, derived from the testicles is the most important, as it is that which contains the fecundating substance and the spermatozoa. In the pathological conditions of which I have been speaking, although the venereal aptitude remains, and although ejaculation can take place, the seminal fluid loses its essential properties, and if both epididymes are similarly affected, the individual is rendered unfruitful. These facts, of which Hunter caught a glimpse, have lately been wonderfully cleared up by Professor Gosselin.*

A knowledge of the physiological fact now stated will enable you to understand how it is that certain young men of sound and vigorous constitution, presenting, moreover, all the attributes of virility, are nevertheless unfruitful. This is a point regarding which it is specially important for the practitioner to be cognizant, as it is one on which he may be consulted. Before seeking for other causes of the sterility complained of, scrupulously interrogate your patient, attentively examine him, inquire whether he has not had at some former time an attack of gonorrhœa, and discover whether, as a consequence of that attack, he has not complete obliteration of the spermatic passages. This fact requires the more attention as at first it might appear that the condition of the individual was similar to that of a eunuch. His state, however, is very different: do not make any mistake on that point. The old man in the last stage of decay, who for many years has been incapable of sexual intercourse, impotent though he be, and consequently unfruitful, bears no resemblance to the man who has been castrated, for his useless testicles still give him the stamp of virility: so long as he retain them, he will retain certain attributes of manhood, such as the bass voice and the beard; but these attributes will at once disappear should he lose the testicles which have apparently ceased to perform any part in the economy. A very curious work of Professor Charles Robin upon sarcocele contains a demonstration of the correctness of this statement.†

A person has double sarcocele, and although it might be supposed that his testicles were destroyed, he does not really become a eunuch till after the surgeon has removed both organs. This is the light in which Professor Robin looks at such cases: they belong to the same category as the two cases of Professor Gosselin—in this sense, that in them there is no disease of the testicles. Sarcocele is an affection which pertains to the epididymis and remains separated by the *tunica albuginea* from the gland, the tissue of which it so much respects that the normal structure of the filamentary tubules is well preserved, although the tubules are generally found displayed upon the surface of the epididymotic tumor.

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* Gosselin: Nouvelles Etudes sur l'Oblitération des Voies Spermatiques et sur la Stérilité Consécutive à l'Epididymite Bilatérale. [*Archires Générales de Médecine* for November, 1853.]

† Robin (Charles): Mémoires de la Société de Biologie. Second Series. Vol. III (for 1856): p. 1867. Paris: 1857.

I have already told you, that the same thing has been observed in cryptorchidous persons: that is to say, that though they are unfruitful, they are not impotent.

That fact being well established, let us resume our subject. To enable a gland to perform its function, there is no necessity for the direct action on it of the stimulus. Irritation of the mucous membrane of the mouth, stomatitis, may induce abundant secretion from the salivary glands, though they are not implicated in the inflammation, just as inflammation of the ocular mucous membrane occasions in a similar manner a more copious flow of tears; just as irritation of the gastro-intestinal mucous membrane occasions a greater secretion of bile and pancreatic juice; or finally, just as irritation of the bladder, cystitis, is accompanied by a secretion of urine which is more abundant than usual, and which is also passed more frequently, even although, as in this case, the affected organ is far removed from the gland the functions of which it sympathetically excites. Well then, in similar sympathetic action, we find a cause of involuntary seminal losses.

It is upon this fact that Lallemand has based almost the whole of his theory of spermatorrhœa. He holds that spermatorrhœa is nearly always dependent upon irritation of the prostate gland and its ejaculatory ducts: and he believes that in most cases this irritation, which also exists in the neck of the bladder, is the result of chronic inflammation of the urethra in the prostatic portion of the verumontanum. According to the illustrious professor of Montpellier, the most frequent cause of involuntary seminal emissions is an old attack of urethritis, or of gonorrhœa; and these seminal losses, he says, are often related to stricture of the urethra.

This class of causes does not admit of being called in question; and the sympathetic irritation which the chronic inflammation of the urethra determines may likewise have as a starting-point affections seated in organs communicating more or less directly with the vesiculæ seminales and ejaculatory ducts. In this way, affections of the rectum, among which may be mentioned hæmorrhoids, the presence of vermicular ascarides, and even—although much more rarely—lumbroid ascarides, may be the cause of spermatorrhœa.

I have stated that involuntary losses of semen are in some cases provoked by obstinate and habitual constipation; but in these cases, the seminal emission is induced mechanically by compression exerted upon the vesiculæ seminales by the excrementitious bolus, as it is being expelled with difficulty and great effort.

Though for the most part these are the causes which induce spermatorrhœa, undoubtedly there are also others. Thus, seminal losses may supervene under the influence of spasm, in a manner similar to that which we saw take place in nocturnal incontinence of urine. I shall afterwards have to address you in a more special manner upon this latter affection; but that I may enable you the better to accurately grasp my views on the subject before us, I shall now recapitulate what occurs in nocturnal incontinence of urine. The patients in whom this affection is observed—chiefly children—generally pass the urine during the day in a stronger stream than other persons: this greater vigor of emission in voluntary micturition shows a greater energy in the contractile power of the bladder. It is a still more curious circumstance, that children who cannot retain their urine in its natural reservoir during the night, can sometimes during waking hours retain it more easily, and for a longer period, than others. How are we to explain this fact, which was long ago pointed out by Bretonneau? During sleep, the bladder enters into a state of erethism, which may be compared to that of the external genital organs; for, as you are aware, there is always a state of erection during sleep in children and young men. The cause of incontinence of urine is a condition analogous to that of erethism: it is due, if you will allow me to use such an expression—it is due to the vesical muscle itself entering into a state of erection. That this take place, it is not necessary that the bladder should be full, nor even that it should contain a great quantity of fluid: so far from that being the case, the urine is emitted as soon as a small quantity has accumulated in the bladder: and that it thus happens is proved by the fact, that it is during the early part of the night that the child is troubled with incontinence of urine. The child is made to pass his water when he is put to bed, and it is within two hours—not after a lapse of eight or ten hours—that he wets his bed. I repeat, therefore, that the occurrence is dependent upon a very energetic contraction, a sort of temporary erection of the vesical muscle, which the sphincter, charged with closing the orifice of the neck of the bladder, is unable to resist.

An analogous mechanical cause may produce involuntary seminal emissions. It is during the early hours of the night that pollutions occur. Under some exciting influence, which at times is occasioned merely by the position in bed of the individual, the vesiculæ seminales enter into a state of erection; or, if you prefer the expression, contract in the same energetic manner in which I have said the vesical muscle contracts; and the result is an ejaculation of seminal fluid without the patient being conscious of the occurrence, just as in the other case there is involuntary emission of urine.

Spermatorrhœa may be the result of atony of the organs which secrete the seminal fluid, as well as of their too energetic contraction. The state of the organs is then quite passive, just as it sometimes is in emission of urine when the incontinence occurs during the day. There are individuals, both children and adults, who are not able to retain their urine, when even a small quantity has accumulated in the bladder: in them, the jet is weak and dribbling, and not strong, as in the other class of persons of whom I have just been speaking. This incontinence depends on a debility of the sphincter, which is absolute, not relative, as in the class of cases previously described, in which the contractility of the vesical muscle is greater than that of the muscular fibres encircling the neck of the bladder. The same takes place in respect of involuntary seminal emissions: the ejaculatory ducts from atony are unable to resist the feeble contraction of the vesiculæ seminales, and to retain the semen which is passing through them, so that there is ejaculation, or, to speak more correctly, an involuntary flow of semen as soon as it is secreted. My comparison of this kind of spermatorrhœa with diurnal incontinence of urine is peculiarly appropriate, as it may be extended to the treatment: the same treatment is equally efficacious in both maladies, as you saw from a case of incontinence of urine which we had in St. Agnes Ward, a case of which I intend to speak when I come to address you regarding the latter malady.

Sufficient attention has not been directed to *impotence* consecutive to spermatorrhœa. How often have I been consulted by persons for their infirmity, and how many of these patients have frankly disclosed it; but how many more have only come to confess it after numerous circumlocutions! Well, then, nearly all who were impotent from atrophy of the testicles, or from cryptorchidia, were found to have had nocturnal incontinence of urine in childhood, involuntary pollutions at puberty, and at the age of manhood, either inability to have an erection when with a woman, or if they had an erection, it was of so transient a character that the penis was hardly introduced ere the copulative act was completed by premature ejaculation, unaccompanied by any voluptuous sensation. Who does not perceive that the phenomena are of the same character, in the incontinence of urine of childhood, in the incontinence of semen of puberty, and in the genesic impotence of manhood? And who does not also perceive, that these successive infirmities are dependent on an imperfection in the nervous system of organic life? Some of these patients tell us that they are frantically addicted to masturbation; and they believe that their impotence is the consequence of these lamentable excesses. But is not the practice of masturbation an indirect proof of my proposition, that there is a bad state of the nervous system? Is it not in fact mental aberration which impels these poor infatuated creatures to their solitary debauchery? If, at a later period, these same persons become impotent, insane, or paraplegic, we must not conclude that this is the direct consequence of masturbation, but must rather regard it as an aggravation of a nervous condition of which masturbation was only the first morbid manifestation.

From the considerations into which I have now entered, there may be deduced certain therapeutic indications, which must be borne in mind in treating spermatorrhœa.

Prior to the date at which Lallemand published his important researches, spermatorrhœa had enlisted the attention of physicians only in a small degree—there was an incorrect understanding of its serious character, and of the disastrous consequences directly and indirectly induced by it: it was treated without method, and by purely empirical means.

Lallemand, by presenting the disease as almost exclusively the result of irritation of the spermatic ducts arising from chronic inflammation, showed that the treatment indicated consisted in modifying the condition of the mucous surfaces, the seat of that inflammation. He recommended, as the measure best calculated to attain this object, cauterization of the mucous membrane of the prostatic portion of the urethra, so as to touch the verumontanum near the opening of the ejaculatory ducts. In support of his opinion, he adduces a great number of sufficiently apposite cases, so that when one reads his work, a conviction remains that the operation now mentioned is often useful. It is not necessary for me in this place to describe to you Lallemand's method of performing this description of cauterization: in relation to that point, I cannot do better than refer you to his "Traité des Pertes Seminales Involontaires," where you will find every possible information as to the most desirable way of carrying out the treatment.

However incontestable may be the utility of this medication in circumstances such as I have now described, Lallemand has in my opinion committed a mistake in generalizing its application; and he has made that mistake, because he would also generalize beyond measure the influence of urethral inflammation upon the production of involuntary seminal emissions. Let me repeat then, that while the utility of cauterization is incontestable in the circumstances which I have pointed out, that is to say in spermatorrhœa arising from inflammation of the urethra, it is not applicable in other kinds of spermatorrhœa, in which an entirely different kind of treatment is required. In them, it is the spasmodic element against which we have to contend; and belladonna, so marvellously useful in nocturnal incontinence of urine, is employed to render real service in that form of spermatorrhœa which presents a certain analogy to nocturnal incontinence of urine. Belladonna, however, is far from being as efficacious in the former as in the latter affection: still, the cases of spermatorrhœa in which this medicine has seemed to me particularly useful are so numerous that I recommend you to give it a trial.

The utility of belladonna in spermatorrhœa, as well as in nocturnal incontinence of urine, does not perhaps depend upon that obtunding influence which it exerts upon the contractility of the vesiculæ seminales, but upon its undoubted influence upon the entire nervous system, particularly on the encephalon and spinal marrow. Its great power is shown by the undoubted special effects obtained from it in epilepsy, tetanus, and many other nervous diseases.

Probably, to a similar special influence is due the real service obtained by preparations of digitalis and aconite in the treatment of spermatorrhœa. To a similar kind of property are attributable the favorable results of my using internally the nitrate of silver, a medicine which certainly produces no stupefying effects.

Believing that there is always spinal irritation, or at least an altered state of the spinal marrow, depending perhaps on congestion of the cord, I frequently advise repeated recourse to dry, or even sometimes to sanguineous cupping over the spine, to the application to the same situation of lotions of tincture of iodine, and a thick woollen stuff impregnated with embrocations of essence of turpentine, over which a very hot iron is passed. Finally, I do not hesitate, in certain cases, to apply moxas and flying cauteries: I act thus energetically particularly when pains supervene in the limbs, the first symptoms of locomotor ataxy, a terrible disease, the existence of which is often announced by spermatorrhœa.

Hydropathy, as an antispasmodic agent, ought to occupy an important place in the treatment of seminal losses; and sea-bathing, really only another form of hydropathy, is likewise exceedingly beneficial.

When spermatorrhœa depends upon an excess of energy in the contractions of the vesiculæ seminales and ejaculatory ducts, I prescribe *warm hip-baths*, directing them to be used as hot as the patient can take them. I also recommend *bags of hot sand* to be applied to the whole of the perineal region: the application ought to be made at night when the patient goes to bed, and it ought to continue each time at least half an hour.

It will no doubt surprise some of you to hear me recommending hot baths, when most physicians order cold baths in the treatment of spermatorrhœa. Perhaps I may be accused of acting in a spirit of contradiction, while in truth I have no such tendency, being always inclined to accept, come from whomsoever it may, the oddest possible modes of treatment, provided their application appears to be devoid of all risk. While, therefore, I now speak in favor of warm baths, while in a more general way, I state my conviction that hot are preferable to cold applications in the class of cases now before us, I have my reasons for so doing. On many occasions, I have pointed out to you the great power of caloric as an antiphlogistic agent, and in contrast, have mentioned the potency of cold as an energetic excitant. The truth of these propositions is absolutely demonstrated by the most commonly known facts. When the hands are immersed in snow, or in iced water, the chill felt in the first instance is soon succeeded by a great increase of temperature: when the hands are immersed in very hot water, in water sufficiently hot to cause temporary congestion of the tissues, the congestion is quickly followed by a fall of temperature, and a notable diminution in the color of the skin. It happens thus, because the ultimate effect of the application of heat is sedative, while that of cold is eminently phlogistic. The sedative power of heat is frequently made use of in medical practice. This is an important question, the complete discussion of which I reserve for a future day. In the meantime, you will understand why I recommend the application of bags of hot sand, and hip-baths, as hot as the patient can bear them, in the treatment of spermatorrhœa. I must inform you, however, that, in the first instance, there is an increase of the symptoms which we are seeking to subdue; but this temporary increase of excitement is of short duration, and amendment is not slow in showing itself.

Although hot hip-baths are useful in those cases of spermatorrhœa in which there is augmented excitability and contractility of the vesiculæ seminales and ejaculatory ducts, they are injurious in that form of the malady which I have designated *passive*. In passive spermatorrhœa, cold bathing, that is to say *hydropathy*, is clearly indicated. It is unnecessary to recapitulate what I have just been saying to explain to you the manner in which the treatment by the employment of cold acts beneficially in the passive form of spermatorrhœa.

In addition to the use of cold, there are certain medicines which ought to be administered internally in this class of cases. Among them, the foremost place is taken by nux vomica, St. Ignatius's bean, and the preparations of strychnia. These medicines, begun in minute doses, ought to be steadily and gradually increased, till they produce their physiological effects.

Nevertheless, gentlemen, it sometimes happens that all these means fail. Another, however, remains to be tried, one which I have been in the habit of employing for a great many years, and sometimes with success. I refer to compression of the prostate gland. Let me now describe the manner in which I employ this mechanical means of treatment.

In 1825, when I was attached as interne to the *Maison de Santé* at Charenton, Dr. Bleynie, the physician attached to the institution, mentioned to me the case of one of his patients, who had suffered from impotence, and had been cured by wearing, by the advice of a charlatan living in the Place Baudoyer, Paris, a sort of boxwood pessary in the rectum. The fact seemed to me strange; and at that period, like everybody else, not understanding the relation of impotence to spermatorrhœa, I was quite unable to comprehend how a cure could be accomplished by means so singular. I only saw in the proceeding a lascivious manœuvre similar to those practiced in bad resorts by libertines seeking to restore artificially for the moment their exhausted virile powers. I did not trouble myself to seek for any other explanation; and the fact mentioned by Dr. Bleynie did not appear to me of much importance. Ten years later, however, I myself used the same empirical treatment; and in then reflecting upon it, I formed a theory as to its mode of acting. I was treating a young man, aged 26, who was suffering from involuntary seminal emissions and absolute impotence. This unfortunate individual was on the point of marrying, and you can understand how this circumstance increased the state of melancholy into which he had already been thrown by his malady; suicidal ideas were passing through his mind. His malady had resisted all the means by which I had endeavored to cure it, when, recollecting the case mentioned to me by Dr. Bleynie, I resolved to try it as a forlorn hope. I advised the patient—who spent the whole day sitting on a chair in his room—to wear within the anus an apparatus which I caused to be made for him. It was a sort of boxwood stem-plug, resembling that of a speculum; when introduced into the rectum, it was kept in position by means of towels. After fifteen days of this treatment, the young man came to see me, when he informed me, to my great astonishment, I confess, that the means had been, to a certain extent, successful. The virile aptitude began to reappear as the frequency of the seminal emissions diminished. Encouraged by this result, I recommended the patient to persevere in the use of the same means. In a fortnight, the cure was sufficiently complete to allow the patient to marry. Being entirely in his confidence, he told me that he was able as any man to perform the conjugal act.

This first case gave me much to reflect upon, and set me, as I have just been saying, to seek for an interpretation of the cure: I asked myself, How can impotence be cured by such strange treatment? It occurred to me that the stem pessary in the rectum acted beneficially by compressing directly the prostate, and indirectly the ejaculatory ducts; and that this pressure compensated for the deficiency of normal contractile power in the vesiculæ seminales. Having formed this theory, I set myself to verify it when similar cases presented themselves. Such cases have frequently occurred in my practice; and although this singular treatment has not always given me the looked for result, I can at least say that, in a very considerable number of cases, the success has equalled my expectations.

Here, gentlemen, is the apparatus which I am at present in the custom of using.

In principle, I have exactly adopted the bandage which persons with large hæmorrhoids employ to prevent the profuse bleeding from which they so often suffer. The apparatus consists of a sort of small cone, made of ivory or vulcanized caoutchouc, fixed by a T bandage, which bandage is applied round the loins and kept in its place by a cincture to which are attached in front, bands passing under the thighs. The stem pessary of my apparatus is longer and more bulky than that of the bandage used for compressing hæmorrhoids, as it is requisite to be introduced higher up the rectum, so as to reach the situation of the vesiculæ seminales. I now use a simpler apparatus—simpler to this extent, that it does not require to be kept in its place by a bandage—an apparatus invented by Mathieu, our maker of surgical instruments. It consists, as you see, of a sort of metallic bung, of the form of a very elongated olive, varying in size between a pigeon's egg and a small hen's egg. This bung diminishes downwards, taking the form of a neck, the diameter of which does not exceed five millimetres [about three-sixteenths of an inch], so that when once introduced into the rectum, it is retained there by the natural constriction of the sphincter of the anus. The bung-like compressor is soldered upon a flat stem of the same metal about three or four centimetres in length and half a centimetre in breadth: the anterior half of the stem is intended to be applied to the perineum, and the other half to the coccygeal region. You see, gentlemen, that this apparatus is of marvellous simplicity, and that when it is once put in its place, it cannot become spontaneously displaced; moreover, when once it has been introduced, patients can wear it with ease continuously for a night and a day, without any necessity to fix it by a bandage. Of course, the size of the instrument must necessarily vary with individual peculiarities, with age, and with the manner in which its presence is tolerated.

I have told you, gentlemen, that that part of the compressor of the prostate which is introduced into the rectum is soldered to a stem intended to protrude externally. This junction is effected in such a way as to prevent the two parts of the apparatus from being perpendicular, and so as to form by their union an acute angle of 75 degrees on the one side, and on the opposite side an obtuse angle of 125 degrees. It is absolutely necessary, in placing the instrument, that the obtuse angle look towards the coccyx, and consequently, that the acute angle look towards the pubes: in this way the superior part of the bulge will of necessity rest on the prostate.

I ought to add, that the length of the internal part of the instrument must necessarily somewhat vary. Digital examination of the prostate, in even a few cases, will suffice to make you acquainted with the fact, that it is situated at a depth varying between two and five centimetres, according to the height and greater or less degree of corpulence of the individual.

I cannot too often repeat that this apparatus has rendered me essential services. I have more than once seen involuntary seminal emissions, which had proved rebellious under every kind of treatment, yield completely, within a few days, under the use of this apparatus: after seven or eight days of its employment, the amendment has been such, that there was not only a rewaking of virile aptitude, but likewise a notable diminution in the general symptoms and mental disorder which accompanied the impotence.

On a future occasion, I shall tell you, that in certain cases of incontinence of urine, I have also found this treatment indicated. In the meantime, let me add, that it is equally useful in both the forms of spermatorrhœa of which I have spoken.

I have now described the means which I employ in the treatment of spermatorrhœa. Quite understand, that I do not pretend that these means are infallible; and still less suppose, that I pretend to cure the serious disorders of the nervous system which too often follow in the train of the malady. Even when the symptoms which have been its starting-point have entirely subsided, the supervened perturbation of the functions of innervation, and the mental disorder, will continue, and will resist all the efforts of art. On the other hand, the cases in which there is a definite cure, or at least a greatly ameliorated state consequent upon treatment, well planned and regularly carried out, occur! thank God, sufficiently often to be sources of encouragement.

I insist upon the necessity of a plan of treatment being *regularly* carried out; for when spermatorrhœal patients too quickly abandon the different means of treatment which I have mentioned, on account of the benefit obtained leading to a belief that the malady is radically cured, the amendment is not maintained, and ere long the symptoms reappear. It is necessary, therefore, that the treatment be persevered in: and this is all the more easy, that it is neither painful nor difficult to carry out.

It must not be forgotten, that an individual who has once been affected with spermatorrhœa is, from that circumstance, more liable to it than another who never before suffered from it. It is necessary, therefore, to take every possible precaution to prevent relapses. For this reason, the remedies to which the symptoms yielded ought to be persevered in for some time after recovery. The patient ought to be advised to revert to the compressor twice or thrice a year, for a fortnight or a month each time; he ought also, according to the indications, from time to time to recur to the hip-bath, cold or hot, and to hydropathy. The medication I recommend will not, I repeat, prove efficacious, unless it be carried out for a long period, and in a methodical manner. Even when it is employed with extreme patience, failures will too often occur.

A few words more ere I conclude:

In September, 1863, a young Irishman came to me, recommended by one of my honorable professional brethren of Dublin. For two years, he had had involuntary seminal emissions during the night. In consequence of my friend Dr. Adolphe Richard having mentioned to me the good results he had obtained in similar cases by *forcible dilatation of the anus*, I intrusted the patient to him. He operated in my presence. From the day on which the operation was performed, there was no recurrence of the emissions; and four months later, in January, 1864, Dr. Richard received a letter from the young man, in which he intimated that the cure still continued perfect.

In what manner does dilatation of the anus act in such cases? This is a question which I cannot answer; but as the operation is totally exempt from danger, it ought to be added to the list of means which one may employ in the treatment of an affection so often rebellious against treatment as that which has formed the subject of this lecture.

LECTURE LXIII.

NOCTURNAL INCONTINENCE OF URINE.

Different Kinds.—Nocturnal Incontinence of Urine not a Morbid State in Lazy and Timid Children.—Nocturnal Incontinence of Urine (properly so called) is an Affection of the Nervous System, specially manifesting itself in an Excess of Excitability and Tonicity in the Muscular Coat of the Bladder.—Nocturnal and Diurnal Incontinence Coexisting depend on Atony of the Sphincter of the Bladder.—Treatment: Belladonna in Nocturnal Incontinence; Strychnine in Coexisting Nocturnal and Diurnal Incontinence.—The Prostatic Compressor.

GENTLEMEN: You have frequently heard me interrogate a young girl, who assists the nurses in St. Bernard Ward, as to the length of time she had been in the habit of wetting her bed. This girl, to whom I have more than once called your attention, was admitted to the clinical wards, about eighteen months ago, for nocturnal incontinence of urine. Under the influence of treatment by belladonna, the symptoms have gradually ameliorated to such a degree that the cure may now be looked upon as certain.

This patient, now 19 years of age, though presenting every appearance of a vigorous constitution, is of an eminently lymphatic temperament. During her residence in the hospital, we have several times had to treat her for attacks of scrofulous ophthalmia, leaving slight specks on the cornea, which have now, however, nearly disappeared. With the exception of these ophthalmic affections, and the malady for which she came into the hospital, her health has generally been good. Some months ago she contracted pharyngeal diphtheria, when attending upon the diphtheritic patients in the nursery ward; but this diphtheria, although it occasioned some anxiety, was not followed by any bad consequences, and afterwards she soon resumed her ordinary good health.

According to the statement of this young girl, her incontinence of urine began when she was eight years of age. Till then, from early infancy, she had had nothing of the kind; and (repeating a statement of her parents), she said that her nocturnal incontinence of urine dated from a great fright which she had experienced. We shall see how much is attributable to this influence. After the fright, no night passed during which she did not wet her bed at least once, and occasionally two, three, or four times. It is a remarkable circumstance of which it is important to make special mention, that during the day and when awake she could retain her urine as well as any one, and that it was only during sleep that she was unable to do so.

Another point worthy of note is, that the irresistible necessity to pass urine seized her in the early morning during the last hours of sleep. This fact can be explained in a few words. The patient informed me that her sleep was deepest towards morning. It was then so profound that it was exceedingly difficult to awake her. When loudly called and well shaken, she neither heard nor felt anything; and when compelled to get out of bed, she seemed still to be asleep when on her legs. During the first hours of sleep, she often got up to satisfy a desire to pass urine; but whether she rose or not, she urinated during the night—and several times in the night the nurse wakened her to prevent accidents—she did not the less wet her bed before morning, even when two hours had scarcely elapsed since she had last voluntarily relieved her bladder.

These details are interesting, and I shall return to them. But let us conclude the history of our patient. Immediately upon her coming into the hospital, she began to take belladonna. The effects of the medicine were at once manifested in a decided manner; and from the very commencement of its use, the frequency of the wetting the bed was reduced to once a night: subsequently, by increasing the doses, she passed several nights without wetting her bed; and at the present time, more than two months have elapsed since such an occurrence took place.

At the same time that this girl was under treatment, we had, in bed 3 of the same ward, a girl, 16 years of age, who was also affected with incontinence of urine. Her infirmity dated from birth. She told us that she generally went to bed at half-past seven in the evening, and wet her bed between eleven o'clock and midnight. She stated that when she slept during the day, no such accident occurred, because she then awoke from feeling the sensation of the desire to make water. She added, that this desire whenever felt was always so urgent, that whether sleeping or awake, she had hardly time to run to the closet. I had recourse to belladonna, and with results as successful as in the other case.

In some cases, however, belladonna has completely failed me; and it has sometimes proved equally unsuccessful when combined with opium, or strychnine. Preparations of strychnine, however, are indicated in certain cases. I have not been more fortunate in the case of a young man in St. Agnes Ward with the recently lauded remedy, mastich. In him, however, the incontinence of urine ceased entirely after a small surgical operation.

This young man, aged 17, had, from childhood, been in the habit of wetting his bed: and the occurrence took place two or three times every night. No modification of his infirmity took place at puberty: but then although his procreative powers seemed very slightly developed, the incontinence of urine became complicated with nocturnal pollutions. For a short time, I believed that the belladonna would have produced the benefit expected from it: in fact, the incontinence of urine seemed to have yielded; but this took place concurrently with profuse diarrhœa occasioned by the medicine, and it was easy to see that if the patient did not wet his bed, it was because he had to get up to stool frequently during the night. After unsuccessfully trying the syrup of the sulphate of strychnine, and mastich, it occurred to me that his congenital phimosis might be the cause of his infirmity. I therefore asked my colleague, Professor Jobert, to perform the operation of circumcision. From the date of the operation, thirteen nights passed without his wetting his bed, then for three consecutive nights he did wet it: finally, there was no return of the nocturnal incontinence of urine during the last nine nights he spent in the hospital, so that when he asked to be allowed to return home, permission was granted under the hope that he was definitively cured.

In bed 1 of the same ward, you lately saw a man in the prime of life, who was affected with incontinence of urine, and in whom the incontinence existed by day as well as by night. This individual was 51 years of age, and by trade a house-painter. He stated that he had had five attacks of painter's colic, but that since he was two years of age, he had never had any paralytic affection.

Two years ago, he felt that there was a diminution of strength in his legs, but this was not accompanied by any decrease of cutaneous sensibility. This state of semi-paralysis, however, was general. The arms began to be affected: the tongue began to lose its freedom of motion, which produced a certain amount of difficulty in speech: the sight became enfeebled. But the symptom which gave this man most uneasiness, and was the principal cause of his coming into hospital, was the incontinence of his urine. Four or five times during the day, within a space of twelve hours, he was obliged to leave his work that he might make water; and at these times, he had no power to retain it. During the night, he wet his bed several times, because he felt no sensation, as when awake, of the necessity of emptying his bladder. Ultimately, this patient died in our wards of saturnine disease of the encephalon.

The form of incontinence of urine with which this man was affected has no relation to that other form of which I have been speaking. I was nevertheless unwilling to omit reference to this case, as I wished to call your attention to the therapeutic means which were employed.

Gentlemen, if nocturnal incontinence of urine cannot be regarded as a serious disease, it constitutes, at all events, a distressing infirmity, which you will frequently meet with in practice, and regarding which moreover, any of you may be consulted at the very commencement of your medical career. I must not, therefore, allow to pass without notice the facts which are presented to your observation; and I must specially insist upon that mode of treatment which was so wonderfully efficacious in the first of our patients, and which has likewise generally proved successful when I have employed it. But before I give you the rules for carrying out this treatment, let me specify the circumstances in which it is indicated, or in other words, state the conditions under which nocturnal incontinence of urine is produced.

There are persons, particularly children, who piss their beds, dreaming that they are pissing against a wall or into their chamberpot, the dream being suggested by a tormenting desire to make water, which desire they satisfy without awaking. There are others—children—who piss their beds from laziness: not wishing to get up when they experience the first intimation of the want to make water, they fall asleep again, and soon lose the power of retention. To this category belong cowardly folk, who, afraid of the darkness of night, neither care to leave their beds, nor call those who might come to their assistance, preferring to wet their beds to giving themselves trouble. In this description of nocturnal incontinence of urine, an affection of much less frequent occurrence than is generally supposed, the accidents recur at long intervals. To cure such cases, there is no necessity for medical intervention: it is generally sufficient to exercise a moral influence over the patients—when they are young children, to threaten them with chastisement, and when they are older, to make them feel ashamed of themselves. It is altogether otherwise in respect of the nocturnal incontinence of urine now before us; and which is the result of a condition unquestionably morbid.

This affection, though it sometimes dates from birth, as in the young girl occupying bed 3 of St. Bernard Ward, does not generally supervene till the patient has attained a certain age. Like nearly all children, she wet her bed till she was fifteen or eighteen months old; and also, like most children, on attaining that age, she lost the habit; but then, after a time, it all at once returned. Generally, it is between the ages of seven and eight, that nocturnal incontinence of urine declares itself. In these cases the occurrence takes place nearly every night, and occasionally, several times during the same night.

What are the causes of nocturnal incontinence of urine? It is not unusual both for patients and their relations to state that the infirmity originated in a fright. You cannot be too much on your guard against accepting this explanation. Moral emotions, particularly fear, are too readily put forward to explain the origin of certain neuroses, although in reality, there is nothing to show that a relationship exists between them and their pretended causes. If a child has had epileptic fits, the parents will probably tell us that the first seizure was consequent upon a great fright. I do not deny that this statement may be quite correct; and in my lectures on epilepsy I have carefully pointed out to you the share which mental emotions have in the production of that dreadful malady. The share is relatively very small, and in one hundred cases in which this morbid cause is adduced, there may perhaps not be one in which it has had the least effect. On examining into the hereditary antecedents of the patient, you will find reasons more than sufficient to explain the existence of the malady.

It is well known that in respect of incontinence of urine, as in respect of many other neuroses, heredicity plays its part in a way which is indisputable.

Some time ago, I received in my consulting-room a young lady of 20 who had this infirmity. Almost every night wetting of the bed occurred during the first hour of sleep; but from that time till she awoke, there was no water passed. During the day, she had the power of retaining the urine for a very long time. Her mother, by whom she was brought to me, told me that she herself had suffered from incontinence of urine up to the age of puberty, when it spontaneously ceased; and that her son, up to the age of twelve, had had the same infirmity.

In July, 1860, a lady, aged 40, brought me her son, who was about to go up for his examination for admission to the military school of St. Cyr: she stated that he had had nocturnal incontinence of urine since he was seven or eight years old. The symptoms, however, did not show themselves more than once or twice a month. This, however, was sufficient to render residence at a military school impossible. The lady informed me that she herself had had the same infirmity till she was twelve years old: she was then a very tall girl, and had attained puberty a year previously. Her mother, who had reckoned on puberty bringing with it a cessation of the incontinence of urine, finding that it was not so, became persuaded that her daughter did not sufficiently set herself to overcome the infirmity, and, acting on this belief, inflicted one morning in presence of the ladies' maid one of those chastisements which are hardly ever administered except to very young children. This punishment produced a deep impression on the girl's mind; and from the date of it, she never again wet her bed.

Similar cases are recorded. It is here, gentlemen, that incontinence of urine and epilepsy present a certain point of contact. It is not at all unusual to meet with cases of epilepsy in the hereditary antecedents of the patients: it is not an overstrained inference to see in the incontinence of urine one of those transmutations of one neurosis into another, to which, upon several occasions, I have called your attention. My view is rendered the more tenable by the fact, that the transmutation sometimes occurs in the same individual, as in some cases which I have brought under your notice. In one of the cases to which I allude, the patient was a child who had been in the habit of wetting his bed till he was nine years old; and who, when cured of that infirmity, became subject to epileptic fits. Hysteria very often plays the same part as epilepsy in these transmutations. Finally, when lecturing on spermatorrhœa, I pointed out the relationship which exists between that affection, nocturnal incontinence of urine, and the neuroses: I showed you that in a great many cases, seminal incontinence succeeded at puberty to the urinary incontinence of childhood, and that impotence at the age of manhood was the accompaniment of seminal incontinence. Then, again, to crown the whole, you have seen epilepsy or insanity terminate the morbid series, thus proving, that each of its parts is of the nature of a neurosis.

I have said, that we must admit our ignorance of the causes—I mean the exciting causes—of incontinence of urine. In some cases, as in that of our young lad in St. Agnes Ward, phimosis was the starting-point of the morbid symptoms: this fact may be explained by the sebaceous secretion between the prepuce and the glans penis, causing an irritation extending by sympathy to the bladder, and rendering that organ more easily excited to contraction by the accumulation of urine in its cavity. Cases of this kind, however, are exceptional; and as a general rule, it may be stated, that the immediate cause of the infirmity cannot be detected.

Perhaps we are better acquainted with the proximate or organic cause of incontinence of urine.

The opinion was long ago stated—and it is still held by some physicians—that this infirmity results from feebleness of constitution: that the subjects of it are weak children of lymphatic temperament, flabby flesh, pale visage, and fair hair. A more attentive examination of the facts will show you, that this opinion is too exclusive. No doubt, nocturnal incontinence of urine is met with in subjects of delicate constitution who are devoid of moral and physical energy; but then, it is met with nearly as frequently in persons possessed of every attribute of vigor, and in the enjoyment of perfect health.

Not to pursue this topic any farther, I repeat, that nocturnal incontinence of urine is a neurosis; and I now add, that it is a neurosis manifesting itself by excessive irritability of the bladder. In fact, the immediate cause of incontinence is this excess of irritability in the muscular fibres of the bladder. Such is the conclusion I have arrived at, in seeking to explain to you the success obtained in treating this affection by belladonna: it is one proof more of the truth embodied in the aphorism of Hippocrates: "*Naturam morborum curationes ostendunt.*"

You know, gentlemen, what takes place in the act of micturition. The urine secreted by the kidneys flows by the ureters into the bladder, where it accumulates, and is there retained by the obstacle to its egress presented by the sphincter. When a greater or less quantity of urine has been accumulated in this manner, the sensation of a want to urinate is felt, which sensation is due to the muscular fibres of the bladder contending against the resistance offered by the sphincter. Although this sphincter, like all the muscles of organic life, is placed external to the domain of the will, its contractions may nevertheless be rendered more energetic by an exercise of the will, so as to enable the individual at pleasure to retain the urine for a longer or shorter period: the constriction of the neck of the bladder by the fibres of its own sphincter is increased by the contraction of the accelerator urinæ and the levator ani muscles. But at last, the want to urinate becoming more pressing, in consequence of the greater accumulation of urine, the retaining, are overcome by the expulsive forces, and micturition takes place. It is the result simply of the contraction of the muscular fibres of the bladder: this action is generally sufficient to empty the bladder, particularly when the sphincter offers little resistance, as is the case in very young children. In older children, and in adults, this action is not always adequate, and then the diaphragm and abdominal muscles assist in micturition.

It is necessary, therefore, for the accomplishment of voluntary micturition, that the resistance of the vesical sphincter be sufficiently powerful to counterbalance the action of the muscles which tend to expel the urine from its natural reservoir.

Should the resistance of the sphincter not be sufficiently powerful, the urine will escape involuntarily—there will be incontinence.

This defective resistance may be either absolute or relative. It may be absolute, as in cases of paraplegia and paralysis. There was an absolute deficiency of resistance in the patient who occupied bed 1 of St. Agnes Ward, who, as you will recollect, suffered from saturnine symptoms, and had paralysis of the bladder. But under such circumstances, there will not only be symptoms of nocturnal incontinence, but also an involuntary escape of urine by day as well as by night; and as the bladder itself will participate in this paralysis, the flow will take place in a passive manner, and not in a strong stream, as observed in the case at present specially under consideration.

In nocturnal incontinence of urine, the deficiency in the resisting power is only relative, inasmuch as then it is the irritability of the muscular fibres of the bladder itself which is augmented.

This irritability, and let me add, this exaggerated tonicity of the bladder, are demonstrated by the fact stated by Bretonneau, a fact which I have also verified, that the majority of the patients suffering from nocturnal incontinence of urine piss by day in a very strong stream. This irritability appears to me to be also demonstrated by the fact, that when the patients are asleep, the penis is almost always in a state of erection. May it not be assumed, therefore, that the bladder participates with the external genital organs in a condition of erethism?

Along with the augmented tonicity of the muscular fibres of the bladder, there may exist a certain degree of atony of the sphincter, and then the patients, as in the case of the young girl of bed 3, St. Bernard Ward, have great difficulty in retaining their urine even during the day. But this, let me say at once, is an unusual occurrence; the vesical sphincter, at least when under the control of the will, preserves its power intact. What shows that here we have nothing to do with atony of the vesical sphincter is, that the individuals when awake are able to retain their urine for a very long time. The young girl, the subject of our first case, told us frequently, that it was so with her. Now, is it possible to explain the existence of this power, if we admit that the contractility of the sphincter is enfeebled?

The incontinence is nocturnal; the incontinence supervenes during sleep, and when sleep is most profound. Interrogate the patients, or rather interrogate the persons who can give information regarding them, and you will learn that persons who wet their beds are generally deep sleepers. Such being the case, it is easy to understand the phenomena.

During sleep, the voluntary contractility of the vesical sphincter being completely annihilated, its organic contractility is insufficient to contend against that of the muscular fibres of the body of the bladder, and consequently involuntary micturition occurs. To produce this effect, it is not necessary that there should be any great accumulation of urine.

Let us now return to our patient in St. Bernard Ward. Although she got up several times during the night to make water, either awaking spontaneously, or being awoke for that purpose so that she might avoid wetting her bed, this occurrence was not prevented, the wetting the bed still taking place towards morning during her last hours of sleep. This is what happens with many of those affected with this infirmity. There is, therefore, no ground for supposing, as do certain authors, that nocturnal incontinence of urine is due to excessive distension of the bladder, and that the overflow is the result of engorgement, as it is generally in retention.

Gentlemen, connected with the question now before us, there is an important point which requires to be elucidated.

The young girl in St. Bernard Ward sensibly improved from the very commencement of the treatment which I prescribed. After taking the belladonna for a fortnight, she was ten or twelve nights without wetting her bed, whereas previously, she had done so every night at least once, and often twice. Meanwhile, her catamenia having appeared for the first time, the nursing-sister, believing that a crisis had come which would radically cure the infirmity, discontinued the administration of the medicine. The incontinence soon returned.

The opinion is pretty prevalent even with physicians as well as with the public, that the establishment of the menstrual function, and, in a more general way, the influence of puberty, are curative of nocturnal incontinence of urine; there is also a prevalent notion, that when the infirmity dates from birth, it will yield naturally at the period of the first dentition, or if not then, at the second; and in the same way, if the period of puberty pass without a cure, it is hoped that marriage, or at all events the birth of the first child, will lead to the desired result. Gentlemen, beware of such illusions! If you participate in them with the family of the patient, when they seek your advice under the circumstances, you will expose yourselves to very awkward disappointment. Unquestionably, as incontinence of urine often ceases spontaneously, so may this cessation take place at some particular epoch, such as that of dentition, puberty, marriage, or the first childbearing; but I regard such an occurrence as a mere coincidence, judging from numerous cases which I see every day, in which the symptoms remain unchanged, although the various changes referred to take place in the organism.

Once, however, I think I met with a case in which there was an evident relation between a first pregnancy and the cure of nocturnal incontinence of urine. The patient was a girl of 18, who from birth had been in the habit of wetting her bed. A vain hope had been entertained that dentition would lead to recovery; at 14, menstruation was established, but there was no amelioration; some one then told the family that marriage would accomplish a cure. The young lady possessed all that was desirable in respect of beauty and fortune; but how could she be given in marriage without telling the man of her infirmity? And where was to be found a conscientious man who would incur the hazards involved in such a marriage? There was found, however, an individual without fortune, who did not shrink from the match. He married the lady, who immediately became pregnant; and ceased from that time to wet her bed. This is the only case of the kind with which I am acquainted; and therefore, I cannot say too decidedly that such cases are altogether exceptional.

Intercurrent diseases occurring in the subjects of incontinence of urine have an influence upon that infirmity. Acute and febrile diseases, particularly the eruptive fevers, whilst they continue, suspend the manifestations of incontinence; and this beneficial influence is sometimes prolonged for some time after the fever has ceased. You, of course, recollect the young girl who was the occupant of bed 22, St. Bernard Ward. I treated her fruitlessly for eighteen months. In the summer of 1861, she had an attack of dothinenteria, and during her convalescence, she passed twenty-four nights without wetting her bed. At a later date, the habit returned. Do you not perceive in these facts an additional feature of analogy between nocturnal incontinence of urine and the other neuroses?

Gentlemen, many measures have been tried for the cure of incontinence of urine, which although it be not a serious affection, and though it be one which generally ceases spontaneously, is nevertheless a deplorable infirmity which medical practitioners are constantly being required to treat. Among the therapeutic agents which have been employed, belladonna (or atropine) occupies the first place. When administered under the conditions existing in the majority of cases, and in accordance with certain rules, it constitutes an eminently useful, nay almost an infallible remedy. It almost always notably diminishes the frequency of the bed-wetting, and in a large proportion of cases, causes it entirely to cease.

The treatment by belladonna, which I adopted from the practice of Bretonneau, has for a long period rendered me undoubted service; and similar benefits have been obtained from it by Dr. Blache and others. You yourselves have been witnesses of its successful employment in the case of the first of the young girls in St. Bernard Ward. I shall give you a summary of that case, and recapitulate the rules of treatment which I followed.

On her coming into the hospital, I caused this girl to take every night a centigramme [one-seventh of a grain] of the extract of belladonna, in the form of pill: obvious improvement was the immediate result. In place of wetting her bed twice every night, she did so only once: and whilst she went on taking the medicine, without increasing the dose, the occurrence no longer took place every night as before. She passed three or four consecutive nights without wetting her bed: she then resumed the habit, wetting it perhaps from two to six nights in succession. That state of matters continued for several weeks. Not allowing myself to be discouraged by the obstinacy of the affection, I increased the doses of the belladonna, running up the quantity by degrees to ten centigrammes [one grain and three-sevenths]. The dose was taken only once in the twenty-four hours: and night was always the time at which it was administered. Under the influence of the doses of ten centigrammes, the amendment was so great, that the patient passed twenty-two consecutive nights without wetting her bed. She had then a relapse for two nights, after which, eight or ten nights elapsed without any recurrence: she then wet her bed for two or three nights in succession, when again, for ten nights, the accident did not recur.

We had assuredly made way; but were still far from having attained a cure. I persevered in the treatment, and increased the dose of the extract of belladonna to fifteen centigrammes [two grains and one-seventh]. During the last fifty days, the patient has been taking the nightly dose of fifteen centigrammes; and during the whole of that period, she has not once wet the bed.*

Gentlemen, this case shows you that when experience has convinced a physician of the utility of a mode of treatment, he ought to persevere in its employment. In respect of the matter now before us, let me repeat my profound conviction that *belladonna* is the most powerful therapeutic agent in cases of nocturnal incontinence of urine in both sexes. Though I am not absolutely certain that I shall invariably cure my patients by this heroic medication, I feel quite sure that I shall almost always afford them a great measure of relief. Strong in my conviction of obtaining a successful result, I carry out the treatment of the case with patience, and I claim that same patience from those confided to my care: in the case which you have seen me treat, it is for you to say, whether the result obtained corresponded satisfactorily to the attempt. Unfortunately, it is not always possible to get the patients to continue the treatment with the necessary perseverance: as soon as considerable amendment is obvious, patients are apt to believe themselves free from their infirmity, and, disregarding advice, to abandon the use of the remedy. Very soon, there is a recurrence of the incontinence, whereupon it becomes necessary to recommence the treatment, and to continue it for a longer time than would have been requisite had the original instructions been strictly followed.

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* Subsequent to the delivery of this lecture in the theatre of the Hôtel-Dieu, no relapse occurred: that the cure was really radical can be affirmed with certainty, as the patient remained in the hospital till the end of 1863, as an attendant in the wards.

Here, gentlemen, is a summary of my rules for carrying out this treatment. I order the patient to take every night at bedtime, one centigramme of the extract of belladonna, or half a milligramme [$\frac{1}{140}$th of a grain] of the neutral sulphate of atropine, which may be administered in pills, or in any other form. If the manifestations of incontinence diminish in frequency under the influence of this dose, I continue the remedy in the same dose for a certain period; but if after eight or ten days, the amendment does not progress, I increase the dose to two centigrammes of the extract. I make no change in the time of administering the remedy. Following the same rule, and guided by the same indications, I progressively increase the dose, till I get it up to twenty centigrammes or even more, according to the more or less decided character of the therapeutic action obtained, and the greater or less tolerance for the drug shown by the individual.

When the amelioration has lasted long enough to justify me in believing that the cure is radical—when for three, four, or five months there has been no recurrence of the incontinence—I do not abruptly discontinue the treatment, but I gradually diminish the dose during from two to ten months or longer, according to the circumstances of the case, which may be one of old standing, and consequently more inveterate.

Belladonna administered according to these rules, and administered with great perseverance, is, I say again, the most powerful therapeutic weapon with which to oppose nocturnal incontinence of urine depending upon excessive irritability of the bladder. In these cases, the belladonna acts by diminishing this excitability, this augmented tonicity of the muscular coat of the bladder: it acts, therefore, in virtue of its physiological properties, which consist in diminishing the contractility of the vesical muscular fibres. These properties have been principally elucidated by the experiments which Dr. Comaille made upon himself.

While I reiterate the necessity of gradually augmenting the dose of this remedy, and continuing to give it long after the incontinence has ceased to manifest itself, I must tell you that frequently when the belladonna has produced the desired effects during the first months of the treatment, it is a good plan to suspend its use, administering in place of it for a time preparations of *nux vomica*.

When nocturnal incontinence of urine does not depend exclusively upon an excess of irritability of the muscular fibres of the bladder, but also on atony of the sphincter, belladonna does not prove so beneficial. In some such cases, there is at first a beneficial effect produced, by its lessening the tonicity of the muscular coat of the bladder when that tonicity is in excess, and so facilitating the resistance of the sphincter; but it is generally against the atony of the sphincter that we have to contend, to arrive at the desired result. Now these are the conditions under which the preparations of *strychnine* are indicated. The preparation to which, on account of convenience in prescribing, I give the preference, is the syrup of the neutral sulphate: on former occasions, I have given you the formula for making it, and have also pointed out the mode in which it ought to be administered.

This is the medicine to which recourse ought to be had at once, when the incontinence is exclusively the result of atony, not merely of the sphincter, but of the entire bladder. This kind of incontinence is recognized by its being both diurnal and nocturnal; and also, by the patients passing their water in a weak stream, while the very opposite is observed in those whose incontinence is exclusively nocturnal.

I prescribed strychnine for our patient of bed 1, St. Agnes Ward; but although it soon modified the paralysis, so that the patient was able to walk much more easily, and for a longer time than before he began to take it, it produced no effect upon the incontinence of urine. Seeing this, I entertained the idea of having recourse to *compression of the prostate*, which I have already brought under your notice as one of the most powerful means which we possess of curing some forms of spermatorrhœa. I believed that by the aid of the compressor of the prostate, I should be able to act on the neck of the bladder, just as in cases of involuntary seminal emissions, I had acted on the orifice of the ejaculatory canal. I gained my object; for the patient was forthwith apprised of his want to urinate, and so was able, which had not previously been the case, to retain his urine when asleep as well as when awake. Although this is the only case of the kind which I can report to you, it is not on that account the less valuable. I advise you to make a note of it.

In addition to the medical and surgical treatment of incontinence of urine, there is another means which I ought to mention; and which consists in impressing upon the patients the importance, during the day, of resisting as long as possible the want to urinate.

Nocturnal incontinence of urine, as I have already stated, depends upon an excess of tonicity and irritability in the muscular fibres of the bladder. Now, whenever a muscle is in this state of tonicity, of abnormal irritability, tending to spasm, the best means of combating that spasmodic tendency is to combat the tonicity of the muscle. To accomplish that object, it is sufficient to carry extension of the muscle to the utmost degree which is practicable: now, it is by habit, that this degree of extension is obtained.

You all know that great eaters have at last larger stomachs than other persons. This increased amplitude of stomach, primarily occasioned by the presence of too large a quantity of food, becomes at last permanent. The muscular coat, from being constantly distended, loses its tonicity; and consequently, the time comes, when the stomach, even though empty, no longer contracts upon itself. In the same way, habitual constipation leads to abnormal distension of the large intestine. This excessive distension of the stomach and intestine is, let me remark in passing, the cause of flatulence and other symptoms regarding which I shall have to speak to you at length on a future occasion.

What takes place in the digestive canal, takes place in the bladder, as well as in all the organs which are hollow: the muscular fibres which enter into the composition of these organs lose in part their contractility by being habitually distended. This is the fact which has to be applied to the special case now before us.

Everybody knows that the bladder is larger in women than in men: this is partly because it is larger naturally in women, but undoubtedly it is also partly owing to women early acquiring (under the pressure of the social conditions in which they are placed) a habit of retaining the urine for a much longer time than is necessary for men. Some men, however, can acquire a similar habit, and remain many hours without relieving the bladder, while others have no power to resist the first sensation of a want to make water. After a certain age, there may be some objections to retaining the urine for a long time, but none exist in youth, and still less are there any in childhood. We cannot, therefore, too strongly advise children and adults affected with nocturnal incontinence, to retain their water as long as possible when awake. Perhaps to some men of science, this little precaution may seem insignificant: it has, however, not the less its importance, and is an auxiliary to the treatment which I have indicated.

LECTURE LXIV.

GLUCOSURIA: SACCHARINE DIABETES.

Presence of Sugar in the Urine not sufficient to constitute Diabetes.—Transient Glucosuria.—Glucosuria Symptomatic of Cerebral Affections.—Alternating Glucosuria in Gouty Persons.—Persistent Saccharine Diabetes.—May in the first instance be Intermittent.—Symptoms.—Polyuria.—Character of the Urine.—Excessive Thirst.—Period of Wasting: it may be First Period.—Phthisis.—Spontaneous Gangrene.—Intercurrent Diseases and a Febrile Condition suspend Glucosuria.—Pathological Physiology of Glucosuria.—Treatment.—Diet the most important part of Treatment.

GENTLEMEN: I have to speak to you to-day of a patient admitted some days ago to the clinical wards: he occupies bed 16, St. Agnes Ward. He suffers from saccharine diabetes.

This man is 36 years of age, and apparently is of robust constitution. However, upon interrogating him as to family antecedents, I learned that two of his brothers had died from disease of the lungs. Although we found in him no sign of thoracic disease, it was incumbent to take into account this piece of family history, as it gave ground for fearing that, in virtue of the hereditary predisposition, pulmonary phthisis, a complication so frequent in saccharine diabetes, might set in sooner than in other diabetic patients. The patient told me that he had been subject habitually to profuse sweating in the hands and feet; but that these sweats had completely ceased from the time of the appearance of the first symptoms of the affection which brought him to the hospital. He maintains, however, that his present symptoms did not come on in connection with a sudden suppression of the customary sweating, but on the contrary that the sweating ceased after the appearance of the diabetic symptoms, this appearance having been sudden, which is unusual. Likewise, all at once, the disease assumed that particular form which has been called *phthisuria*, being that form of phthisis in which there is excess in the quantity of the urinary secretion, and which is characterized by the phenomena of consumption, which, as a general rule, do not show themselves in diabetes till the last stage of the disease.

The patient states that last year, in the month of June, consequently about nine months ago, he was mowing in the meadows, the weather being exceedingly hot. To appease a devouring thirst, he swallowed an enormous quantity of milk and water on returning home. From that day, his health, which till then had been unexceptionable, underwent obvious deterioration. He became tormented by unquenchable thirst, and at the same time completely lost his appetite, so that for a fortnight he had no relish for food. This, gentlemen, is a peculiarity in the case which you must note, for it is opposed to the general rule, an excessive appetite being usually met with in diabetes.

The patient naturally disposed to take alarm, observed himself with the greatest care, and entered into the minutest details with me. He told me, that taking fright at his want of appetite, he had had himself weighed from time to time, and that he found he had lost some kilogrammes in weight. He had also remarked, that when he made water in his garden, an unusual looking trace was left on the ground and grass; and that the bees alighted and settled on the place he wet, to derive from it the juices which they usually rob from the corollæ of flowers.

Uneasy as to his condition, which was becoming worse every day, he sought admission to the hospital at Rheims, where he was under the care of the late Dr. Landouzy, one of our worthiest and most regretted provincial brethren. Dr. Landouzy subjected him to a treatment in which alkaline drinks played the most important part, and under the influence of which the symptoms improved so quickly that he requested to be allowed to return home. His thirst became less urgent, and his plumpness returned. However, he was soon obliged to return to the hospital; but he again left it, having again notably improved. As formerly, his amendment was of short duration: in despair, he resolved to seek farther advice, and with that view came to Paris to seek a cure at my hands, but this I could not promise him.

His glucosuria is of a bad kind, against which medical treatment cannot prevail. I can only, like my honorable colleague at Rheims, check the symptoms; and like him, I can only temporarily arrest them. Whatever I do, the disease will resist my efforts; and if this man agree to remain with us, you will see the extreme symptoms of consumption supervene, and ultimately prove fatal.

Gentlemen, do not lose sight of this man, for it is not often that you have an opportunity of studying saccharine diabetes in our hospitals. This does not arise from the rarity of the disease: on the contrary, as has been remarked by Copland, by Graves (of Dublin), and by his countryman Sir Henry Marsh, glucosuria is common—much more common than is generally supposed. In many cases, it escapes observation, in consequence of its not producing any great disturbance of the system, the patients pursuing their usual occupations without seeking medical aid. When diabetic patients come to consult a physician, it is often for anomalous symptoms, the signification of which they do not know, and which do not excite any suspicion in their minds of the possibility of their being diabetic.

Sir Henry Marsh, in the course of his researches into the nature of this disease, interrogated, upon this point, all the patients whom he thought might have diabetes; and he found it in many of them who only complained of dyspeptic or nervous symptoms. In my own clients, as well as in the patients of other physicians to whom I have had the honor to be called in consultation, chemical analysis has revealed glucosuria in the urine of persons, who, to all appearance, had no serious symptoms, and were apparently in the enjoyment of excellent health.

Let me add, however, that since the publication of the researches of modern observers have called attention to the disease, it has been much less common than formerly for glucosuria to pass unnoticed. In some cases, the symptoms as stated by the patients have no significative character; but amid the different symptoms which they mention, we can generally lay hold of some signs of great value, by which we are put on the way to a correct diagnosis, which we complete by examining the urine and finding that it contains glucose.

However, gentlemen, before laying before you the remarks which I have to make on saccharine diabetes, there is a point on which we must have no misunderstanding; viz., *that the presence of sugar in the urine is not enough to constitute the disease called saccharine diabetes*, any more than the presence of albumen in the urine is enough to constitute Bright's disease.

Our great physiologist, Claude Bernard, to whom medicine is indebted for the most exact ideas we possess regarding the pathogeny of diabetes, has taught us that sugar appears temporarily in the urine under a great number of different conditions of the economy. This transient, temporary *glucosuria* may supervene from the ingestion of particular articles of food or medicine, such as ether for example, which seem to act by imparting an augmented activity to the glucogenic function of the liver. In these cases, it is true, the sugar appears only in small quantity. There are other cases, however, in which it appears in greater abundance, as in persons under the influence of violent emotions and moral impressions. This *acute glucosuria*, to adopt the name given to the affection by the eminent professor of the College of France, ceases spontaneously and quickly.*

The appearance of sugar in the urine of hysterical and epileptic patients, which has been pointed out by Drs. Michéa and Alvaro Reynoso, as well as by others, is likewise referable to a temporary disturbance of the nervous system. The fact, however, I must state, has not been confirmed by all practitioners, although it is given by the physicians who I have named, as the result of a great many minutely reported cases.

This accidental glucosuria may be also the consequence of an affection seriously implicating the nervous centres. It will be transient or permanent, according to the transient or permanent character of the affection on which it depends.

Goolden, Istrighson, Paggle, and others, have reported cases of temporary glucosuria supervening consecutive to concussion of the encephalon from falls or blows on the head.

From the examples which I could cite, and some of which are detailed in the memoir published by Dr. Fischer,† I shall recapitulate the case given by Dr. Szolskaski (de Savigny-sur-Beaune) in the Union Médicale for 23d April, 1853. The patient became glucosuric after a fall, in which he received a fracture of the cranium, with depression of the bone at the middle of the sagittal suture. On the day following the accident, symptoms of diabetes declared themselves. There were urgent thirst, polyuria, and the urine contained glucose. The glucosuria ceased spontaneously at the end of five weeks, at the same date that the other symptoms disappeared.

To such cases ought to be added those originating in falls on the feet, succussions, violent efforts, fractures of the vertebræ, and blows on the back, chest, or limbs. The development of diabetes under such circumstances is explained by the traumatism of the spinal nerves, sympathetic nerve, and spinal marrow—a traumatism acting more or less directly on the ganglionic elements or sympathies which enter into the composition of the cerebro-spinal nervous system, and which are accumulated most abundantly upon the floor of the fourth ventricle, where they terminate; and whence proceed the nerves of organic life which preside over the vaso-motor visceral system.

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* BERNARD (Claude): Lecons de Physiologie Expérimentale Appliquée à la Médecine. Paris, 1853.

† FISCHER: Archives Générales de Médecine, for September and October, 1862.

It is, however, important to recollect, that traumatic glucosuria, though usually transient, sometimes continues for a long time after the cessation of the cause in which it originated. Eight cases of this kind derived from different authors are recorded in Fischer's work.

Gentlemen, A. Becquerel,[*] and before him Dr. Leudet (of Rouen),[†] have described cases of persistent glucosuria symptomatic of serious alterations of the brain; and more recently, Dr. Levrat-Perroton took as the subject of his thesis, sustained before the Faculty of Paris in 1859, *a case of glucosuria* caused by a colloid tumor inclosed within the fourth ventricle.

You have seen the man, between thirty-five and thirty-six years of age, who came into St. Agnes Ward for an attack of polyuria which seemed to date far back. According to the information we received, he had at a previous time passed saccharine urine, but on admission to our wards his urine was free from sugar. He fell into a state of profound cachexia, and speedily sunk, having had *purpura hemorrhagica* in the last days of his life.

The following is an account of the examination of the brain made by Dr. Luys: The anterior wall of the fourth ventricle was more vascular than in the normal state: the large venous trunks were delineated on its surface. Moreover, on looking more closely at the brain, some tawny spots were observed disseminated in the upper parts below the superior processes of the cerebellum: there were also observed some other similar spots below the origin of the auditory nerves.

In making a transverse section of the region, Dr. Luys ascertained that the whole of the gray substance was the seat of unusual vascularity, which gave it a pink color. The histological examination of the tawny spots showed that these unusual discolorations were due to fatty degeneration of all the nervous cells of the corresponding parts. These nervous cells, in place of presenting regular shapes, fringed prolongations, and circumscribed nuclei, were all transformed into an irregular granular mass exclusively constituted by aggregated yellowish granulations more or less loosely attached to each other. So complete was the alteration of tissue, that it might be said that the histological elements had reached the last stage of retrograde evolution, and had ceased to exist as anatomical individualities.

Dr. Luys had, in the previous year, communicated to the Biological Society the history of a similar case which occurred in his practice. The patient, a man of fifty, diabetic for two years, was seized in the last period of his existence with all the symptoms of pulmonary phthisis, under which he sunk. This patient had also double cataract.

At the autopsy, there was found great vascularity, with brownish discoloration of the anterior wall of the fourth ventricle, which was also notably attenuated. The histological examination disclosed a remarkable turgescence of the minute capillaries, and showed that the presence of the yellow patches, in some places tawny and brownish, was solely due to a peculiar degeneration of all the cells of the nervous tissue. All the cells in progress of retrograde evolution were filled with yellowish granulations having jagged, half-destroyed edges, and presenting only some scarcely recognizable fragments.

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[*] Becquerel: Etudes Cliniques sur le Diabète et l'Albuminurie. [*Moniteur des Hôpitaux*, 1857.]

[†] Leudet (de Rouen): Recherches Cliniques sur l'Influence des Maladies Cérébrales sur la production du Diabète Sucré. [*Gazette Médicale de Paris*, 1857.]

These clinical facts confirm the results of the remarkable experiments of Claude Bernard upon the production of artificial diabetes, experiments to which I shall have to call your attention when I discuss the pathological physiology of glucosuria.

There are also cases in which temporary glucosuria appears consecutively to irritation affecting directly the liver. Claude Bernard mentions a case in which after a kick from a horse in the right hypochondrium, the patient had sugar in his urine till he recovered from the accident. Similar cases would perhaps have been more frequently recorded, had the attention of physicians been more directed to the relation between diabetes and diseases of the liver.

When the influence of gout on the liver is considered, it may be asked, whether it is to direct irritation of that organ or to sympathy, that we ought to ascribe the alternations of diabetes which occur, and which sometimes seem to be manifestations of the gouty diathesis, and to succeed in fits the other manifestations of that diathesis.

Claude Bernard states that sometimes gouty patients whose urine contains a great deal of uric acid, suddenly present symptoms of saccharine diabetes; and whose urine is then found to contain a considerable quantity of glucose. He supports this statement by the testimony of Dr. Rayer, who had observed cases of this description: and he says, that he himself had seen a very characteristic case of the same kind.

The glucosuria which is accidental, symptomatic, and transitory, does not constitute, I repeat, the special disease for which the name of "saccharine diabetes" ought to be reserved; and in which, examination of the dead body does not reveal any appreciable lesion which is at all characteristic. Glucosuria, in fact, no more constitutes diabetes, than albuminuria, when symptomatic of disease of the heart or serious fevers, constitutes the special affection which is called "Bright's disease."

This remark does not apply to *intermittent diabetes*, or *periodic diabetes*, both of which perhaps are only different forms of true diabetes. In intermittent diabetes, sugar only appears in the urine during digestion, but this form of the affection often at last becomes continuous diabetes: and periodic differs from intermittent diabetes only in the sugar existing at distinct periods, and at long intervals.

To constitute diabetes then, it is not only necessary that the urine contain glucose, but also, that there exist certain special phenomena the value of which cannot be appreciated, unless specially studied in the patients.

In general, the first thing which strikes one is the great, sometimes unquenchable, thirst, which torments the individuals: sometimes, it is the only symptom of which they complain, and for which they apply to the physician for relief. Its importance is so great, and so universally recognized, that when it is present, the idea that diabetes exists is immediately suggested to the physician who is consulted, and the same view often also occurs to the patients themselves, whom it exceedingly alarms. It frightens them still more, if the unquenchable thirst be, as it generally is, coincident with more frequent micturition, and with the passing an increased quantity of urine.

The two leading symptoms, then, of saccharine diabetes are: immoderate thirst, and augmented urinary secretion.

They are not, however, in themselves absolutely decisive as to the nature of the disease; for we meet with both in that kind of non-saccharine diabetes, more appropriately named polyuria or *polydipsia*. We shall even find, that they are perhaps more prominent symptoms in polydipsia than in glucosuria.

If in either of these forms of diabetes, the quantity of urine passed in the twenty-four hours corresponds to the greater quantity of fluid drunk by the patient; if, as generally happens, the quantity of urine greatly exceeds the quantity of liquid taken into the stomach, this excessive urinary secretion is generally much more decided in polydipsia than in glucosuria. It is in cases of polydipsia that from 15 to 100 kilogrammes of urine are passed in the twenty-four hours.

In saccharine diabetes, the urine often, at first, presents nothing remarkable: when it becomes increased in quantity during the course of the disease, the increase is not so enormous as in the cases I have just referred to; and in the last stage, the quantity is sensibly diminished. Finally, it is not uncommon, as was stated in 1845 by Dr. Contour in his excellent inaugural thesis, for saccharine diabetes to exist, although the urinary secretion be normal in quantity. Moreover, in the same individual, immense variations occur in the quantity of urine secreted under the influence of causes which are very different and often inappreciable.

In most cases, however, more than the normal quantity of urine is passed. During the day, the patients are obliged to empty the bladder much more frequently than is usual with them, and during the night, the want to make water is still greater, obliging them to get up four, five, or six times.

The urine presents decided modifications in its physical properties and chemical composition.

When passed, it is at first transparent, and lighter in color than the urine of health, being in some cases nearly colorless; when examined on some particular day, it will be found to have a yellow tint like amber, or to be slightly green: pretty often, it is frothy. Its density is increased; and this increase of density is one of the leading characteristics of glucosuria: from the normal specific gravity of from 1015 to 1022 it rises to 1030, and (according to Bouchardat*) even to 1074; whilst, on the other hand, in polydipsia, it falls to 1007, to 1004, or even lower. When allowed to remain at rest for some time, it becomes whitish, resembling clarified whey in appearance, or, to adopt Cullen's comparison, like a mixture of a small quantity of honey with a large quantity of water. Cullen's comparison is all the more applicable that the odor of honey and water resembles that of saccharine urine, which, when, it dries on linen, leaves traces similar to those of strongly sugared water. Moreover, we have seen in the case of our patient in St. Agnes Ward—and others have recorded similar facts—that when the urine was spilt on the ground, the flies came to suck up its sugar. Finally, the urine of glucosuric patients, deflects to the right polarized light, a circumstance which has been turned to account in diagnosis.

Saccharine urine, like normal urine, is acid in proportion to its properties and chemical composition; and its acidity is sometimes augmented by the presence of carbonic and acetic acids, the products of its fermentation.

It has long been believed, on the authority of eminent observers, among whom it is enough to mention Thenard, that the urine of diabetic patients contains neither urea nor uric acid. New analyses, however, made by MM. Macgregor, Chevreul, Bouchardat, and others, have shown that there is as much *urea* in the urine of glucosuric patients as in that of persons in good health, and that in both it is proportionate to the quantity of azotic aliments which they take. *Uric acid*, it is true, is seldom found in saccharine urine, but the presence of sugar is not incompatible, as has been alleged, with the presence of uric acid, as is evident from the latter being in sufficient quantity to deposit crystals.

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* BOUCHARDAT: Du Diabête Sucré, ou Glucosurie: Son Traitement Hygiênique. Quarto, Paris, 1851.

When albumen is found in the urine of glucosuric patients, it is when they are in the last stage of the disease; it is, therefore, an unfavorable, and not, as Thenard and Dupuytren supposed, a favorable symptom: on this point, clinical observation is in complete accord with the results of physiological experiments.

The pathognomonic character of glucosuric urine is the presence of more or less sugar, but sugar of a particular kind, *glucose*, which is similar to starch-sugar or grape-sugar. I shall not enter into the details of the different analytical processes by which its presence is ascertained. You can always easily obtain *caustic potash*, should you be unable to procure more sensitive reagents, such as the solution of Frommherz, of Barreswil, of Fehling, or of Quevenne. On putting a certain quantity of caustic potash into a glass tube containing the urine, or more simply, into a metal spoon, and heating it at the flame of a spirit-lamp or candle, the liquid, as soon as it begins to boil, will assume a reddish-brown color should it contain glucose, but this will not be the case in respect of any other urine submitted to the same test.

Besides the intense thirst experienced by the patients, there is another symptom of great value, although it cannot be regarded any more than the other as characteristic of saccharine diabetes, since it is also a symptom of polydipsia, in which indeed it often exists in a more aggravated form: this symptom is an excessive appetite—a real boulimia.

This unnatural appetite is observed in almost all diabetic patients. It may exist to such an extent as to seem an impossibility to satiate them; and it is said that some have eaten in twenty-four hours a mass of alimentary matter equal in weight to the third of the weight of the individual's body.

Notwithstanding the ravenous appetite, and the perfect digestion of the food, nutrition is badly accomplished, the nutritive functions being perverted by the disease; and, consequently, diabetic patients rapidly lose flesh, and wasting of the body inevitably leads them to the tomb.

The proposition now enunciated is far too absolute if formulated in this general manner. No doubt diabetes in its last stage is a consumptive disease: in cases such as that of our patient in St. Bernard Ward, the malady, progressing with excessive rapidity, and passing, so to speak, at once into its second stage, immediately induces great emaciation, justifying its being called *phthisuria* or diabetic phthisis; but, generally speaking, this great wasting does not occur in the first stage, which is sometimes very prolonged.

This occurred in the case of a man aged 28, whom you have very recently seen in bed 3 of St. Agnes Ward. Since his admission on the 24th March last, I have been struck with his emaciation and feverish state. The presence of these two conditions have led me to announce to you unhesitatingly a most unfavorable prognosis, and to predict a speedily fatal termination.

Two years ago, this man was thrown out of work, and reduced to be a deliverer of newspapers. He was thus jaded, and without the means of restoring the waste of the body by adequate alimentation. He soon became lean, pale, and at last very weak. Yet, it was not till five weeks had elapsed, that he began to feel intense thirst and increased appetite. He had been three weeks in this state at the date of his admission to the hospital: he then had a very marked typhoid appearance: his step was tottering, his look expressionless, his countenance sad, and his tongue dry and rough. He had headache. Eight days previously, he had had epistaxis. His liver was enlarged, and very hard: it extended three finger breadths below the false ribs, and occupied the epigastric region: it was neither hobnailed nor painful. The pulse was 112, small, and not rebounding. The skin was hot and dry.

When admitted to our wards, the patient was drinking from nine to ten litres of fluid daily, and passing a proportionate quantity of urine. The specific gravity of the urine was from 1.029 to 1.030 in place of 1.015, the natural weight: it reduced, with energetic action, the solution of Frommherz, and assumed a dull red color, when boiled with caustic potash. The disease was evidently saccharine diabetes.

I prescribed ten grains of levigated chalk to be taken daily; and I ordered at the same time a restorative diet. The treatment, however, produced no improvement. Ten days later, I tried the effect of the inhalation of oxygen, which was continued for five days; and this also was productive of no amelioration. Two days later, on the 11th April, the fever increased, the appetite failed, and the thirst diminished: at this time, the patient was not drinking more than about two litres of fluid a day, and passing a proportionate quantity of urine. His tongue was dry, his debility extreme; and he had fallen into such a state of marasmus as to be unable to leave his bed. He died two days after this in a state of sub-delirium, having been ill only five weeks. During the last three days of his life, the urine was not at all copious, but it nevertheless contained a large quantity of sugar. It is right to add, that from the time this man came into the hospital, I was struck by the almost bronzed appearance of his countenance, and the blackish color of his penis.

At the autopsy, we did not find any morbid state of the suprarenal capsules. The kidneys were neither more voluminous nor more vascular than natural: they were neither in a state of hyperæmia nor hypertrophy; for from the disease not having been of long duration, and the urine not having been excessive in quantity, they had not suffered from excessive functional action. It was otherwise with the liver, which was at least twice its normal volume: the length of the right lobe was nineteen centimetres: the left lobe, which extended to the spleen, was twenty centimetres in length: and the total length of the liver was thirty-four centimetres. The entire surface of the organ was granular: it was of a uniform grayish-yellow color: it was very dense, resisting pressure so much as to prevent penetration by the finger. It creaked under the scalpel; and the surface of the cut was granular in place of being smooth. There was well-marked cirrhosis; but the cirrhosis was hypertrophic. The morbid change did not involve the fibrous, so much as the secreting tissue. The fibrous capsule, and the trabeculæ which segment the liver, were increased in thickness; and there was a still more decided augmentation of the volume of the acini, which were visible projecting from the surface, and also visible on the surface of the section, to which they gave a granular character. Thus, there was hypertrophy of the liver consequent upon excessive functional activity, hypertrophy specially involving the secreting tissue of the organ. This was evident upon microscopic examination, by the aid of which one could see that the hepatic cells, so far from being destroyed or atrophied, were increased in volume and in number.

Gentlemen, I cannot too earnestly call your attention to this morbid alteration of the texture of the liver, which is completely in accord with the theory of Claude Bernard. The glucose increases within the system from the time that it appears in large quantity in the urine; and the result is hypertrophy of the secreting tissue of the organ by which the glucose is produced.

Grant that the immediate cause of glucosuria is a particular state of the liver—has it not elsewhere a more remote starting-point? I likewise attach special importance to the anatomical state of the floor of the fourth ventricle. Now, I ought to tell you, that there was nothing different visible to the naked eye in the fourth ventricle of this man from the appearance presented by the fourth ventricle in any other subject. The vascularity was not greater, and the color was the same. Moreover, microscopic examination did not disclose, amid the cells of the nervous tissue, or beneath the lining membrane of the ventricle, the hematic deposits, the granular globules which have been pointed out as occurring in some cases of diabetes.

It appears, then, that in these cases there is complete proof of lesion of the liver, of its being hypertrophied, and in a state of abnormal functional activity, the result being glucosuria.

Gentlemen, you will often see diabetic patients not only not become thin, but actually become fat. A near relation of my own has been affected for six years with saccharine diabetes, accompanied by voracious appetite; and yet, he has manifestly increased in plumpness. There has in this case been no deterioration of general health nor of mental capacity.

Graves relates a similar case. The patient was a gentleman of Dublin, who had had glucose in his urine for seven years: his appetite was wonderful, his physical vigor extraordinary, and he continued to direct with great intelligence and activity extensive agricultural concerns which he had in the country.

It has been said, that in consequence of the *cutaneous secretion* being badly performed in diabetic subjects, their skin is dry. As a general rule, this is true; but nevertheless, there are exceptions to the rule. The person, of whom I was speaking a minute ago, generally had his body bathed in perspiration; and Graves mentions patients in whom he had seen copious sweating. Profuse perspiration is sometimes, though very exceptionally, met with even in the second stage of the disease, in which the skin is almost invariably exceedingly dry.

Coincident with this perversion of the cutaneous functions, there is another symptom, one which is met with much more frequently in women than in men: I refer to an eczematous eruption in the private parts, sometimes accompanied by very painful itching. When you are consulted by women who are becoming elderly, for intense itching in and around the vulva—when on examining the parts, you find that there is eczema, and learn that it has come on irrespective of the menstrual periods, or of any leuchorrhœal discharge, and that the pain it occasions is so great as to prevent sleep, the probable existence of glucosuria will suggest itself. You will often be told by the patient that this eruption, apparently an altogether local affection, is coincident with excessive thirst, and profuse discharge of urine, which secretion, on applying the potash test, you will find contains sugar.

It is not a very unusual occurrence, that saccharine diabetes should manifest its existence only by anomalous nervous symptoms, which are only explained by referring them to this disease, and the nature of which is first revealed by chance leading to the discovery of sugar in the urine. I say that it is frequently by chance that we are led to a correct diagnosis; for when the glucosuria is unaccompanied by polyuria, the quantity of urine passed in the twenty-four hours remains normal, or is very little augmented. There is also very often, I repeat, an absence of the dyspeptic symptoms of which I spoke at the beginning of the lecture, as well as of voracious appetite and excessive thirst.

The anomalous nervous symptoms to which I refer may consist in diminished motor power, or in perverted sensibility. A remarkable case of exalted sensibility occurred in my practice.

The patient was a woman about sixty, who although she retained an appearance of perfect health, had, for three years, complained of feeling constant pains in the whole of the right side. These pains, which the patient compared to twingings and cramps, sometimes aggravated momentarily, allowed her no respite: touching the parts increased the pains, and even the mere contact with her clothes often produced this effect, though somewhat firm pressure on the painful parts occasioned no disagreeable sensation. Notwithstanding this hyperæsthesia, the affected side retained, as perfectly as the other, its motor power and muscular strength.

The general health, I repeat, was otherwise good: all the functions of organic life seemed to be performed with the greatest possible regularity. The lady's appetite remained as good as it ever was, and there was no dyspepsia: latterly, she had had usually a slight degree of constipation. The nervous symptoms had continued three years, but it was only for one year that she had been known to have glucosuria. At this date, and for some days previously, the patient having suffered from somewhat urgent thirst, attention was directed to her urinary secretion, although it was not much more copious than usual. Upon being chemically examined, it was found to contain a considerable amount of sugar. From that time, the proportion of sugar varied much, sometimes entirely disappearing, and at other times reappearing in large quantity. The nervous symptoms have not undergone any change.

Enfeebled vision, premature presbyopia, is one of the most common as well as one of the most remarkable of the symptoms of saccharine diabetes, which are referable to the nervous system, symptoms which become more and more prominent as the disease advances.

A man, for example, in the prime of life, tells you that for some time his sight, hitherto perfect, has obviously deteriorated—that for some time he has been unable to read without placing the book at a greater distance from him than was formerly necessary, and that then spectacles had become requisite—that with each succeeding month he had to use stronger and stronger glasses. From this fact alone, you have reason to think that the patient has either albuminuria or glucosuria. This symptom alone, even when others are absent, will suggest to you the propriety of examining the urine, and by that examination your diagnosis will be cleared up.

This presbyopia, an ordinary symptom in persons affected by saccharine diabetes, increases rapidly: it is observed in the first stage, and being dependent, as I have been telling you, upon disorder of the nervous system, becomes more evident as the disease approaches nearer to its second stage. In some cases, transient or permanent amblyopia is observed in diabetic subjects. It is the result of an organic change in the retina; and is only met with in the second stage of diabetes. At other times, cataract, generally cataract in both eyes, is developed in diabetic subjects, during the latter months of their existence. The reality of diabetic cataract has been established by the researches of Claude Bernard; and the cases have been collected by Dionis, Leudet, and Græfe. In a memoir by Lécorché,* you will find an interesting discussion on the nature of these cataracts, and the manner in which they are formed.

During the second, or to speak more correctly, the consumptive stage of diabetes, which in some persons sets in suddenly, the digestive functions become impaired: the appetite is depraved, and the patients have a disgust for food: they suffer from gastric pains, which increase after eating, from nausea, eructation, vomiting, and diarrhœa, which latter, after alternating with constipation, at last becomes very profuse. The mouth, acid and dry, as in persons suffering from thirst, is hardly moistened by the thick, frothy saliva which forms whitish tracks upon the bright red tongue, on the mucous membrane of the cheeks, and on the commissure of the lips. Although there be no sugar in this saliva, diabetic patients often complain of always having a sweet taste in the mouth. This, Claude Bernard explains by comparing the phenomenon to one analogous to it observed in dogs into whose bloodvessels a decoction of meat has been injected: they immediately lick their lips, thereby indicating the existence of an agreeable sensation. He says, that there is ground for believing, that both in the dogs experimented on, and in the diabetic patients, the substance (existing in the blood in large quantity) is carried with the blood into the capillaries of the mucous membrane of the mouth, where it acts on the sentient extremities of the nerves, as if it had just been directly absorbed by that mucous membrane. But though there be no sugar in the saliva of diabetic patients, their sputa contain it: the sputa consist of bronchial mucosity, secreted by the patients frequently in great abundance in consequence of their very often becoming phthisical in the last stages of the disease.

Under these circumstances, they are subject to a dry cough, which seems to be excited by a troublesome tickling in the larynx. This cough soon becomes a source of anxiety; and auscultation, which at first yielded only negative signs, reveals the existence of pulmonary tubercle, which passes rapidly through the different stages of evolution.

Then, likewise, the derangement of the nervous system becomes more manifest. The mind becomes affected, and hypochondriasis, which in a few cases appears as one of the first symptoms of the disease, assumes formidable proportions, and attains such a degree as to be insanity. The procreative power, often increased at the beginning of glucosuria, diminishes, and is ultimately entirely lost. Sensations of internal heat, alternating with rigors, and a greater sensitiveness to external cold, concur in giving proof of perturbation of the nervous system, and bad state of the circulatory functions. It is then also, that the patients become the subjects of *albuminuria*.

The impaired condition of the circulatory and nervous systems are still more strongly manifested by those remarkable symptoms to which the attention of the medical world has been directed by Dr. Marchal (de Calvi).† I refer to *spontaneous gangrene*, simulating that affection which has been called *senile gangrene*, which is observed in the course of serious fevers; and regarding which I addressed you at considerable length in my lectures on dothinenteria. In diabetes, this gangrene of the tissues is evidently connected, for the most part, with that condition of the arteries to which the name of *arteritis* has been given. But whether this arteritis precede the formation of the fibrinous plug, or whether, on the contrary, it occur as a consequence of the formation of this clot (which is the anatomical cause of the gangrene, by its obstructing the circulation of the blood in the parts about to mortify), it is unquestionable, that the symptoms supervene under the influence of a peculiar predisposition, of a general state of the economy, related to the disease which has glucosuria as its characteristic sign.

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* LÉCORCHÉ: De la Cataracte Diabétique. [*Archives Générales de Médecine* for May, 1861.]

† MARCHAL (De Calvi): Recherches sur les Accidents Inflammatoires et Gangréneux Diabétiques—Théorie Nouvelle du Diabète. Paris, 1864.

This spontaneous gangrene chiefly attacks the lower extremities. Let me here give you the details of a case in point, which occurred in a young American lady whom I attended during her residence in Paris. She had left France to return to New Orleans: during the following year, saccharine diabetes, with which she was affected, had not presented any appreciable modification, when all at once, symptoms supervened which rapidly proved fatal.

Her husband sent me the following history of what occurred. It is contained in a long letter, a translation of which, with your permission, I shall now read:

"Since I saw you, till November last, the state of my wife did not present any material change, but at that date she was greatly prostrated by a choleraic affection. She recruited, however, and, with a wonderful rebound, regained strength and plumpness.

"At that period, acting in accordance with, or rather going somewhat beyond your advice, she abstained from farinaceous food: she entirely discontinued the use of bread. Great improvement was the result. She became strong, and also a little fatter than she was in summer. During the whole winter, she remained free from the itching, and in better health than in the previous year.

"In the beginning of March, a small ulcer, about as large as a pea, appeared on the outside of the fourth toe of the left foot. The foot became inflamed; and with the view of subduing the inflammation and pain, poultices of linseed meal were applied. The limb was kept in a horizontal position.

"About two weeks later, all the bones of the toe were affected and the ulcer extended round the toe, which soon got into such a state that it became necessary to remove it: this was done without the patient feeling the slightest pain. The pains, however, returned, and became so severe, that it became necessary to use opiates freely; and the question was, whether the patient would have sufficient physical energy to resist the progress of the gangrene. It advanced slowly and insidiously; and before stopping, had nearly reached the instep.

"Such was the state of matters on Thursday, 13th April. The position of the patient was considered critical; but immediate danger was not feared. She passed an exceedingly agitated night, but was strong enough in the morning to be able to rise without assistance. She complained of great heat, of great obstruction in the throat, and of a feeling of being suffocated. Her attempts to make water were very frequent, and it seemed as if micturition was difficult. Between two and four in the morning, her agitation increased exceedingly; and the pulse became very perceptibly weaker. Later in the morning the agitation ceased, and gave place to tranquillity, making semblance of the looked-for sleep having come at last: the patient, however, did not sleep, but gradually became weaker, and died about ten o'clock without a struggle, passing away as softly as a child falls asleep."

This spontaneous gangrene occurs sometimes in other parts of the body, as in the chin, nose, continuity of the limbs, and in the walls of the chest: there are even examples of it affecting the lungs.

The case, gentlemen, which I have just related shows how death occurs in glucosuria; but it is very unusual for the disease to terminate in this mode.

As a general rule, diabetic patients sink slowly, succumbing under the complication of tubercular pulmonary phthisis.

In other cases, the patients are carried off by cerebral apoplexy.

In 1846, when doing duty at the Necker Hospital, I had under my care a woman admitted with saccharine diabetes: the disease was presenting nothing unusual in its progress, when, fifteen days before death, the patient was seized with acute catarrh accompanied by high fever. *From that time, the urine ceased to contain glucose.* Eleven days after the commencement of this bronchitic attack, very violent otalgia of the left ear supervened; and next day, we found that there was hemiplegia of the right side, with complete paralysis of the limbs, accompanied by slight muscular contraction, and some diminution of cutaneous sensibility. The face did not seem to participate in the paralysis. The patient fell into a state of stupor and died.

At the autopsy, we found small softened masses seemingly infiltrated with blood, situated in the corpus striatum, optic thalamus, and some parts of the gray substance near the circumference of the left hemisphere of the brain. The meninges seemed to be in a healthy state; and there was no appearance of any lesion of the dura mater in the situation of the petrous portion of the left temporal bone.

In the lungs, there were tubercular masses in the second stage, and some cicatrized cavities.

The kidneys, though hypertrophied and much injected with blood, were not softened; but in the situation of the cleft, there was more swelling, a deeper red color, and more infiltration.

In my notes of the case, no mention is made of the state in which the liver was found. At the date when the memoranda were taken, the part which the liver plays in saccharine diabetes was not known.

Since the beautiful researches of Virchow have more specially directed the attention of pathologists to the subject of arterial obliteration and the part which it plays,* I have often asked myself whether these local gangrenes of which I was speaking a little while ago, whether the cerebral and renal affections which occurred in the case of which I have summarily given you the history, were not caused by real embolia occupying either small arterial branches or more considerable trunks. I am aware that our illustrious brother of Berlin would entertain very little doubt on that point; but many years, probably, will elapse, before these opinions acquire citizenship in our country, or even in that of M. Virchow. It will still be an open question, whether the arterial obliteration is effected *in situ* by the same pathological process which causes the local gangrene, or whether the gangrene is the consequence of obliteration produced by a migratory clot, or by a local morbid change taking place in the vessel.

In the case, with an abstract of which I have just presented you, I laid stress on the fact, that from the time the patient was under the influence of the acute affection, the urine ceased to contain glucose.

Gentlemen, it is a remark of all observers, that when an acute disease supervenes in diabetes, sugar no longer appears in the urine, so that one might suppose the patient was cured of the diabetes. Now, Claude Bernard has proved by his experiments that perfect activity of the digestive function is the primary essential condition requisite for the liver secreting sugar, and that any deterioration in the performance of that function, be the cause what it may, so long as it lasts, puts a stop to the diabetes.

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* Virchow: Gesammelte Abhandlungen. Berlin, 1862.

Though fever is one of the causes, it is not the only cause of disturbance of the hepatic functions; and some other active pathological condition may produce a similar effect, the patient, during a certain period, not presenting the characteristic sign of diabetes.

In relation to this point, Claude Bernard gives the history of an individual who had diabetes in a very obstinate form. For the first few days, it diminished under the influence of remedies, but tolerance for the remedies was then acquired, and the disease returned with its former intensity.

"In these cases," says the illustrious physiologist, "what takes place is quite natural: by each new medication, the functions are disturbed, including those of the liver, and the production of sugar is arrested, but only to be resumed as actively as ever. We must never, therefore, be deceived by such results, nor consider a patient as cured, in whom we have, by the administration of any medicine whatever, temporarily prevented the appearance of sugar in the urine."

The physician, when he is called in to cases of saccharine diabetes, ought always to have in his mind the judicious remarks which I have now quoted.

Gentlemen, before discussing the question of treatment (the most important part of our subject), it is necessary to enter into some considerations relating to the *pathological physiology of glucosuria.*

Rollo, one of the first writers on diabetes, believed that it originated in impaired digestion, in a derangement of the functions of the stomach, the secretions of which acquired, he supposed, the morbid power of transforming into sugar the alimentary substances which it received.

At the beginning of this century, Nicolas and Gueudeville regarded the disease (which they called saccharine phthisuria) as the result of an affection of the intestines. In their opinion, the chyle, in consequence of a morbid change in the intestinal secretions, in place of being composed of nitrogenous materials, consists of less perfectly elaborated elements, of saccharine matter not suitable for the complete nutrition of the body.

These theories, you perceive, differ little from that which has been advanced in our own day by a chemist, M. Bouchardat.

Cullen—who for a short time had adopted the opinion of Mead, that diabetes is dependent upon a certain state of the bile, an opinion founded on the fact that the disease is sometimes met with in persons suffering from an affection of the liver—Cullen seemed nearer the truth, when, abandoning that view, he returned to the idea (too vague no doubt) that the immediate cause of diabetes is a defect in the assimilating function, that function by which the food is converted into proper nutrient fluids. He makes out, however, but a weak case for his theory, for he adds: "I formerly communicated this idea to Dr. Dobson, who adopted it, and published it; but I must confess that the theory is beset with difficulties, which cannot at present be solved."

It was not till our own time, that the problem was approximatively solved; and the pathogenesis of diabetes still remains, and probably must remain for a long time, enveloped in much obscurity.

The science and art of medicine are indebted for the more precise knowledge now existing regarding the disease to the researches of that eminent physiologist whose name is so to speak linked with it. These researches would possess a real unquestionable value, had their only result been the overthrow of the theory of the chemists, who, judging of what takes place in the living organism by the results of experiments in their laboratories, were assuming the direction of the therapeutics of diabetes. Had Claude

Bernard done nothing more than upset the chemical theories of diabetes, he would have rendered an immense and never-to-be-forgotten service to medicine.

A fundamental fact was at once brought to light. It had always been believed that the proximate principles found in the animal economy were derived exclusively from the vegetable kingdom, which it was supposed had alone the power of producing them, animals only extracting and assimilating them, so as to destroy them. In respect of sugar, in particular, when found in an animal, it was said that it had been introduced with the food; and it was consequently argued, that the quantity of that sugar must vary with the nature of the aliment. Now, as it was also believed, that the saccharine substances, or the feculent substances which are transformed into sugar under the influence of the digestive juices can alone supply it for intestinal absorption, it was again concluded, that it ought to be found in herbivorous animals fed on feculent substances, but that its presence could not be expected in carnivorous animals fed only on nitrogenous or fatty matters, which, it was said, could not be transformed into sugar by the known digestive processes.

Claude Bernard has demonstrated the inaccuracy of these views. He has proved that sugar is found in all animals, and in equal proportions in the different species in the animal series irrespective of the nature of their food. Latterly, G. Colin has shown by numerous experiments, detailed in a memoir read to the Academy of Medicine, that even nitrogenous substances can be transformed into sugar by the process of digestion.

Claude Bernard, however, has shown that the production of sugar in the animal economy is not only irrespective of the nature of the food, but takes place even independently of alimentation, sugar having been detected in the blood of animals who have not lived an extra-uterine life—in the fœtus of birds, as well as in the fœtus of the mammalia.

If food be one source of the sugar, it is evident that it has likewise another. What is that other source?

I could not, gentlemen, without transgressing the limits of my teaching in this place, enter into the details of this important physiological question: I must, therefore, send you to the works of the eminent professor of the College of France for the full development of this important subject. I shall, on the present occasion, confine myself to a recapitulation of the facts which more immediately bear upon glucosuria.

As soon as it was discovered that the sugar in animals does not come only from the food, it became evident that an organ must produce it, at least in part: it became evident also, that there must be a special function by which the elaboration of saccharine matter is effected; and then came the question: What apparatus is charged with the performance of this function?

In searching in the different tissues and organs for this saccharine matter, which analysis has shown to be similar to glucose (the sugar of starch), or, to express the fact more correctly, to the sugar found in the urine of diabetic patients (which differs a little from the sugar of starch), Claude Bernard was struck by the circumstance, that in whatever kind of animal he experimented on, the liver was the only organ impregnated with it. From this, he concluded, that this gland, like other glands, ought naturally to be impregnated with the products of its secretion, just as the kidneys are impregnated with urine, the testicles with spermatic fluid, the pancreas with pancreatic juice, and the salivary glands with saliva. Sugar was also found in the liver of persons suddenly cut off by death when in a state of health. In the liver then, in addition to the bile—the only secretion with which the liver was formerly supposed to be charged—there is elaborated the saccharine matter found in animals which (like the fœtus) cannot have derived it from aliment, at one time supposed to be its only source.

Claude Bernard, in pursuing his researches, attained demonstrative evidence the most unquestionable, that the liver performed this special office of secreting sugar. On analyzing the blood which comes to the liver from the intestines by the vena porta, and that which leaves the liver by the hepatic veins on its way to the vena cava, he discovered that the blood of the vena porta did not contain a trace of sugar, while that of the hepatic veins contained it in large quantity: again, he found that the blood of the vena cava, of the right auricle of the heart, and of the pulmonary artery, contained sugar in quantities progressively diminishing with increase of distance from the liver. Here, then, was an absolute solution of the problem: the saccharine matter was evidently formed in the liver, where it was met with in greatest abundance.

Thus, then, the liver performs two functions: one being the secretion of bile, the products of which, poured into the digestive canal, are subservient to intestinal digestion; the other being the secretion of sugar, the products of which, not excreted like the former, re-enter the general circulation, or at least that part of the circulation extending from the liver to the heart and from the heart to the lungs.

This double function, performed by a single organ in the higher classes of animals, is quite distinct in molluscs, and is still more separate in insects, which have an organ for the elaboration of bile and another for the elaboration of sugar.

I told you, gentlemen, that the saccharine matter secreted by the liver returns into the general circulation, or at least into that portion of the general circulation between the liver and the heart and between the heart and the lungs. Restricting still more, at this point, the limits of my subject, I now reach the more direct applications of physiology to the pathology of diabetes.

Claude Bernard found that upon searching for sugar in animals killed at different periods of digestion, he discovered that in animals killed at an interval of some hours after their last repast, sugar existed only in the tissue of the liver, and in the vessels going from the liver to the lungs. What then had become of the sugar? As none was found in the blood which had traversed the lungs, it must have been destroyed in them, or before it reached them. How is this destruction accomplished? The theories invented to explain this are not satisfactory; and we are consequently obliged to confine ourselves to a statement of the fact, without endeavoring to explain it. Be the explanation what it may, the phenomenon is one of so much importance, that when it ceases, life ceases; what we do know of it, however, is sufficient so far as the question of glucosuria is concerned.

I have just told you, that when the animal is killed fasting, the sugar secreted by the liver is not found in the blood which has traversed the lungs. It is otherwise, however, when the sugar is searched for two or three hours after a repast. At that stage of digestion, sugar may be found in all the arteries and veins of the body: it is found at that stage, even in the renal arteries, in too minute a quantity it is true, to traverse the kidneys, so that no trace of it is contained in the urine, any more than in the other secretions. It may happen, however, that the secretion of sugar is increased to such a degree that some of it passes off in the urine. However that may be, this sort of saccharine overflow beyond the lungs—whether it be or be not greater than natural—continues about three or four hours, after which the sugar is only found in situations short of the lungs.

From these experiments, it results, that the secretion of sugar in the liver presents variations like all the other secretions; and this primary fact will account for what is observed in diabetic patients, whose urine is more or less charged with glucose, according to the period of the day at which it is passed, whether nearer or more distant from the time of meals.

These oscillations in the quantity of sugar in the blood at the different stages of digestion would seem to indicate, that although the nature of the alimentary substances may not exert an influence upon the production of sugar, alimentation is at least the source whence the organism derives the saccharine matter. Claude Bernard, however, has demonstrated that the liver is the sole source of the sugar. I cannot, I repeat, enter into the details of this question, and must, therefore, refer you to the lectures which our illustrious physiologist has devoted to the subject. He will tell you that neither sugar introduced into the intestinal canal nor glucose coming from the reaction of the digestive juices upon the feculent substances augment the quantity of sugar in the liver nor in the vessels leading from it; farther, that the sugar and alimentary glucose are destroyed by the liver, and are therein transformed into a peculiar emulsive substance. Recollect this fact: it is of importance that you know it as a guide to the treatment of diabetic subjects.

Gentlemen, I told you that under certain circumstances, sugar passes by the urine, and that when this occurs, it is the sugar of diabetes, that is to say, sugar which, secreted by the liver, has passed through the lungs before reaching the great circulation. Animals which after having fasted for a long time, eat saccharine or feculent substances, pass urine containing sugar directly derived from these substances. The extreme rapidity with which intestinal absorption takes place in animals after fasting explains this phenomenon. In consequence of this rapidity, the sugar contained in the absorbed fluids is carried in mass towards the liver. There a part of this sugar, passing into the vessels belonging to what Claude Bernard calls the chemical circulation of the liver [*circulation chimique du foie*], is destroyed; the other part is carried into the torrent of that other circulation which he calls the mechanical circulation of the liver [*circulation mécanique du foie*]. This collateral circulation, much more rapid than the former, has as its apparatus the vessels of the portal system, which, in place of being imbedded in the lobules of the liver, circumscribe them, and anastomose with the hepatic veins, which pour their contents into the inferior vena cava. The sugar thus poured in greater or less quantity into the general circulation, passes into the urine, where its presence may be detected for a longer or shorter period. Its presence there, however, is only transient, and is a phenomenon in no way peculiar; for a similar result is observed in respect of all alimentary substances taken in too large quantities. Thus, for example, Claude Bernard, in his "Leçons de Physiologie," mentions a healthy man, who became temporarily affected with albuminuria from swallowing a large number of raw eggs after a long fast. It was observed that some hours after this repast of eggs, the individual's urine became very albuminous; and a certain time elapsed before it regained its normal character.

Though digestion has a remarkable influence, not only on that form of glucosuria in which the sugar is directly derived from the ingesta, and also on the oscillations in the quantity of sugar in the urine and secreted by the liver, this influence (probably depending upon activity in the function of hepatic secretion), is not the only influence in operation.

From the moment that sugar is secreted by the liver, every cause which increases or diminishes the secreting function of that organ will also be likely to increase or lessen the quantity of the sugar in the organism.

Here it is, gentlemen, that the beautiful experiments of Claude Bernard in connection with the production of artificial diabetes, have thrown such a flood of light upon this important question of glucogenesis and glucosuria.

Like all other glands, the liver is under the dominion of the nervous system. Acting on the latter, we may act indirectly on the former, in such a way as to stimulate, modify, diminish, or even annihilate entirely the function with the performance of which it is charged.

Claude Bernard says that if you prick the medulla oblongata of an herbivorous or carnivorous animal in a certain situation within the fourth ventricle (limited superiorly by a transverse line uniting the two tubercles of Wenzel, and inferiorly by another line extending between the origin of the two pneumogastric nerves), after some time, sugar will exist in large quantity throughout the body, and appear in the urine. The irritation excited by pricking the nervous centres is transmitted to the liver by the spinal cord and the branches of the great sympathetic which preside over the functions of the liver, the secretion of sugar is augmented, and the blood, saturated with it, carries it to the lungs in so large a quantity that part passes through them without being destroyed, and reaches the general circulation, whence it is eliminated by the stomach (as we infer by its being found there mingled with the gastric juice), and eliminated to a still greater extent by the kidneys, which accounts for its presence in the urine.

On the other hand, if you divide the pneumogastric nerves in the neck, or divide the spinal cord above the origin of the branches of the great sympathetic which go to the liver, the secretion of sugar is arrested.

You see then, gentlemen, that experimental physiology explains the existence of glucosuria in those cases in which it is connected with lesions of the fourth ventricle of the brain, cases similar to those of which I have given you examples.

Experimental physiology also explains what occurs in cases similar to those reported in Dr. Fischer's memoir, cases in which saccharine diabetes was recognized as the starting-point of more or less serious affections of the encephalon, spinal cord, spinal nerves, and great sympathetic. This is the place to lay before you the beautiful experiments undertaken by a very eminent German physiologist, Dr. Schiff, for the purpose of confirming and testing the experiments and conclusions of Claude Bernard: they throw fresh light on the question which now engages our attention.

Since the date of the celebrated experiment of Claude Bernard, it has been known that glucosuria is produced by pricking the medulla oblongata in the central part of the floor of the fourth ventricle, between the origins of the auditory nerve and the par vagum.

The presence of sugar in the urine results from an excess of sugar in the blood. Now, in reference to this point, Schiff proposed to himself the following problems, which he solved with his usual sagacity.

1. Does the excess of sugar in the blood depend upon there being an abnormally less rapid destruction of sugar in the organism, or upon there being an increased production of it?

2. Adopting either hypothesis: Does pricking the floor of the fourth ventricle act by paralyzing or by exciting the medulla oblongata?

3. How is the nervous action, whether paralyzing or exciting, transmitted to the organs which directly produce glucosuria?

Schiff proved that excess of sugar in the blood is really the result of a modification of the function of the liver, by extirpating the livers of frogs, examining their blood three weeks after the extirpation, and finding that it then contained no sugar. It is very evident from this experiment, that simple pricking of the floor of the fourth ventricle is insufficient to produce glucosuria, that the intervention of the liver is absolutely necessary, and, in fact, that it is the organ by which the sugar is formed.

Schiff likewise confirmed the views of Claude Bernard, by tying portions of the liver in frogs previously rendered glucosuric. He found that by this proceeding the quantity of sugar in the urine diminished proportionately to the artificial diminution of the volume of the liver.

Does diabetes arise from an excessive production of sugar by the liver? or, does it arise in consequence of the non-formation of the ferment [*le ferment*], which, under normal conditions, ought to destroy the sugar contained in the blood?

Schiff, with a view to decide in favor of one or other of these hypotheses, tied larger and larger portions of the liver in frogs which had been made glucosuric. It is very evident that if diabetes is dependent upon the non-formation of the sugar-destroying ferment, a time will of necessity come, when, notwithstanding the partial destruction of the liver, notwithstanding the exceedingly minute quantity of sugar produced by the small remnant of liver, the sugar (being no longer destroyed) will accumulate in the blood, and glucosuria will appear—the frogs will again become glucosuric. Now, the sugar diminished in the urine of these frogs, in proportion as the volume of the liver diminished. Hence it follows, that the cause of diabetes is increased production of sugar by the liver, and not non-formation of that ferment which is destined to destroy the sugar.

The solution of the first question then is, that "glucosuria is the result of an excess in the production of sugar in the organism."

But what is the *nature of the change in the nervous system* which gives rise to glucosuria?

Andral pointed out, more than twelve years ago, that there was hyperæmia of the liver in diabetes. Is this hyperæmia the immediate cause of diabetes? and if so, how is the fact to be proved by experiment?

To produce hyperæmia of the liver, Schiff availed himself of an anatomical peculiarity in the frog. In the frog the liver does not receive the whole of the venous blood of the abdomen: only a portion of it is carried to the organ by the hepatic vena cava, while another portion is conveyed directly to the heart by a second vena cava without passing through the liver. To produce hyperæmia of the liver, it is sufficient to force all the venous blood of the abdomen to traverse the liver; and that can be accomplished by tying the second vena cava. Two hours after the application of the ligature, the frogs were glucosuric.

A similar result follows removal of the spleen; this proceeding causes immediate hyperæmia of the liver, and rapidly consequent glucosuria. Likewise, when traumatic hyperæmia is produced by pricking the liver, the result is glucosuria. But does pricking the floor of the fourth ventricle produce glucosuria by causing hyperæmia of the liver?

According to Schiff, this pricking irritates the vaso-motory nerves of the liver, which causes dilatation of the vessels of the organ; and as a primary consequence, hyperæmia; and as a secondary consequence, glucosuria. This is in accord with Claude Bernard's theory as to the regulating influence which he attributes to the great sympathetic. The explanation given by Schiff rests upon his belief, that the vaso-motory nerves which regulate the contractions of the vessels belonging to the abdominal organs arise from the optic thalamus and crura cerebri, are united in the medulla oblongata, where they are side by side with the other vaso-motory nerves of the body, then descend by the antero-lateral columns, diverge from one another, finally, leave the medulla, traverse the ganglia of the spinal cord, and terminate upon the vessels of the abdominal organs. Supposing this anatomical view to be correct, pricking these nerves at their origin, as they pass in the vicinity of the fourth ventricle, would produce diabetes. Thus, pricking the fourth ventricle will lead to no special result; but any lesion involving the vaso-motory nerves from their origin in the crura cerebri to their termination in the abdominal organs, will produce glucosuria: the floor of the fourth ventricle is, however, the most convenient locality on which to operate, as the vaso-motory nerves are there concentrated within narrow limits. Thus, direct irritation of the vaso-motory nerves of the liver by means of galvanism produces glucosuria: a similar result follows when frogs are poisoned with strychnine or opium, and likewise succeeds a prolonged tetanic state.

If, on the contrary, we were to cut through the anterior columns of the medulla, by which are transmitted the branches of the sympathetic which go to the abdominal viscera, irritation could not be transmitted to the liver, and consequently pricking the fourth ventricle would not produce glucosuria.

It, therefore, follows, that pricking the fourth ventricle produces glucosuria by inducing hyperæmia: that this hyperæmia arises from irritation of the vaso-motory nerves of the liver: that the irritation, again, results from pricking the fourth ventricle, because these nerves are at this point near their origin, collected together; and finally, the irritation is transmitted to the liver by the anterior columns of the medulla.

Thus we arrive at the solution of the second and third problems proposed by Schiff; and which I stated a few minutes ago.*

Glucosuria produced by pricking the fourth ventricle is considered by Schiff to be a special form of the affection; and he calls it *irritative* glucosuria. It has an essentially fleeting character, for it does not continue more than some hours, or a day. It is subject to the general law, that irritant action is rapidly exhausted: the irritation is soon deadened, and quickly becomes extinct.

Paralysis, on the other hand, has durable effects: glucosuria of paralytic origin is likely to be permanent. It is this form of glucosuria which Schiff says he discovered. He produced *paralytic* glucosuria by dividing the anterior columns of the spinal cord, because that operation necessarily implied section of the bundles of vaso-motory nerves which traverse them. The section ought to be made at a point corresponding to the fourth cervical vertebra, or nearer the medulla oblongata. The vessels of the liver, when thus deprived of their vaso-motory nerves, become distended, engorged with blood, hyperæmic; and the animals become glucosuric. Glucosuria thus induced continues for several days, and even for several weeks, as Schiff has shown by experiments on rats and rabbits; it is therefore different from irritative glucosuria, which is very transient. Paralytic glucosuria gives a fair picture of diabetes properly so called, that disease which is so rebellious under treatment. The diabetes produced experimentally by destroying the nervous centres, and likewise gangrenous diabetes, are paralytic forms of the disease.

Gentlemen, it is not only local lesions of the nervous system which promote the secretion of sugar in the liver; Claude Bernard has proved that this result is also caused by general nervous disturbance.

Similar results are also produced by excitement of the hepatic gland, occasioned through the medium of the digestive canal, or in a still more direct manner. Thus, Dr. Leconte, Professeur Agrégé of our Faculty, has produced artificial diabetes in dogs by poisoning them with the nitrate of uranium administered in small doses. Again, Dr. Harley, by injecting into the branches of the vena porta irritating substances, such as ether or a solution of ammonia, has observed that the injection, on arriving at the liver, produced there a local and direct excitement; and after some time, he has detected sugar in the urine of the animal upon which he operated. Finally, I have mentioned to you, on the authority of Claude Bernard, a case of accidental diabetes, which supervened in an individual consecutively to a blow in the region of the liver; and I may add, that I have seen a similar occurrence in a man who received a kick in the right side from a horse.

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* See p. 324.

But, an opposite effect is produced if the excitation of the gland is more than necessary to stimulate its secreting power, and amounts to irritation: the secretion of sugar is then greatly diminished. This is a fact in pathology to which I have more than once had occasion to call your attention.

It is to excitation proceeding unsuitably far that we must attribute the diminution of the saccharine secretion which takes place under the influence of fever, or of acute disease supervening in the course of saccharine diabetes. Under such circumstances, the glucosuria, as I have already mentioned, is sometimes temporarily suspended.

Finally, gentlemen, excessive secretion of sugar in the liver is, as the older physicians would have said, the "proximate" cause of saccharine diabetes. It is unnecessary to seek for an explanation of this in the chemical reactions which take place in the digestive canal and bloodvessels, which are very different from reactions under our control, such as can be produced in experimental glasses; but to which, nevertheless, an attempt has been made to liken the reactions of vital chemistry.

Physiological facts find their explanation in the pathology of the disease which we are now studying. The influence of local lesions of the nervous system, or of direct excitement of the liver, explains the pathogenesis of diabetes when symptomatic of the cerebral or hepatic affections of which I have spoken. In those cases upon which morbid anatomy throws no light —and such cases are numerous—there is reason to believe that the diabetes is dependent upon disturbance of the nervous system reacting upon the hepatic secreting function. Though the nature of this disturbance may escape our observation, it at all events manifests itself by a variety of symptoms, such as gastric disorder, disturbance of the sensory, motory, and intellectual powers, as well as of the senses and organs of generation. These facts have a direct bearing upon the *treatment* of saccharine diabetes.

Once more, gentlemen, let me impress upon you the importance of the fact, that diabetes must be considered both as a disease and as a symptom, just as albuminuria may be either the consequence of that kind of *nephritis* which is called *albuminous*, or be an epiphenomenon common to many diseases in which there is no lesion of the kidneys. For example, both albuminuria and glucosuria may supervene in diseases of the liver, pancreas, lungs, brain, spinal marrow, or great sympathetic nerve.

Observation has shown that in these cases there may be an organic lesion of the liver, brain, or lungs, while in others there is no appreciable material lesion; or that if any such lesion exist, it is only of a temporary nature, as is probably the case in epileptic fits and hysterical convulsions, as also when patients are under the influence of inhalations of ether or chloroform.

In all these cases, we must suppose that there is a modification of the hepatic or pulmonary hæmatosis, and that this modification is sufficient to determine an intermittent or continuous passage of sugar into the urinary secretion. Cerebral lesions, particularly of the medulla oblongata, disturb the regularity of the respiratory functions; and likewise, when there is direct lesion of the lungs, there is imperfect hæmatosis. MM. Reynoso and Michéa have laid great stress upon this local etiology of glucosuria, both in relation to acute and chronic lesions. The same view may be taken of lesions of the liver, the other organ of hæmatosis, and the active agent in glucogenesis. It is easy to see that an organic lesion of the liver may so modify the function of the organ, as to cause sugar to pass into the urine in greater or less quantity, according to the greater or less extent of the lesion, as is proved by the experiments of Claude Bernard and Schiff.

Glucosuria may also be produced by a modification of the circulatory apparatus of these organs, even when the modification is only temporary. Indeed, there is observed in fits of epilepsy and hysteria an asphyxial stage; that is to say, stasis of the blood in the lungs, in the right side of the heart, and probably also in the liver. This asphyxia would cause a diminution, or at least a modification of hæmatosis, which would account for the intermittent passage of glucose into the urine.

Moreover, there can be no doubt as to the part which the nervous system performs in the production of glucosuria. The physiological experiments upon the medulla oblongata, pneumogastric nerve, great sympathetic nerve, and spinal marrow, completely establish this fact: and every day we find physiological experiment confirmed by clinical experience.

It is unnecessary to expatiate at greater length on this point. I have already gone into such details that I do not think that I need upon this occasion re-enter upon the subject.

To conclude, let me remind you that glucosuria not unfrequently exists in women who are pregnant or giving suck. For this interesting discovery, we are indebted to MM. Blot* and Reveil. In these cases, the sugar is never produced in great quantity: in nursing women, however, there has been found, on chemical analysis, as much as ten or twelve grammes in a thousand grammes of urine. These facts have been confirmed by other observers: but I ought to add, that M. Leconte† has arrived at conclusions different from those of MM. Blot and Reveil. It is, therefore, still an open question, and one to which, upon a future occasion, I must direct your attention.

I have now come to the question of *treatment.*

In the class of cases of which I have just been speaking, and in all those in which glucosuria is accidental and temporary, medical intervention is almost superfluous, because the affection will cease spontaneously, and, as a rule, in a short time. We have, therefore, only to consider in a special manner the treatment of persistent diabetes.

Diet is of the utmost importance. Clinical observation has shown that feculent food increases, while an almost exclusively animal diet diminishes, the quantity of sugar which diabetic patients pass in their urine. This is not because when we keep patients on an animal diet, we deprive them of the alimentary substances which furnish saccharine matter in greatest abundance. We have seen that saccharine matter is produced when the diet is animal as well as when it is vegetable, though in less quantity: we have also seen that sugar taken as aliment is transformed in the liver into a special substance very different from diabetic sugar; and that the latter is exclusively a hepatic secretion. An animal diet suits diabetic patients better than a vegetable diet, because the latter, particularly when it is feculent, increases the excess of functional activity both of the liver and kidneys—it is because vegetable are much more diuretic than animal substances, as is shown by a much larger quantity of urine being passed by herbivorous than by carnivorous animals. The precept laid down by Rollo, and followed since his day, of giving diabetic patients a diet as nitrogenous as possible is in accordance with the teaching of physiology.

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* Blot: De la Glycosurie Physiologique des Femmes en Couches, des Nourices, et d'un certain nombre de Femmes Enceintes. Gazette Médicale, 1856, p. 720: and Comptes Rendus de l'Académie des Sciences, for 6th October, 1856.

† Leconte: Comptes Rendus de la Société de Biologie, for the year 1857, p. 60.

Nevertheless, gentlemen, it is necessary to guard against adopting extreme views, and believing that diabetes demands an exclusively animal regimen, involving rigorous abstinence from every other kind of food. In fact, you will meet with diabetic patients who pass very little glucose in their urine, whilst they adhere to a regimen consisting only of green vegetables containing a large quantity of chlorophylle, such as spinach, sorrel, cabbage, and cress; nay, even when they take acid fruits, such as currants, raspberries, and cherries.

In a disease in which disturbance of the nutritive functions plays unquestionably a leading part, it is of the utmost importance to avoid everything which might increase that disturbance; and consequently, it is necessary to vary the food so as not to induce loathing, a speedy effect of the exclusive use of the same kind of aliment.

Though a diet consisting chiefly of animal food is the most appropriate for diabetic patients, their regimen ought to contain a certain quantity of herbaceous vegetables, which are much more easily digested than feculent substances. I not only sanction, but I even recommend, the use of red fruits: failing them, I allow other fruits to be eaten, such as pears, apples, and even grapes, although they contain a large quantity of glucose.

Gentlemen, I cannot too emphatically raise my voice against the abuse of giving an exclusively animal diet in diabetes; and I must speak quite as strongly against the abuse of alkalies, which have been prescribed, particularly of late, as unquestionable specifics in glucosuria.

Though an exclusively animal diet immediately diminishes the thirst and excessive diuresis, it soon occasions intolerable loathing, and the health of the patients, which had seemed to be improving, is again deranged, and indeed becomes worse than it had been previously. On the other hand, however, if we rest satisfied with greatly diminishing the proportion of feculent nutriment, and allow the use of fruits and green vegetables, both appetite and strength are maintained; and although there may be a large quantity of glucose in the urine, there is hardly any derangement of health. I have had diabetic patients under treatment for ten years who could not be supposed to be passing glucose, unless, from time to time, the urine were chemically examined.

I have no objection to patients eating a small quantity of bread. This is a point in respect of which I take largely into account the taste of the individual, not, for example, interdicting its use to that numerous class of persons who cannot eat unless they are allowed some bread. I recommend bread made of the flour of rye or wheat, and not bread made of gluten, which has a disagreeable taste, in reality has no advantage, and is only prescribed in accordance with a chemical theory.

Pharmaceutical means are useful in assisting us to obtain a better regulation of the digestive functions.

Alkalies are unquestionably beneficial. During last century, this fact was recognized: lime-water was the remedy then prescribed for appeasing the burning thirst, and diminishing the urinary secretion of diabetic patients. At the present time, the alkaline remedies in use are of infinite variety, whether we administer such medicines as the carbonate of lime, the bicarbonate of soda, or magnesia in the form of powder, or give natural mineral waters such as those of Vichy and Pougues, which contain alkaline ingredients in greater or less quantity.*

The unquestionable usefulness of these remedies in the treatment of saccharine diabetes must not be allowed to lead us to the conclusion that they act as alkalies, that is to say, by producing within the economy the same reactions which we see them produce in our laboratory experiments.

You are aware that sugars of the class *glucoses*, in which is comprised the sugar of diabetes, are destroyed by the caustic alkalies, potash, soda, lime, &c., being changed into peculiar brown acids, with a rapidity greater in proportion to the greater concentration of the alkali, and its greater elevation of temperature.

It is on this fact that some chemists have founded their theory of glucosuria and saccharine diabetes. They say that the absence of sugar from the urine of a healthy man shows that the saccharine matter is derived from the aliment, and is destroyed by the blood which in its normal state is sufficiently alkaline to effect this transformation; and that therefore glucosuria is the result of the blood not being sufficiently alkaline to accomplish this change. I should not pause to recall to your recollection this chemical theory, victoriously assailed by Claude Bernard, had it not attained a widespread celebrity, had it not been received at first with a certain infatuation which is well remembered: for in fact, it has been refuted by the chemists themselves. First of all, it has been shown by Professor Poggiale, that to burn glucose in contact with alkalies a temperature of 95° C. is required; a fact which is of itself sufficient to overthrow the theory of which I have been speaking. In the second place, Professor Poggiale observed, that when glucose and an alkaline salt—the carbonate of soda or of potash, for instance—were injected simultaneously into the vessels, the quantity of glucose which the animal experimented upon passed in its water was exactly the same as when glucose was alone injected.

Here then, the chemical explanation is at fault, as is always the case when we endeavor to apply chemical laws to the phenomena of the chemistry of the living body. Still, however, the clinical fact remains, that alkalies are unquestionably useful in the treatment of saccharine diabetes. Their action has a powerfully alterative effect upon the digestive canal, and imparts regularity to its functions: alkalies do not act by curing the diabetes, but by replacing the patients in certain conditions in respect of nutrition, in virtue of which the abnormally excessive production of sugar does not take place. This statement may seem almost a paradox. Let me explain what I mean. Under the influence of alkalies, the same takes place in respect of diabetes which takes place in respect of gravel. These remedies do not act by producing alkalization of the urine, but by regulating the renal secretion.

Were we to accept the theory of alkalization of the blood in diabetes, it would be necessary to administer alkalies in the greatest possible quantity, and indefinitely to continue to employ them. This, gentlemen, would be an error most prejudicial to the patients.

I cannot too strongly impress upon you, that alkalies ought only to be given as adjuvants in the treatment, in moderate doses, and for not more than eight or ten consecutive days once a month.

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* See the Dictionnaire Général des Eaux Minérales et d'Hydrologie Médicale, vol. ii, p. 563, 965. Paris, 1860.

Other medicines may be associated with them. Tonic remedies may be given, such as rhubarb, for example, during eight days once a month, in doses of from fifteen to twenty-five centigrammes after each meal.

In the wards of my lamented colleague Legroux, you saw a glucosuric patient who was simultaneously treated by arsenical preparations and hydropathy. Under this treatment, the state of the man to whom I refer became greatly ameliorated.

Hydropathy is a very powerful medication in the treatment of diabetes, by its action on the great organs of the economy; as is indeed every means by which the assimilative functions are stimulated.

I am unwilling to terminate this lecture, already so long, and so full of details which perhaps you have considered superfluous, without saying two words on the immense influence of *exercise*. A diabetic patient who takes daily very active exercise on foot, may, without in any way changing his regimen, temporarily regain his lost health. I have known glucosuric individuals, who, during the hunting season, have ceased to drink and to urinate in excess, have regained strength and appetite, and, in spite of their fatigues, have recovered virile power lost at the onset of the disease. Exercise cannot be too strongly recommended to these patients: it may be said, that when there is combined with a suitable, but by no means very severe regimen, the daily exercise of which I have just spoken, diabetes, particularly in fat persons, constitutes an indisposition rather than a very severe malady.

By means of a well-devised hygienical system and regimen, aided by the judicious and prudent administration of medicines, we may hope to cure a few, and to relieve a great many, diabetic patients. I refer, of course, to patients who have not reached the last stage of the disease; for in that stage of wasting, the malady is beyond the resources of art.

LECTURE LXV.

POLYDIPSIA.

Cases.—Non-saccharine Diabetes may supervene in the Offspring of Polyuric, Glucosuric, and Albuminuric Parents.—Intercurrent Cerebral Affections may cause the Cessation of Glucosuria as well as of Albuminuria.

GENTLEMEN: Some days ago, a man left the Hôtel-Dieu, who had been several months in our clinical wards for polydipsia. He presented, perhaps, one of the most remarkable examples which are to be met with of this affection. Although not completely cured, his improvement was so great, that, of his own accord, he asked to be allowed to leave the hospital.

The treatment to which this patient was subjected consisted in his taking the extract of valerian in quantities rapidly increased to very large doses. That mode of treatment afforded me still more satisfactory results in a previous case of a similar kind.

Perhaps some of you may remember having seen the patient to whom refer in St. Agnes Ward. Like the man whose case is before us to-day, he was affected with polydipsia and polyuria. Every day, he drank thirty-two litres of tisane, and passed a proportionate quantity of urine. Professor Bouchardat, then principal apothecary at the Hôtel-Dieu, analyzed the urine at different times, without finding in it the least trace of glucose. It is a remarkable circumstance that the skin of this patient's face was frequently the seat of very intense erythema, unaccompanied by fever, and coincident with excessive thirst and excessive urinary secretion: after two or three days, this erythema disappeared, but in a short time reappeared. In other respects, the man's health was good. I prescribed extract of valerian, which was by successive augmentations increased to the enormous daily quantity of 30 grammes [457½ grains]. There was, under this treatment, a simultaneous diminution in the thirst and in the urinary secretion; and after four months of the treatment, the cure was complete. When I saw this man at a later period, his health was very good.

In a similar case, reported by Dr. Rayer, the success of the treatment was still more rapid.

The patient, a young lad, was consumed by an unquenchable thirst: he urinated in proportion to the quantity of fluid he drank. His urine was almost as light as water, inodorous, colorless, insipid, and exceedingly profuse in quantity. The little patient did not grow thin, ate well, and, with the exception I have now stated, enjoyed perfect health. The polydipsia and the polyuria, both simple in their nature, were apparently produced by a nervous affection, essentially different from diabetes, and, in fact, having nothing in common with it, except the profusion of the urinary secretion. The symptoms yielded, within three weeks or a month, to valerian, administered in the form of powder, which, practically, is the same as giving the extract. Several methods of treatment, particularly the treatment by opium, had entirely failed. Dr. Rayer has employed the same treatment with advantage in other cases.

Although the result has not quite realized my expectations in the case which has supplied the subject of the present lecture, it does not the less deserve to be taken into consideration by you.

In our patient, the disease had begun, according to his own account, four years previously: it was recognized under the following circumstances. The young man, then twenty, had been admitted to the surgical wards of my honorable friend and brother, Dr. Langier, for a symptom which was in itself insignificant. He was in the habit of sometimes complaining of pains in the lumbar region, particularly in the right side, pains having no relation to the surgical affection for which he came into hospital, and of which he was able to give a very imperfect account. It was evident, however, that he drank a great deal, and urinated proportionately. He was then drinking about six litres a day, and, according to his own expression, making as much water as any four men. This circumstance attracted the attention of M. Langier, who begged M. Bouchardat to examine the urine. It was found to contain sugar, but in small quantity. After some time, the patient was transferred to the clinical wards of Dr. Rostart, where he only remained five weeks. When he left the hospital, the quantity of drink which he took in the twenty-four hours varied between 18 and 20 litres, and the urine he passed amounted to 25 litres.

He was soon obliged to seek admission at La Charité. He was then passing daily as much as 32 litres of urine. In the wards in which he was placed, he was subjected to an almost total abstinence from drinks; and to quench his craving thirst, he was given some ice or lemon to suck, allowing him to take food in accordance with the promptings of his appetite. The unfortunate young man resigned himself to this severe regimen, which he endured for eight months, although he suffered so cruelly from it that one day he seized the chamber-pot, and drank the contents to the last drop! It may be said with truth, that under the influence of the regimen his state was greatly ameliorated, in this sense, that the urine he passed did not exceed 10 pounds in the course of the twenty-four hours. Nevertheless, as he felt his strength decreasing, his sight failing, and his body wasting, he asked to be allowed to leave the hospital.

He remained at home for a year; but at the end of that period, the pain in the kidneys having returned, the thirst having become very intense, and the quantity of urine very great, he was admitted to the Lariboisière Hospital. At that time, he was daily drinking 14 litres of fluid, and passing from 18 to 20 litres of urine. Some traces of sugar were detected in the urine. A plan of treatment was instituted, the basis of which consisted in administering preparations of iron, opium, and cinchona; and giving a diet consisting chiefly of animal food, from which feculent substances were excluded, and in which bread made of gluten was substituted for common bread. This treatment did not produce the slightest influence on the disease: it did not diminish the quantity of sugar (which was small) in the urine; and the amount of urine passed was greater than before. At the end of ten weeks, the patient left the hospital, in no degree benefited by the treatment; but in a few days, he was readmitted, when he went into the wards of my excellent friend, Dr. Pidoux.

Dr. Pidoux prescribed for him a diet consisting of five pounds of animal food, of which three pounds were ham, and two pounds roast meat: he was also ordered toasted bread, and a litre of wine in addition to the ordinary rations of convalescent patients. He was likewise put on bicarbonate of soda, and powder of valerian, of which latter he took 10 grammes daily from the first day.

There was soon a diminution in the quantity of urine: it fell from 29 to 11 or 12 litres in the twenty-four hours.

The extraordinary ease with which this man bore alcoholic drinks independently of his wine—he took 6 litres of a vinous tisane—induced Dr. Pidoux to try the effects of brandy. Within two hours, and at intervals of half an hour, he took a litre of what is known in commerce under the name of "*trois-six*:" he every day took the same quantity without appearing to be in the least degree inconvenienced by so doing. He stated that from the beginning he had acquired so great an immunity as to be able to drink large quantities of stimulants, without feeling the slightest symptoms of being drunk. On several occasions, he took for a wager twenty litres of wine, gaining his wager without producing any effect on the nervous system.

After remaining three months in Dr. Pidoux's wards, he felt able to resume his employment, one involving hard work and fatigue, as servant to a horse-dealer.

But once more, the amendment was of short duration: some months later, he returned to our wards.

I resumed the valerian treatment. I began at once by giving 10 grammes of the extract in the twenty-four hours; and I progressively increased the quantity till I reached 30 grammes. The result was a speedy diminution in the quantity of urine from 29 to 6 litres, the thirst proportionately abating. Unfortunately at last, the tolerance for the valerian was lost, and as soon as taken, it was vomited. The patient began to lose his appetite, and in place of eating four times as much as an ordinary man, which he had been doing, he was satisfied with "the four portions" of the hospital, which represent about 50 décagrammes [1 lb. 1 oz. and 10 drachms] of bread, 20 décagrammes [6 oz. and 10 drachms] of meat, and

50 centilitres [1 pint and 12 fld. oz.] of vegetables. Some convulsive phenomena having showed themselves, it became necessary to suspend the treatment. At the end of a fortnight, he was passing 16 litres of urine in the 24 hours.

At this period, the patient, by permission, spent some days at home: on the fourth day, he returned to the hospital, drinking 33 litres in the 24 hours, and passing from 37 to 43 litres in the same space of time. Not daring to resume the valerian, I tried belladonna in doses of one centigramme; and—strange to tell!—this man, who could drink 20 litres and a litre of alcohol of sp. gr. 0.835 [90 *degrés centésimaux*]* without being intoxicated, experienced violent effects from this minute dose of belladonna; and each time it was repeated, similar results were produced. I then had recourse to preparations of strychnia, medicines which render such great services in nervous diseases. The syrup of the sulphate of strychnia not being tolerated, I gave the tincture of nux vomica, which also I was obliged to discontinue, although in the first instance, under the influence of this medicine, the quantity of urine fell from 37 to 18 litres.

Then, after allowing the patient to rest for a time, I resumed the valerian, which I again caused to be administered to the extent of 10 grammes a day, as on the first occasion of my prescribing it; but I did not increase the dose to more than 12 grammes. In twenty-five days, there was a manifest improvement. The thirst had considerably diminished, and there was much less urine passed. When the man left our wards, he had for some time not been drinking more than four, three and latterly, two and a half litres a day, while his daily quantity of urine had gone down to five, four, and at last even to three litres and a half. This patient was completely impotent, like the generality of polydipsic and diabetic patients.

The history which I have now related presents some resemblance to the case of the patient of whom I spoke in my last lecture on saccharine diabetes, that patient at whose autopsy Dr. Luys found a lesion of the fourth ventricle. In neither of these patients was the polyuria entirely simple, for both had glucosuria at the beginning of the malady.

Now, there is no glucosuria in polydipsia properly so called, in that disease known by the names, *false diabetes*, *insipid diabetes*, *hydromania*, *polyuria*, and *urinæ profluxio*.

In polydipsia, the urine is clear like water, and never contains a trace of sugar: its density, in place of being greater than that of normal urine, in place of rising to 1.030 or even to 1.074, goes down to 1.009 and 1.001. The quantity of urine passed in the twenty-four hours is always much greater in polydipsia than in glucosuria; and, though in excess of the quantity of fluid taken by the individuals, it neverthless bears a relation to it. Thus, the patient in St. Agnes Ward, who between two of my visits was drinking six, eight, fifteen, and up to forty litres of fluid, passed within the same period, eight, ten, sixteen, thirty-seven, and even forty-three litres of urine in the twenty-four hours.

Another polydipsic young man, who died in our wards with purpura, had had sugar in his urine at the beginning of the malady, like the other patient of whom I have just spoken. There is, therefore, a relationship, which cannot be ignored, between glucosuria and polydipsia, a fact which is in harmony with the physiological experiments of Claude Bernard. The illustrious Professor of the Collége de France, by irritating certain parts of the floor of the fourth ventricle, produced sometimes albuminuria, sometimes saccharine diabetes, and at other times polyuria.* Is it not, therefore, very probable that a perturbation of the nervous system, the essential nature of which is still unknown, is the principal cause of these three maladies, which at a first glance seem so entirely distinct from one another?

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* "*Un litre d'alcool à 90 degrés centésimaux*;" rather more than a quart of alcohol at sp. gr. 0.835, or 57½ over proof, which is a trifle stronger than the rectified spirit of the British Pharmacopœia.—TRANSLATOR.

As I have just told you, polyuria, saccharine diabetes, and also sometimes albuminuria, may, in succession, attack the same individual: it is also not unusual to see suffering from non-saccharine diabetes, children whose predecessors were either glucosuric or albuminuric.

I was lately attending, in consultation with Dr. Bergeron, my excellent friend and colleague in the hospitals, a polyuric young lady, whose case I shall now relate to you in a summary manner.

When I saw her, she was nineteen years of age, and had all the appearance of perfectly good health. She was the granddaughter of a diabetic subject, who had had glucosuria for ten years, without any notable derangement of health. She had great obesity. I have already told you, when speaking of saccharine diabetes, that it is a more common disease among fat than thin persons, and that to compensate, as it were, for this peculiarity, it exercises a much less disastrous effect upon the constitution of the former. The glucosuria was rapidly diminished at the onset of the disease by the use of alkalies; but then it reappeared. It lasted ten years, as I have told you, and ceased suddenly and definitively upon the day on which he was seized with cerebral symptoms, due probably to cerebral hemorrhage followed by softening of the brain. In the eighteen months during which the symptoms continued, the patient became exceedingly thin.

Gentlemen, let me pause for an instant to call your attention to this fact, this strange coincidence. Does it astonish you to see lesions of the encephalon sometimes producing saccharine diabetes, as in the cases I quoted when lecturing on glucosuria, and in the experiments made on animals by wounding the fourth ventricle, while in other cases the malady ceases upon the production of cerebral lesions of another kind?

I shall afterwards have to call your attention to facts of this description in relation to albuminuria. You ought to remember a man, aged 57, who lay in bed 14 of St. Agnes Ward. He had Bright's disease, with general anasarca, and pulmonary infiltration. I did not believe that he could live a month. He was suddenly struck by hemiplegia; the albuminuria disappeared; the general health was restored. Some months later, when I sent the poor paralytic to the Bicêtre, there had been no return of the albuminuria since the occurrence of the cerebral hemorrhage. It is a remarkable fact, that during my long and busy medical career, I have only seen three recoveries from confirmed Bright's disease; and in all the three the albuminuria ceased, and the general health became re-established. This took place in one of the cases upon the patient becoming a decided epileptic: in the two other cases, recovery followed attacks of cerebral hemorrhage which had left the patients hemiplegic. This is an additional proof of the immense influence of the nervous element in the production of albuminuria, saccharine diabetes, and polydipsia.

Let us now return to the case of the young girl. Her grandfather, as I have said, was diabetic. One of her uncles died of Bright's disease. She herself had, in childhood, always been delicate, or *lymphatic*, to use the common expression. In May, 1856, when she was fourteen years of age, she showed symptoms of chlorosis; soon afterwards, ardent thirst supervened; the urine became aqueous, its specific gravity being hardly higher than that of distilled water; the general health was radically deteriorated. Under the influence of valerian and chalybeates her state improved, her strength returned, and the quantity of urine passed in the twenty-four hours fell from ten to six or seven litres. Soon the urine was down in quantity to three litres and a half; and up in specific gravity from 1.003 to 1.019. From the year 1856 to the year 1862, the polydipsia continued, increasing and diminishing, however, without its being always easy to assign a reason for the change. Mineral waters, sea-bathing, hydropathy, and valerian afforded temporary amelioration. The patient has become well-developed, tall, and plump; but the catamenia have not appeared, although different kinds of emmenagogue treatment have been perseveringly employed.

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* CLAUDE BERNARD: Leçons de Physiologie Expérimentale Appliquée à la Médecine.

Gentlemen, the only morbid phenomena of the first stage of polydipsia are excessive, sometimes inextinguishable, thirst, and the emission of an abnormally large quantity of urine. However—notwithstanding assertions to the contrary by the majority of authors, who on this point have only repeated the statement made by the first—along with the thirst, the appetite is not only usually augmented, but becomes exceedingly increased. You recollect the frightful quantity of aliment consumed in the twenty-four hours by our patient of St. Agnes Ward: you have heard of the terror he inspired in the keepers of those eating-houses where bread is allowed without extra charge to the extent of each customer's wishes. I have been told, that after he had taken one or two meals at one of these eating-houses, he was presented with money to prevent him coming back to dine.

With the exception of this ferocious appetite, and the burning thirst, the digestive functions do not seem to be at all disturbed. Digestion is accomplished with perfect regularity: the general health continues good. In such cases, polydipsia constitutes a very inconvenient infirmity rather than a disease.

In some cases, it is a fleeting symptom, but in others it is a malady which lasts as long as life. It sometimes makes its appearance in childhood, becomes more strongly developed at puberty, and then continues, resisting all curative measures; or, should treatment moderate the symptoms, or cause the affection to cease for a time, a complete cure is almost never obtained.

But do not suppose, gentlemen, that matters remain long in so favorable a position. Insurmountable anorexia, diarrhœa and wasting soon succeed the boulimia, the symptoms becoming more and more alarming: the skin acquires a withered clay-like appearance, the breath becomes fetid, and symptoms of tubercular phthisis show themselves just as in saccharine diabetes.

For a long time, gentlemen, adopting the opinion of my predecessors, I believed that polydipsia was a less serious disease than glucosuria; but that is a point in respect of which experience has greatly modified my views. Having seen in my private practice and hospital wards a great number of glucosuric patients retain good health for a long time, although I did not employ any very active treatment, I have, on the other hand, had the pain to see nearly all the polyuric patients whom I had to treat, waste away rapidly, and die much earlier than those who had saccharine diabetes. In the majority of glucosuric persons, let me add, that I can easily modify the quantity and character of the urinary secretion, while I find that I can be of little use to polydipsic patients. The young lady whom I saw with my honorable friend Dr. J. Bergeron (and of whom I have just been speaking), is a fresh proof that there are some fortunate and rare cases, in which polydipsia does not greatly disturb the general health, even while it resists, with disheartening obstinacy, treatment the most diversified and the most rational.

If sometimes this remarkable affection has as an evident starting-point, strong mental emotions—if it be a not unusual epiphenomenon of certain nervous affections, particularly of hysteria—still, generally speaking, its causes are utterly unknown.

The antispasmodic, or to use a more appropriate expression, the *valerian* treatment, is the best treatment for polydipsia. At least such is the conclusion to be drawn from the case of the patient who has just left the clinical wards—from a similar case which I treated in a similar manner six years ago—and from the facts reported by Dr. Rayer. Hydropathy has also seemed to have been of great use in some cases.

LECTURE LXVI.

CEREBRAL RHEUMATISM.

Cases of Cerebral Rheumatism occurring in a Drunkard and in a Woman who had been Insane.—The Cerebral Symptoms are generally due to Individual Predisposition.—Of Delirium in Diseases in general.—Six Forms of Cerebral Rheumatism: the Apoplectic, the Delirious, the Meningitic, the Hydrocephalic, the Convulsive, and the Choreic.—These Divisions are somewhat Artificial.—Description of these Forms.—Nature of Rheumatism.—Meningitis rare; Symptoms and Lesions of this Affection generally absent.—The Cerebral Phenomena are not the consequence of Metastasis, but are generally owing to some Morbid Cerebral Predisposition, such as previous habits of Drunkenness, or some former Neurosis.—They are not brought on by the administration of Sulphate of Quinine.—Treatment.

GENTLEMEN: You could see a few days ago at No. 16, in St. Agnes Ward, a remarkably robust man who was suffering from acute articular rheumatism.

When he was twelve years old, he for the first time fell ill of that complaint, which then affected his lower limbs chiefly, and lasted three months. Six years later, he had a second attack, when all his joints were involved, and for about three months again. At the age of twenty-one, he had a third attack, during which all his joints were successively affected, and which lasted four months. It would be difficult to meet with another case in which the rheumatic diathesis was more marked; and yet the patient declares that he has never had anything the matter with his heart, has never had palpitation, shortness of breath, or œdema. But you shall hear by and by that his heart was seriously affected, and that Dr. Bouillaud's law again proved true in this case.

Twelve days before admission, this patient felt vague pain in the small finger-joints, without any marked fever or malaise. Fever next set in, the left wrist swelled, and grew very painful, and the malaise became general. On the day of his admission, February 19, his fever was pretty sharp; his pulse was 118; his skin perspiring, the left wrist very much swollen, together with the synovial sheaths of the extensors, and of the long abductor of the thumb. The small joints of the carpus were painful. Both knee-joints, especially the left, seized since the previous day only, were painful also. The right knee, which had been affected for two days, contained a small amount of serous fluid.

I mention all these details in order that no doubt should exist in your mind as to the case being one of acute articular rheumatism, and that you may follow the migration of the symptoms.

A very rough systolic bellows-murmur and a soft diastolic one were also heard over the base of the heart, and could be traced into the large arteries.

From the frequency of the pulse, the heat of skin, the intense thirst, and the general aspect of the patient, I prognosticated a severe attack of rheumatism, of prolonged duration. I prescribed a scruple of sulphate of quinine, and in the evening my clinical assistant cupped the precordial region in six different places.

On the following days the pulse remained as frequent, but the state of the joints changed; on the 21st, all swelling of the left wrist had disappeared, but there was still redness of the synovial sheaths of the wrist and the hand; on the 22d, the tibio-tarsal articulation and the right foot were painful, the left hand and wrist were free, but the right hand was affected; on the 24th, the lower limbs were free, but the right hand was still red and swollen; the elbows were free, while the shoulders were painful.

The patient felt much better, and hoped to be soon able to eat. For the previous two days, the dose of quinine had been increased to two scruples; on the 22d the patient took thirty grains of quinine.

On going round on the evening of the 24th, my clinical assistant observed no unusual symptom; the pain in the joints had diminished, and the patient was very much pleased with his condition. An hour later, however, he complained of not being able to see, and shortly afterwards he began to vociferate, called out "Thief!" rushed out of bed, and fell down. On being put back to bed by two attendants, he struggled with them, exhibiting considerable strength, and then, dropping back, died. All this took place in less than a quarter of an hour.

When the body was examined, there was found pretty marked injection of the whole of the pia mater covering the brain, but the meninges were nowhere thickened, and nowhere adherent to the gray matter of the brain. There was not a trace of effusion into the subarachnoid space. The choroid plexuses were not appreciably redder than they normally are. There was no intra-ventricular effusion. The brain was remarkably healthy, and thin sections of it showed that it was not more vascular in one point than in another; there were not even the interstitial puncta which are sometimes observed when there is meningeal injection. The corpus callosum, thalami optici, corpora striata, cerebellum, and medulla oblongata were of firm consistency, and exhibited no alteration whatever. In short, it would have been difficult to see a more normal brain in aspect and texture.

The basilar and cerebral arteries were perfectly healthy, with unaltered walls; no coagulum could be seen which could be referred to thrombosis or embolism.

As to the cardiac lesions, I need not dwell on them, but will merely state that the bruits heard during life depended, as had been diagnosed, on a double lesion of the ventriculo-aortic orifice, constriction of the aperture, and incompetency of its valves. Contrary to the patient's assertion, however, these lesions were of very old date, although they had not given rise to functional disturbances. There was intimate adherence of the whole of the pericardium to the heart. The heart was very large, especially on the left; its cavities were empty. There was congestion of the lungs, almost amounting to that found in asphyxia. The kidneys were large, and of a violet hue. The liver was red, and of a very large size. There was nothing worth noticing in other organs.

There was no trace of effusion in either knee; the synovial membrane was not in the least injected, except to a very slight degree in the outer cul-de-sac of the left knee. There was no redness of the synovial membrane, nor any effusion into the wrist-joints, or the other articulations which were affected on the preceding day.

I do not think that any one of these details is superfluous. The present subject is a controverted one, and raises many questions of doctrine; for it may be asked whether, in cerebral rhumatism, there be metastasis from the joints to the brain, mere functional disturbance of the brain without any lesion, or rheumatic meningitis? You may now imagine what interest attaches to the pathological appearances found in such instances.

The subject of this case, then, had previously suffered from three attacks of acute articular rheumatism, which had each lasted from three to four months, and had left persistent marks on the pericardium and the heart. The fourth and last attack was only eight days old, when the state of the articulations and the general condition suddenly improved, while soon afterwards cerebral symptoms developed themselves, beginning with slight disorder of vision, and ending in delirium of only a quarter of an hour's duration, followed by sudden death.

On examination of the body, slight injection of the meninges is alone found to account for the brain-symptoms. The case was certainly one of acute articular rheumatism, and the symptoms which preceded death were unquestionably those of cerebral rheumatism. But they had made their appearance so suddenly, and so taken us by surprise, that, after my attention had been thus aroused, I determined on investigating the case further. I then got the information that the patient had been a hard drinker, or, in other words, a drunkard. He could drink largely without getting drunk, but his companions said that he was *stupefied* by drink. For three months previously he often had at night attacks of dyspnœa and nightmares. His previous history, therefore, told a sad tale of drunken habits, and his brain was in an unfavorable condition when rheumatism attacked him for the fourth time. We shall see, presently, what conclusions can be drawn from this respecting the etiology of cerebral rheumatism. In the meantime allow me to call your attention most particularly to the fact that this patient was a hard drinker, spending his days in a chronic state of inebriation, and that for some time past he was subject to nightmares, and lastly, that he complained of mistiness of vision a few minutes before his fatal seizure.

I pass on to another case, that of a woman aged sixty-three, who is lying in bed No. 2. She is a charwoman and porter, so that she may be a little addicted to spirit drinking. She relates her story as follows: On the Sunday previous to her admission into hospital, she had, after doing her work, gone to Notre-Dame to mass, but she could not follow the service as usual, and did not understand it, and at the same time she felt an acute pain in her right shoulder. After church she went to a house to do some work as charwoman, although she felt queer. She did part of her work in a mechanical manner, and then sat down in a stupid state in a dark corner of the kitchen, where she remained silent and motionless. Her employer kindly sent her home in a cab. On her way there, she complained of acute pain in her right shoulder, and was conscious of a singular undefined change which had come over her mind. She got into bed, slept well, and woke the next morning speechless. She wanted to drink, but could not make her husband understand her wish; she pointed to the water-bottle with her left hand, but she could not even make those elementary gestures which we would use if we wanted to ask for drink from a person who did not understand our language. She evinced impatience, as aphasic patients generally do, at her inability to speak and gesticulate, and at others being unable to understand her meaning. She had then in her mind a distinct wish to show her anger by calling her husband a bad name, but although she had a definite idea of this in her own mind, she could not speak out. During the whole of Monday and part of Tuesday, she had not the least power to articulate a single word or to make an intelligent gesture. A medical man was called in, who ordered a few leeches and a purgative. In the course of Tuesday evening she began to speak, but in a sputtering manner, and when she was admitted into the hospital on Wednesday, she spoke in the same sputtering way, although she could pretty distinctly relate how her complaint began. She complained on that day of violent pain in the right shoulder and the left knee.

As her mental condition interested me very much, I pressed her with questions, whether she had suffered from any nervous complaint at some previous time, and she at last told me that she had been rather *nervous* lately, especially since the revolution of 1848. Rents were with great difficulty collected from small lodgers, and one of these even threatened to shoot her husband, on the latter claiming the rent. She was so frightened that she became quite mad with fear, and that she had to be taken to the Salpêtrière three days afterwards, and remained in that hospital for thirteen months in a state of fierce mania. Now, I maintain that the brain of this woman must have been in an abnormal condition to account for her becoming insane after a violent altercation, and especially for her remaining so long in a state of furious mania.

At this present moment the patient is feverish, her tongue is white, and she feels sick whenever she tries to sit up in bed. Her right shoulder and elbow and her left knee are painful, but they are neither red nor swollen.

There is nothing wrong about the heart or lungs. The most remarkable circumstance about her cerebral condition is an irresistible tendency to doze, exactly as if she had been struck with apoplexy. She begins a sentence well, but by degrees speaks less and less intelligibly and rapidly, then stops and drops off to sleep. When she is shaken sharply, she wakes up, looks around with wondering eyes, answers questions clearly, but soon dozes off again.

Thus, gentlemen, the subject of my first case was a hard drinker, who suffered habitually from nervous symptoms traceable to a morbid cerebral condition, while the patient in my second case is an extremely nervous and somewhat insane woman. There was, therefore, in both these cases, a predisposition to the manifestation of cerebral symptoms in the course of rheumatic fever. But before I proceed any further, I wish to make a digression which will enable me to bring before you my views on the various modes of manifestation of delirium, and on the variable significance of this psychical disorder.

You know already from experience that some individuals rave for the least thing, and in order to illustrate how far this predisposition to delirium may be pushed, I will relate to you the following case. A few years ago, while making a post-mortem examination at the Hôtel-Dieu, both my clinical assistant and I pricked ourselves. My assistant, whose mother was of a nervous temperament, and who had himself been a sleep-walker as a boy, became affected with boils and with very serious nervous symptoms, occasionally even with fearful delirium. Five or six days after I had wounded myself, I had at the site of the wound a carbuncle which gave me great pain, but brought on no fever, and I then had a succession of boils, unattended with fever or delirium. Thus, one and the same cause, namely a prick inflicted under the same conditions, gave rise to exactly similar anatomical lesions, but to general reaction, which was perfectly different in the two cases, and very manifestly because of the difference in the amount of vital resistance in the two subjects. This is the condition to which the ancients have given the oft-derided name of idiosyncrasy. Thus, again, how many individuals do we meet with who are delirious when they have the least fever, and how many children are seized with convulsions when they get at all feverish.

Febrile delirium generally comes on at the *onset* of diseases. Both delirium and convulsions are then correlative phenomena of the rigor; but it must be added that there are certain acquired predispositions. Thus, Dupuytren called attention to the nervous delirium following upon injuries, and justly compared it to *delirium tremens*. Now, this delirium occurs in individuals addicted to spirituous liquors, without there being any relation between the lesion and the intellectual disturbance, for it comes on after any kind of injury as well as after the most skilfully performed surgical operation. You are aware, also, how frequently delirium complicates pneumonia when occurring in drunkards. In the former case the injury, in the latter the inflammation are the determining cause which brings on, with the aid of the existing predisposition, the mental disturbance which is the dreaded prelude of a fatal termination.

Hereditary influence plays also a most important part in the production of nervous disturbances; and hence it is we frequently find that women whose mothers were insane or of a nervous temperament are seized with eclampsia during labor.

As to the prognostic value of delirium, it chiefly depends on the nature and intensity of the disease in the course of which it shows itself. Take, for instance, typhoid fever and cholera, on the one hand, and scarlatina and measles on the other.

Stupor and delirium are, as it were, normal symptoms of typhoid fever, which always presents nervous phenomena, such as sleeplessness, stupor, vertigo, weakness, and delirium. These symptoms appear very simple to you then, because they form part and parcel of the complaint; and you take them into account merely in order to confirm your diagnosis. But if they show themselves in the course of a disease of which they are not usual accompaniments—in articular rheumatism or pneumonia, for instance—they at once excite anxiety and alarm.

See what happens in cholera, in the most virulent form of the complaint even. The intellect is unimpaired; and although the patient may shriek from the pain of the cramps, he gives rational answers to questions that are put to him. The brain, therefore, is not involved until the so-called typhoid stage of the disease sets in. Take, again, a case of acute peritonitis, when the whole of the abdominal cavity is involved, and the inflammation spreads by contiguity to the intestinal walls themselves; no delirium, no nervous symptoms will show themselves. Such phenomena, therefore, do not depend on the seat of the disease, but on its nature. I must add that, while speaking of cholera and typhoid fever together, as abdominal diseases, for the sake of illustration, I followed the usual custom, although it is a bad one.

Let us now examine the relation of delirium to diseases of the skin, and to eruptive fevers, which are no more cutaneous affections than cholera and typhoid fever are abdominal diseases.

In scarlatina, delirium is, as it were, the rule, as it is in typhoid fever; but not so with measles. If, therefore, delirium should come on about the fifth or sixth day of an attack of measles, it should excite alarm, because this nervous symptom does not form part of the natural evolution of the disease. In some cases the delirium is proportionate to the intensity of the disease, although nervous symptoms do not constitute part of the complaint. Thus, discrete variola is not attended with delirium, while confluent variola is nearly always so. Delirium also occurs in erysipelas of the face, when the face and scalp are simultaneously involved.

The usual course of a complaint should therefore be carefully considered before a prognosis is established. If nervous symptoms be proper to the disease, they should give rise to no great anxiety; but if they be of unusual occurrence, they should be taken into serious account. Thus, delirium occurring in a case of lead poisoning shows that the brain is affected, and compels a modified prognosis; while eclampsia supervening on albuminuria points to uræmic poisoning, and, therefore, to a grave affection.

Let us now examine the relation which exists between nervous symptoms and rheumatism.

Articular rheumatism has no great tendency to develop cerebral manifestations: bear this well in mind. However intense the fever and the pain may be, this complaint does not usually give rise to ataxic phenomena, to delirium, or to somnolence; the intellect is unimpaired. And yet some cases do occur in which the rheumatism is complicated with brain-symptoms, occurring independently of the intensity of the disease, of its gravity, as well as of its extent.

Recall to mind the two cases which you saw in my wards, and which suggested this lecture. The woman lying at No. 2 was suffering from an extremely mild attack of rheumatism, which had scarcely given rise to any marked degree of fever. The intensity of the disease had, therefore, nothing to do with the development of brain-symptoms; and yet, from the second day of the attack, symptoms somewhat apoplectiform in character began to show themselves, giving rise at first to aphasia of forty-eight hours' duration. The man at No. 16 was suffering from articular rheumatism, which was well marked, but not excessively so; the joints that were simultaneously affected were not many, the fever was moderate, and yet formidable brain-symptoms supervened, which carried him off in less than an hour. But this patient was a hard drinker, habitually stupefied by excesses in drink; while the woman I spoke of just now was of an exceedingly nervous temperament, and had been insane. Bear these facts well in mind, because they throw, in my opinion, the greatest light on the ætiology of cerebral rheumatism.

Be careful also not to confound such cases with others in which brain symptoms occur constantly, as the consequence of a true typhoid state, such as the purulent arthritis of pyæmia, or of puerperal fever. You may recollect a man who was lately at No. 2, in St. Agnes Ward, and on the front of whose chest a heavy bag had fallen, while he was engaged in unloading a cart. He complained of an acute pain in the right flank on the day of his admission, where deep fluctuation could be felt. Two days afterwards his knees swelled, and next his wrists and shoulders. Tremor of the lips, carphology, and delirium then came on, so that I diagnosed pyæmia and secondary arthritis. On examining the body after death, I found an enormous collection of pus inside the chest, and inflammation of the joints which had been attacked. This latter condition had given to the case a semblance of rheumatism, but it was not an example of ordinary acute articular rheumatism. I could not repeat it too often that in this latter complaint brain-symptoms are the exception, while they are the rule in infectious or purulent diseases.

Before I describe to you the symptoms and the forms of cerebral rheumatism, I will first tell you of the premonitory symptoms which may precede it. In some instances there are none, and they were certainly absent in the case of the woman at No. 2; while in that of the man at No. 16, they consisted in a slight disturbance of vision, which lasted a few minutes. This was a premonitory symptom, however short its duration may have been; in some instances, it has preceded by a day or two the occurrence of brain-symptoms.

It has been also said that an exaggerated anxiety shown by the patient points to a mental condition which should put us on our guard, because acute articular rheumatism does not, in general, alarm the patient. Hallucinations and stupor are symptoms, therefore, which indicate the possible supervention of some cerebral complication.

Dr. Vigla and other observers have also mentioned excessively copious perspiration, and the presence of a miliary eruption as premonitory symptoms. But profuse perspiration is of usual occurrence in rheumatic fever, and this does not indicate that brain-symptoms are imminent. And as the miliary eruption is only the result of the perspiration, it cannot be regarded as a premonitory symptom.

Now, what are the forms of cerebral rheumatism? Six forms have been admitted: 1st, the *apoplectic;* 2d, the *delirious;* 3d, the *meningitic;* 4th, the *hydrocephalic*, described by Dr. Marrotte; 5th, the *convulsive;* and 6th and lastly, the *choreic*, of which I have related instances to you. All these forms, in my opinion, are mere modifications of the cerebral condition, and are only required for the sake of description. They are in reality an expression of the same cause, and of the same anatomical lesion, if there be one, and they no more deserve to be regarded as distinct species than the delirious or convulsive form of typhoid fever or of scarlatina.

I will now review successively these various forms, and I will begin with the *apoplectic*. Older authors have admitted it already, and it is mentioned by Storck, Musgrave, and Sauvages. But to what confusion did not the term *apoplexy* give rise in those days? All cases of sudden death were ascribed to it. So that we are justified in believing that the term apoplectic cerebral rheumatism was applied to complications which did not always involve the brain.

Nor do I admit, like Musgrave and Sauvages, that hemiplegia occurring in an individual laboring under gout or rheumatism is on that account due in every case to apoplexy. Yet cases do occur in which, while the rheumatic attack goes on, transient hemiplegia shows itself which one cannot but refer to the rheumatism. I have, on a former occasion, related to you the history of a young girl who was admitted into the hospital, suffering from intense fever, excessive rachialgia, like that announcing variola, and from paraplegia. For three days, I looked out for the eruption of variola; on the fourth day, I ordered her to be cupped, when all symptoms of paraplegia disappeared, but amaurosis and hemiplegia immediately set in. I then called the case an instance of *rheumatic hemiplegia* or of *cerebral rheumatism.* A few leeches were applied behind the ears, and two days afterwards pain was complained of in some of the joints, upon which the amaurosis and hemiplegia disappeared. For such cases of transient hemiplegia, which, from the manner in which the paralysis alternates with other symptoms, is so manifestly due to the rheumatic diathesis, I admit the denomination of *rheumatic apoplexy*.

Hemiplegia may also arise from cerebral embolism in the course of rheumatism. One of the vegetations formed on the cardiac valves, as an event of endocarditis, is suddenly detached, and by blocking up a cerebral artery gives rise to sudden asphyxia of the brain by cutting off its supply of blood. If, now, collateral circulation makes up for the obstruction, the hemiplegia disappears, and the case is one of rheumatic apoplexy in the narrow sense of the word; but rheumatism is only indirectly at fault here, and the case is very different from the one in which rheumatism attacks directly the brain or its membranes, as it does the joints. I am, therefore, inclined to admit two varieties of rheumatic apoplexy, one dependent on congestion, and the other on embolism.

The rheumatic apoplexy of older authors, terminating in sudden death, remains still to be explained. In the majority of cases, the cause of death is not then to be found in the brain, but in the pericardium, the heart, or the large bloodvessels. Thus pericarditis, when the effusion is rapid and considerable, may cause death by suddenly arresting the heart's action. Thus also, acute endocarditis may give rise to the same accidents, through organic or dynamic obstruction to the heart's action. Thus, lastly, a coagulum may form in the cardiac veins, in the right heart or the pulmonary artery, as a result of that remarkable tendency to spontaneous coagulation which the blood evinces in rheumatism, and this thrombosis will bring on asphyxia, which will carry off the patient rapidly, if not suddenly, in a state of stupor which may be erroneously regarded as cerebral.

Facts, ascertained within recent years, allow us, therefore, considerably to restrict the number of cases of rheumatic apoplexy; and we can scarcely say that the patient at No. 16 died from this cause. He complained, it is true, of mistiness of vision, and for a quarter of an hour, he was violently delirious; but I do not see that there was in his case the *apoplectic stroke proper*.

In other instances, the symptoms are, to a certain degree, those of apoplexy, as, for example, in the case of the woman lying at No. 2. You remember how she felt irresistibly drowsy, and how she sat down in a dark corner of the kitchen, and how afterwards she dropped off to sleep while talking to me; you may recollect also that for forty-eight hours she suffered from aphasia. Such a case, with sudden well-marked cerebral symptoms, simulating those of hemorrhage or congestion, is, I believe, an instance of apoplectic cerebral rheumatism. I will only mention, incidentally, *apropos* of rheumatic apoplexy, cases of profound stupor following on delirium in the course of an attack of acute articular rheumatism; or, again, those instances in which eclampsia comes on suddenly, and is succeeded by the usual stupor. But I will dwell more on cases like that of the young girl who had spinal pain and paraplegia, hemiplegia, and amaurosis, and at last pains in the joints. It is clear that in her case the rheumatism, whatever be the notion formed of its nature, attacked successively the spinal cord, the brain, and the articulations, probably affecting, in each region, similar anatomical tissues, but giving rise at each spot to very different symptoms. The lesions were too transient, however, to admit of their being ascribed to apoplexy, in the sense of Musgrave and Sauvages.

In one of our hospital nurses here, Seraphine, we have had another instance of these rapid fluctuations of functional disturbances. She has not menstruated for some time past, as she has now reached the period of life in which that function generally ceases. Three years ago her left wrist-joint was attacked with rheumatism, which caused marked swelling of the joint, and redness of the skin. She was then suddenly seized with vertigo and a sensation of weight in the head, and her limbs became so *paralyzed* that she was unable to work. The pain in the head and nucha diminished, and her upper limbs regained some power, but the lower ones were still extremely weak, while she had a fixed and acute pain in the lower part of the spine. She was chiefly treated by veratria, and afterwards by spirits of turpentine. Her complaint resisted treatment for a long time; on several occasions she vainly tried her strength, and it was only after the lapse of *fifteen months* that she was enabled to resume work. She has enjoyed pretty good health since then, although her lower limbs have felt rather weak, and she has occasionally suffered from headache and numbness of the limbs.

During the summer of 1859, her finger-joints were painful and swollen; and her right foot, which was also swollen at the time, continued so until fresh symptoms showed themselves.

On January 11, 1860, during the night, she complained of violent cephalalgia and of pain in the right shoulder; the headache diminished, but was succeeded by acute pain in the lower part of the spine, and numbness of the lower limbs; the swelling of the foot had disappeared. Eight days afterwards, the same symptoms returned. She complained of violent pains in the head and of mistiness of vision, of loss of power and numbness of the arms, chiefly the right; the pains next settled in the back, and the lower extremities became chiefly paralyzed.

From January 20 to January 31, she was cupped along the spine on three separate occasions, and took turpentine capsules. Her symptoms improved markedly under the influence of this treatment; she had occasional headache, but it was never so violent as in the beginning; the pains in the back were also less intense, but the legs still continued to be very weak.

On February 7, the lower limbs were stronger, and the pain in the back less; the hands, on the contrary, were more painful, while pain was complained of in the back of the neck and in the head.

On the 8th, the legs were less numb, and felt stronger; but the hands, especially the right one, were numb. Eight turpentine capsules were ordered.

On the 10th, the patient felt stronger, and the numbness of the limbs was less.

On the 16th, the improvement was more marked, and the patient could darn. The turpentine was repeated.

On the 23d, there was no pain at all down the spine, but slight pain was felt in the joints.

On March 1, the patient resumed work.

On the 8th, at twelve o'clock in the day, she was seized with violent rigors, and, after a few twinges in the arms, with a very acute cephalalgia, accompanied by a distressing sensation of intracranial pulsation. Some pain was also felt along the spine.

On the 9th, the headache had diminished, but the pain down the spine was worse. I prescribed two pills, containing one-fifth of a grain of veratria each.

The patient complained of weakness still on the following days. The same treatment was persevered in.

On the 29th, the right leg was still weak, while for the first time the left elbow became painful. The veratria was repeated.

On the 30th, pain in the elbow less.

On April 1, right foot swollen; left foot sometimes swells also, but less frequently. Veratria pills again repeated.

On the 16th, the patient felt well enough to begin work again. For a few days previously, her menses had returned. All swelling of the feet had disappeared.

In this case, gentlemen, you see rheumatic arthritis precede for a few years the symptoms of paralysis, and thus account for their production. Could any doubt remain, an analysis of the phenomena would remove it at once, for the symptoms of paralysis and those of articular rheumatism proper will be seen to alternate in the most significant manner. At one time cephalalgia and sensorial disturbances are present; at another, spinal pain and weakness of the lower limbs; sometimes, again, the cerebral and spinal symptoms are replaced by painful swelling of the joints.

This woman, then, had alternately cerebral and spinal rheumatism; and the cerebral symptoms which she exhibited were of an apoplectiform character.

There is just now in one of my wards a poor woman of the name of Marie, whose very stout appearance would never lead one to suspect that she may labor under a nervous affection. And indeed the symptoms which she presents are not due to hysteria, but are evidently referable to the rheumatic diathesis. She first had an attack of acute articular rheumatism which seized on the wrists, and then attacked the head, producing stupor for a day or two. The spinal cord was next attacked, and paraplegia followed. For the space of four months, this poor woman thus presented mobile symptoms, suddenly shifting from one organ to another, from the brain to the spinal cord, and from the cord to some part of the limbs.

The other day you saw that she was utterly unable to walk, she felt giddy and stumbled; she had a vacant look, and her tongue was furred; she had great difficulty in collecting her thoughts and in expressing them in words; she looked as if she were drunk. To-day she has had mydriasis and imperfect vision, which set in suddenly. She occasionally suffers from exquisitely painful neuralgias, and I mentioned her case when on the subject of neuralgias. Her symptoms are sufficiently well marked, and their relation to rheumatism sufficiently clear to justify one in giving to them the name of *rheumatic apoplexy* of the brain and spinal cord.

This, in my opinion, is the apoplectic form of cerebral rheumatism, and it should not be ascribed, as Musgrave and Sauvages do, to the occurrence of effusion in the nervous centres.

I now pass on to the *delirious* form, which is more frequent than the apoplectic. The delirium has nothing peculiar about it in the majority of cases, and resembles that which occurs in other diseases, such as typhoid fever or variola, with this difference, however, that it generally terminates in death. This form usually runs an acute course. It lasts one, two, or three days, and is succeeded by stupor, the patient dying comatose, that is, with apoplectiform symptoms. In some cases, the delirium runs a slow course, and becomes truly chronic.

In 1861, I had under my care, at No. 7, in the male ward, a young man suffering from acute articular rheumatism, who for a whole month was delirious. Sometimes the delirium resembles puerperal mania, that form which lasts from eight to fifteen, thirty days and more, and either disappears spontaneously, or after the administration of a purgative or of bark. The interesting case recorded by my colleague, Dr. Mesnet, is an instance of this.* The subject of it was a young man, twenty-three years of age, who had just experienced heavy money losses, and who had been guilty of various excesses, and was therefore depressed in mind and weakened in body. He first complained of some vague pains in his joints, and then exhibited symptoms of pleurisy, which remained stationary; after this fresh pains set up in some large joints, the knees, the arms, and afterwards the ankles. These pains, which had come on suddenly, prevented all motion, and were accompanied by diffuse redness round the joints, without intra-articular effusion. No doubt existed as to the rheumatic nature of these symptoms. When the knees and shoulders became involved, the patient's mind got, as it were, benumbed; he lay in a state of hebetude, answered slowly questions put to him, had a difficulty in finding words and in collecting his ideas, and showed indifference to everything. A few days later, the relation between his cerebral condition and his articular pains became manifest; when the pains disappeared, the patient's intellect was more confused and acted more slowly; when they returned, he talked more. His prostration was soon succeeded by agitation, by violence, hallucinations of sight and hearing, illusions, delirious fancies; he believed himself to be the object of suspicions, of pursuit, and the victim of machinations, &c. A few days afterwards, in addition to his delirium, his movements became choreiform, he kept constantly flexing and extending his fingers, and could not raise his hand to his lips; he spoke in a curt interrupted manner, and swallowed with rapidity and with a kind of convulsion. The delirium, which had at first come on in paroxysms, became continuous as soon as the symptoms of chorea showed themselves. The patient, under the influence of hallucinations, was constantly trying to get up in order to avoid the evil-disposed persons by whom he fancied himself to be surrounded, or in order to run away from the importunate voices which annoyed him. Sulphate of quinine in gradually increasing doses was prescribed, and a remarkable improvement followed. The choreic movements, the agitation, the hallucinations, the delirious fancies, ceased, but the intellectual confusion continued for another fortnight. After that it disappeared of itself by degrees; health and strength returned, and a complete cure was at last obtained after two months and a half.

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* Archives Générales de Médecine, June, 1856.

I will return presently to this coincidence of rheumatism and chorea, but I have related this case in order to show you that the delirious of cerebral rheumatism may present two very distinct varieties: 1st, an acute form, which is grave and ends fatally; 2d, a chronic form, which is much less fearful. A third variety might be admitted, namely, that which is brought on by purulent arthritis; but there is not then cerebral rheumatism properly speaking. The delirium is a result of the suppuration, and is analogous to that which comes on in pyæmia or in diseases of low type. It begins with a slight disturbance of the intellect, and after a time the delirium becomes more marked and more continuous; there are muttering and carphology. Such a condition differs widely from that which characterizes cerebral rheumatism.

I now come to the *meningitic* form. The name given to this variety is as bad as that of delirious or apoplectic, as I will presently show; but let us first inquire what its symptoms are. I need not remind you that the invasion of ordinary meningitis is generally announced by vomiting, pain in the head, which is sometimes fearful, by constipation, and in children by convulsions. Now, these symptoms never occur in the so-called meningitic form of cerebral rheumatism. Thus, vomiting is generally absent, and there is only delirium, which is remarkable on account of the suddenness with which it sets in, and which rapidly passes on to stupor. Such is the course run by the disease even when, after death, dissection shows the lesions which characterize meningitis. This form, then, as far as the symptoms are concerned, does not differ from the delirious, from which it cannot be distinguished during life. It is consequently an anatomical, not a clinical, form of the disease. In some rare instances, however, as in a case recorded by Dr. Marrotte, considerable effusion may take place rapidly, and symptoms may then show themselves indicative of compression of the brain, such as hebetude, dilatation of the pupils, and coma. The case is, then, one of true acute hydrocephalus.

I next pass on to the *choreic* form, which has not been sufficiently described by authors, but which deserves a special place by the side of the preceding varieties. I have already told you, when I spoke of chorea, that Dr. G. Sée* was the first to ascribe this complaint to a rheumatic diathesis acting on the brain or the spinal cord. This view is based on authentic cases, but it should still be accepted with a certain amount of reserve, for, like all innovators, Dr. G. Sée has exaggerated the importance of these cases, and of the inferences which might be drawn from them; his exaggeration served to awaken more the attention of the profession.

Dr. Sée asserts that a child who has had one or several attacks of acute articular rheumatism will sooner or later have St. Vitus's dance. You know that rheumatism pretty frequently shows itself during the convalescence of scarlatina; now, chorea pretty often comes on after this rheumatism. Conversely, a child who has had one or several attacks of chorea will sooner or later have rheumatism. It evens happens in some rare instances to find chorea come on during an attack of acute articular rheumatism. Recall to mind a case which I related to you in my conference on chorea, that of a girl who was caught round the body by a man on a dark staircase, and became shortly afterwards affected with unilateral chorea. This was soon replaced by acute articular rheumatism, and when the rheumatism got well, the chorea returned. But this is not all: experience has shown that chronic endocarditis is pretty frequent in choreic children, and that pericarditis is equally so in children and adults who are subject to St. Vitus's dance. Now, as you are aware, endocarditis and pericarditis are both brought on by rheumatism.

Thus the relations of articular rheumatism to St. Vitus's dance are proved directly and indirectly.

Lastly, cases of acute articular rheumatism do occur in the course of which chorea manifests itself. The relation between the two affections cannot then be doubted, and I may remind you of a case in illustration of this, which I have already related to you, that of a young girl, the daughter of a tailor in the Rue Richelieu, whom I saw in consultation with Dr. Legroux. She had been suffering from acute articular rheumatism for the last ten days, and for two days previous to my visit, violent chorea had set in, with delirium, inability to eat or drink, and constant and violent vomiting. She died from the violence of the chorea. We can, therefore, infer from such cases that acute articular rheumatism is sometimes transformed into St. Vitus's dance, that is, into a cerebral affection, which is sometimes grave and sometimes mild. A choreic form of cerebral rheumatism should, therefore, be admitted in my opinion.

I now pass on to an important question, namely, what is the *nature* of cerebral rheumatism?

When one thinks of the facility with which rheumatism generally attacks and brings on inflammation of serous membranes, the first thought which occurs to the mind is that cerebral rheumatism is merely meningitis. We very frequently see rheumatism pass from the joints to the pericardium, or to the pleura, and less frequently to the peritoneum. Now, if one considers that the arachnoid membrane is identical with the pericardium and the pleura, there is no reason for refusing to admit that it may, like them, be affected in articular rheumatism. Hence, when great cerebral disturbances occur in the course of a rheumatic attack, one is tempted to say that there is arachnitis, just as one would say there is pleuritis if pulmonary symptoms arose. Reasoning is, therefore, in favor of those who maintain that rheumatic meningitis and pleurisy are similar. But let us examine whether anatomical experience confirms these theoretical views.

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* De la Chorée. Rapports du rhumatisme et des maladies du cœur, avec les affections nerveuses et convulsives. Mémoires de l'Académie de Médecine, t. xx, 1850.

In the majority of post-mortem examinations, nothing has been found except occasionally some congestion of the pia mater, as I found in the man who lay at No. 16. In most cases, I repeat, nothing at all is found, no fluid in the ventricles or in the arachnoid sac, no injection of the cerebral tissue.

As no material change can be detected, the advocates of meningitis appeal to the rheumatic nature of the meningitis, and to the fact that rheumatism is a complaint in which there is no tendency to suppuration. I admit that articular rheumatism does not show any tendency to form pus, or to leave fibrinous deposits inside joints. The advocates of meningitis, therefore, assert that, when rheumatism attacks the meninges, meningitis results, just as arthritis follows when the disease seizes upon a joint; and, as in rheumatic arthritis, no fibrinous exudations occur, neither are these met with in rheumatic meningitis.

To this purely theoretical way of reasoning from analogy I will oppose a practical argument also drawn from analogy.

The serous membranes of the pericardium and pleura are anatomically identical. Now, we daily meet with cases of rheumatism in which the pericardium and pleura are involved, but within a few hours of the occurrence of such complications physical signs reveal the existence of unquestionable organic lesions. Thus, on auscultating the lungs, the respiratory murmur is found to be less distinct, while on applying the stethoscope over the heart, a friction-sound, like the creaking of new leather, is heard. After the lapse of another twenty-four hours, all the physical signs of effusion, or indicating the presence of false membranes, are detected.

Now, since the arachnoid is anatomically identical with the pericardium and pleura, how, it may be asked, should it be privileged to escape the common law, and why should not fibrinous deposits and other exudations be found in its sac, as in that of the pericardium and pleura? But as no such changes are found in the bodies of individuals who have died of cerebral rheumatism, we are justified in coming to the conclusion that meningitis was not set up.

It would be vain to try and account for the absence of the lesions of meningitis by the rapidity with which death occurred, and to assert that the same thing would happen in the case of pericarditis and pleurisy if the patient were to die at the onset of these complications. This argument might apply to the case of my patient at No. 16, as he died very rapidly; but it cannot be applied to all the cases which have been observed. For this pretended arachnitis has been known to last from two to six days, and as no lesion was found in such cases, we are compelled to admit that there could have been no meningitis. In cerebral rheumatism, then, we find neither the symptoms nor the usual anatomical changes of inflammation of the meninges.

But what is it that happens in such cases, and what is my opinion of the nature of cerebral rheumatism, if not in all, at least in the majority of instances? Allow me to make a digression, in order to explain my meaning thoroughly.

When the brain and the spinal cord, or the peripheral nervous system, are concerned, one is, in general, satisfied with explanations which are by far too easy. Thus paralysis, which sets in suddenly, is ascribed to congestion or softening of the brain, or hemorrhage into it. The existence of hemorrhage and softening is frequently demonstrated; but it is not so with congestion, which is too easily admitted, and without any other reason than that there could have been no other lesion.

But let us analyze analogous cases. An individual becomes affected with chorea, which resists treatment for the space of four months, and which, as usually happens, is complicated with paralytic and convulsive phenomena. For, as you are aware, one half of the body is, in such cases, weaker than the other, so much so indeed that the dynamometer marks 1 for one side and 19 for the other. There is, at the same time, then, muscular paralysis, convulsion, and, frequently, even disturbance of peripheral sensibility, anæsthesia, or hyperæsthesia. The nervous system is, therefore, sufficiently disturbed to induce the belief that the spinal cord is disorganized, and even the brain itself, as there is often impairment of the intellect.

But if you examine the bodies of persons who have died from the violence of the chorea, and most carefully search in the brain and spinal cord, you will find neither intense congestion, nor softening, nor extravasation; in a word, you will detect no serious lesion which can adequately explain the symptoms noted during life. Up to the present time, at least, no such lesion has been detected.

The same thing happens in other neuroses, in tetanus, for example. An individual has undergone a trifling surgical operation, say a month ago; he is nearly well, and there scarcely remains a few granulations which require to be touched with caustic before the cicatrization is complete; when, suddenly, stiffness is complained of, first in the jaws, then in the neck, without any fever being lighted up, and is soon followed by the fearful convulsions and rigidity which characterize tetanus, and last from four to eight days, until death closes the painful scene.

Here is a very grave and powerful neurosis, affecting motility and impairing the intellect at the last, since death is preceded by stupor; and yet, on dissection, nothing is found, absolutely nothing.

Look at hydrophobia again. Nothing has been found after death to explain the phenomena of that fearful complaint. There is just now, in one of my wards, a woman suffering from tetany, that curious affection in which one or both hands are rigid, the fingers straightened, with their ends closely pressed together, and giving to the hand the appearance of a beggar's hand stretched out in the act of begging. This paroxysmal complaint is merely after all local tetanus, affecting the forearm and hand, and is unquestionably of nervous origin. But it does not depend on a definite lesion, such as inflammation of, or hemorrhage into, the nervous centres; and although it may possibly be due to transient paroxysmal congestion, the fact can be more easily assumed than demonstrated. At all events the nervous system must be modified in some way or another in this complaint.

In an outbreak of amaurosis which suddenly attacked the inmates of the Fénélon Asylum, ophthalmoscopic examination of the eye detected nothing abnormal. Hemeralopia often occurs epidemically in large barracks, or on board ships, independently of any change in the hygienic circumstances of the individuals, and unattended with abnormal appearances in the eyes that are affected. It gets well almost spontaneously, disappearing in the same way as it came on, and leaving us as much in the dark as to the cause which produced it as to its anatomical constitution. Shall one say again that congestion was present? but such an explanation would be unsatisfactory.

In true meningitis, when disturbances of innervation exist, these are not due to inflammation of the cerebral meninges, but to the circumstance that the brain-substance itself is involved in the congestive or inflammatory process. We find after death anatomical proofs of this extension of the inflammation. But the case is very different with cerebral rheumatism, in the course of which true symptoms of meningitis do not show themselves, while after death no meningeal or cerebral lesions are discoverable. We are thus led to infer, both from clinical observation and from reasoning, that in consequence of the cerebral rheumatism, the nerve-substance has probably undergone a modification analogous to that which is believed to occur in tetanus, hysteria, &c., a modification the nature of which is yet obscure and not anatomically demonstrable, but which nevertheless exists, as everything at least seems to indicate, although it cannot be referred to any nosological type.

From these considerations, to which I might add many others, I hold to the opinion that the phenomena of cerebral rheumatism are, in general, those of a neurosis, much more than of an inflammation or even a congestion having definite anatomical characters which can be easily made out.

I now pass on to the *mode of occurrence* of cerebral rheumatism. According to most writers on the subject, there must already be articular rheumatism before cerebral complications can arise; in other words, cerebral rheumatism cannot occur at the very first onset, or at least has never been known to precede the joint affection. It may be, however, that this happens more frequently than is believed, as facts will show. I had lately under my care a man who complained of a very intense pain along the spine, and who was paraplegic. I at first thought that he was going to have small-pox, but no eruption occurring at the usual time, I examined him more carefully, and suspected acute myelitis; but a few days afterwards he was seized with articular rheumatism. Previous to affecting the joints, the rheumatism had therefore attacked the spinal cord first.

I have already mentioned the case of a young girl who was admitted under my care, three or four months ago, with symptoms of threatening variola. At first she presented symptoms of spinal, and then of cerebral, disturbance, with amblyopia, but all these disappeared as soon as acute articular rheumatism showed itself.

In both these instances, then, spinal and cerebral lesions preceded the joint affection. Other cases of the kind may occur, as you may imagine, and it may happen that light is not thrown on them by the supervention of rheumatism in the joints, so that they may be mistaken for cerebral fever, instead of being recognized as cerebral rheumatism. Cases like mine justify one in admitting the possible occurrence of primary cerebral rheumatism, although the joint affection is more commonly the first to show itself. The same conclusion applies to the other manifestations of rheumatism, such as endocarditis, pericarditis, and pleurisy.

You probably recollect the case of a young man who was admitted under my care, with acute endocarditis, as shown by fever and a blowing murmur at the heart's apex. After a few days, the rheumatic nature of the complaint was proved by the supervention of pain in the joints. The patient had never had rheumatism before, and it was clear that the disease had attacked the endocardium primarily, instead of secondarily, after the joints.

By the side of this case, I will relate another, in which actual proof was not obtained, as the rheumatic influence which caused endocarditis did not subsequently give rise to articular disease, just as it must have sometimes occurred that primary cerebral rheumatism has not been followed by arthritic manifestations. The subject of this second case was a young girl who had an attack of acute endocarditis, which ran its course without being corroborated by the supervention of articular rheumatism. But there is no reason why we should not say that the case was one of primary rheumatic endocarditis, not followed by articular rheumatism, just as we meet with instances of rheumatism without endocardial complication. Such cases are very rare, and in the immense majority of instances articular rheumatism precedes the cerebral, cardiac, or pleuritic manifestations.

I have told you that cerebral rheumatism was, in my opinion, a neurosis, and not a rheumatic inflammation, and that the cerebral centre could be attacked primarily before the joints, so that you may infer from those statements what my answer would be to the question whether cerebral rheumatism is due to *metastasis*. I must, in the first place, define what I mean, and what should be meant, by the term metastasis, and I will do so by giving you examples in illustration.

Acute articular rheumatism is an affection with multiple manifestations, which involves four, ten, thirty, and sometimes a hundred joints at the same time, as when it attacks simultaneously the articulations of the hand, foot, and vertebral column. Now, when the disease migrates from one knee to the other, we do not say that there is metastasis, but simply that the rheumatic influence which yesterday affected the right knee to-day involves the left knee, and will probably attack some other joint to-morrow. It is the same morbid cause which seizes on various articulations in succession, and affects parts with which it has a pathological relation. Such a case is not one of metastasis.

But I will now give you instances of true metastasis. An individual has the mumps, that strange epidemic complaint, which is in the highest degree contagious, and is characterized by sudden swelling of first one and then of the other parotid gland, the secretion of which is diminished or suppressed, and is accompanied by intense fever. All these symptoms disappear, after four, six, or eight days at the most, some diminution of the salivary secretion alone remaining. This disease is generally of a mild character, but not always so, for it occasionally happens that the swelling of the glands goes down suddenly, and that the patient exhibits nervous symptoms which are sometimes extraordinary. He remains in that condition for a day or two, and then a testicle is suddenly affected in a man, or a mammary gland in a woman. This is an instance of true metastasis. The primary lesion disappears, while an organ essentially different from the first becomes the seat of disease. There is metastasis because there is no necessary relation between the primary morbid phenomenon and the testicle, none at least analogous to that which exists between rheumatism and the joints.

Now, in cases of cerebral rheumatism, is there metastasis? Certainly not, because the rheumatism does not leave the joints to seize upon the brain; it has spread, dispersed itself, I would be disposed to say, so as to involve a part which it had not previously attacked, but it still persists in the joints after it has affected the brain. Hence there is some ground for supposing that, in involving the brain or the meninges, the rheumatism has merely selected a fresh seat, in the same way as when it spreads to the pericardium or the pleura. It is true that, when the pleura is seriously inflamed over a large area, the rheumatism leaves the joint after a few days, but, I repeat, not through metastasis, but in accordance with the law laid down by Hippocrates: Duobus laboribus simul obortis, non in eodem loco, vehementior obscurat alterum.

In the case of the man at No. 16, the articular pains were present a few hours before the cerebral complication arose; they had become less severe under the influence of quinine, but they did not disappear suddenly, as they do in real metastasis. As to the woman at No. 2, she exhibited cerebral symptoms during the continuance of the pain, and after those symptoms had disappeared, the joints continued to be painful.

One may often be misled into believing that the rheumatism has left the joints when it attacks the brain, from the severity of the cerebral symptoms masking that of the joint-affection. The patient, in his delirium, tosses about wildly, moving in every direction the limbs which he previously kept motionless on account of the pain in his joints; and because he is no longer conscious of this pain, those about him believe that his joints are no longer affected. But this is evidently a mistake; the articular rheumatism still persists, for there are still swelling and redness, and exquisite sensibility, but the latter is masked by the delirium, and by the different nervous condition in which the patient is. Under whatever aspect we view the question, therefore, either in the light of general pathology or in that of the phenomena observed in the course of an attack of cerebral rheumatism, we are justified in not regarding the implication of the brain in rheumatism as due to metastasis.

Let us now inquire into the possible *causes* of cerebral rheumatism, and, first, into those that are independent of the peculiar treatment employed, for certain modes of treatment have been accused of favoring cerebral complications in rheumatism. When I related to you the case of the woman at No. 2, who had almost simultaneously articular and cerebral rheumatism, I asked you to bear well in mind the fact that in 1848, after violent emotions, she had manifested symptoms of brain disease, and had been treated for insanity at the Salpêtrière for thirteen months. This woman then showed an unquestionable predisposition to brain disease (whether this was mania, epilepsy, or lipomania, it matters little.) Subsequently, on becoming affected with articular rheumatism, before the disease has spread much, she is seized with cerebral rheumatism, which gives rise to stupor of forty-eight hours' duration. The patient got well nevertheless.

On the other hand, the man at No. 16 was addicted to drink; he was in a state of constant excitement, and was, as it were, stupefied by spirituous liquors, so that his brain was predisposed by this permanent irritation to get disordered. On his being affected with acute articular rheumatism, his brain gets soon implicated, and he dies.

In 1825, I attended, in St. Martin Street, a merchant suffering from acute articular rheumatism, who exhibited such severe brain-symptoms that I told his friends in the most positive manner that he would not get well. I was, in consequence, requested to cease attendance; but after a few days the patient's fierce delirium disappeared, and he recovered perfectly. Some time afterwards he had a second attack of acute articular rheumatism, attended with excessive pain, considerable swelling, livid discoloration of the integuments, and I detected gaseous crepitation, deep inside some joints, indicating an incipient stage of gangrene. This time the patient died.

Now, all the brothers and sisters of this man had been, or were, insane, and it was a matter of surprise with those who knew his family that he was the only member of it who had not yet become insane. On his getting rheumatism, however, he is seized with cerebral rheumatism, in consequence of his hereditary predisposition to diseases of the brain. I have, in another lecture, mentioned the case of a woman, many members of whose family were insane, and who, on falling ill of rheumatism, was carried off by cerebral rheumatism.

In cases, therefore, of cerebral rheumatism, we learn, from the previous history of the patient, that he has at some period or other shown grave cerebral symptoms, or that there exists in his family an hereditary predisposition to grave neuroses.

The same thing may occur in other diseases besides rheumatism. Thus, in individuals in whose family history neuroses or insanity may be traced, or whose brain is constantly stimulated by the use of spirituous liquors, fearful cerebral symptoms may develop themselves in the course of an attack of variola (as you very recently saw an instance in one of my wards), or after some severe injury. There is, therefore, an hereditary or acquired nervous predisposition, in virtue of which some individuals are liable to cerebral complications during the course of various affections, and especially to cerebral rheumatism in the course of an attack of articular rheumatism. Let us next inquire whether, as some practitioners believe, any particular mode of treatment favors the occurrence of cerebral rheumatism. There are, as you know, two chief and opposite methods of treating articular rheumatism. Some practitioners advocate *bleeding*, others administer *quinine*. The former ascribe to quinine the production of cerebral rheumatism; the latter, in their turn, accuse the practice of bleeding of bringing on that complication. In this discussion, which has been carried on without due regard to truth, and, occasionally, to rules of good breeding, the advocates of the quinine treatment have, apparently, come off worse, and for this reason:

Few practitioners nowadays open a vein in acute articular rheumatism; of fifty physicians attached to the Paris hospitals, perhaps not more than four bleed, while the rest prescribe quinine. Now, say that each of them has ten cases of articular rheumatism under his care, there will then be 460 cases treated by quinine, and only 40 by bleeding. As a matter of course, the proportion of cerebral rheumatism will be much greater in the first than in the second group of cases, in fact, in the ratio of 460 to 40. But the conclusion has been drawn that a greater number of cases of cerebral rheumatism occur in the practice of those who give quinine. This is true if mere numbers be taken; but it is false if the numbers be compared in their logical proportion.

Dr. Beau and Dr. Briquet, who give quinine, affirm that it is a most dangerous practice to bleed in acute articular rheumatism, and they ground their opinion on the specious fact that repeated bleedings increase the amount of fibrin in proportion to that of the other constituents of the blood, and, therefore, favor the tendency to inflammation of the brain chiefly, which is weakened by the anæmia induced. On the other hand, the advocates of bleeding assert that, by exciting the brain, quinine renders it liable to the rheumatic influence. Truth is mixed with error in these statements. More than sixty cases of cerebral rheumatism are now on record, seven or eight of which proved fatal after a course of bleeding, practiced, as Dr. Briquet expresses it, in the orthodox manner. In other cases, which proved fatal, the patient was bled two or three times only, and was cupped several times. Of those who died in consequence or in spite of the sulphate of quinine, some had taken a small quantity of the drug, others moderate, and others, again, a very large amount of the salt.

The woman at No. 2 had not been bled, and had not taken quinine; she was nevertheless attacked with cerebral rheumatism, but got well.

The man at No. 16 took, for four days, a scruple; for three days, thirty grains; and on the last day of his life, forty grains of quinine; and these moderate doses did not bring on tinnitus aurium or mistiness of vision.

In cases reported by Drs. Bourdon, Requin, and Gubler, cerebral rheumatism came on when the patient had only taken ten grains of quinine. Dr. Beau quotes a case in which, in spite of the cerebral rheumatism, he persisted in the administration of quinine, and the patient recovered.

To sum up, then, cerebral rheumatism does not seem to be brought on by any treatment in particular; it depends on the existence of a special predisposition, acquired or hereditary, which I have endeavored to bring out prominently, and is not caused by bleeding or by quinine.

Now, as to the *treatment* of cerebral rheumatism. The cases should be divided into two groups: those in which the disease is merely imminent, and those in which it has broken out distinctly. Thus, when an individual, suffering from articular rheumatism, shows signs of commencing nervous excitement, becomes garrulous, complains of feeling hot all over, and speaks despairingly of his own case, cerebral rheumatism may be diagnosed as imminent; but can we do anything for the patient? I believe that the best plan consists in encouraging the articular manifestations of the disease. If they have abated, attempts should be made to bring them back by means of sinapisms, or of blisters to the joints. Opium and musk should, at the same time, be given internally. This again, is, in my opinion, the most rational line of practice to pursue when the brain has become involved. I have succeeded in curing three patients who were under my care from the onset of the cerebral rheumatism by means of musk and opium. I have failed in other instances, and twice recovery took place without any active treatment having been had recourse to; one of these two last cases was that of the woman at No. 2, which is an illustration of the *vis medicatrix naturæ*.

LECTURE LXVII.

VERTIGO A STOMACHO LÆSO.*

"VERTIGO AB AURE LÆSA."—"VERTIGE LABYRINTHIQUE."—*Stomachic Vertigo is often mistaken.—Symptoms which characterize it often considered to depend on Cerebral Congestion, and consequently the Treatment adopted often aggravates it.—Vertigo depending on Lesions of the Labyrinth resemble Stomachic Vertigo.—Treatment of Stomachic Vertigo is that of Dyspepsia.*

GENTLEMEN: A woman, aged fifty-seven, having symptoms to which I wish to direct your particular attention, has occupied bed 29 *bis* in St. Bernard Ward for about two months.

This patient generally enjoyed good health, till she reached the critical period of life, when she suffered much from menorrhagia, by which her strength was greatly enfeebled. At the same period, she likewise began to suffer from a feeling of weight and pain in the epigastric region, and from pain shooting through the abdomen, back, loins, and precordial region, in which latter situation, she felt tingling sensations. She retained her appetite, but had a disgust for farinaceous vegetables, and cold food, which increased the feeling of weight in the epigastrium. The digestion of cold food was difficult, and attended by general discomfort and flushing of the face. Warm food, on the contrary, calmed the pains which arose during the intervals between repasts.

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* Vertigo Stomacal. Vertigo per consensum ventriculi of the old authors.

Gentlemen, let me repeat the history given by the patient of the symptoms to which I now wish to call your special attention.

Four days before her arrival at the hospital, she had left her native place, where, for six previous days, she had been subjected to much fatigue and mental worry. Without feeling at that time exactly that she was an invalid, she observed that she was losing her appetite, and that her digestion was becoming unusually painful.

On the day of her leaving, after having made her usual breakfast on coffee and milk, she set out to walk the three leagues which separate her village from the town of Saint-Quentin. She had formerly, more than once, accomplished this journey without being at all fatigued; and this was even the case since her arrival at Paris six days previously. She was proceeding on her way, in company with one of her children, when, after having walked hardly three kilometres, she was all at once seized with giddiness and weight in the head, unaccompanied by severe pain, eye-dazzling, or any syncopic tendency. It seemed to her that the earth was opening in front of her, her legs bent, and she felt as if being irresistibly borne towards an open abyss which she believed she saw at her feet. This singular hallucination was accompanied by nausea, and a desire to vomit: in fact, she did bring up some of the food which she had taken in the morning, and, subsequently, a small quantity of clear fluid. She nevertheless retained unimpaired her consciousness, and was perfectly aware of the error of her senses, at the very time she was screaming with terror and beseeching her son to prevent her from falling into the abyss. This extraordinary condition lasted ten minutes. In describing it to me, she said that her sensations might be compared to those experienced when looking down on the ground from the top of a steeple. Her giddiness was so violent, that to prevent her from falling, it was necessary to make her sit down, and afterwards lie down on a bed brought to her from the nearest house. The sensation of weight in the head was soon succeeded by acute pain: to use her own expression, she felt "as if her skull was being split." For several hours she was unable to bear the movement of a carriage, so that it was necessary to leave her for some hours in a house where she had been hospitably received. During the evening, the symptoms seemed quite to disappear, and the patient felt well enough to return home on foot: in fact, she was the better of the walk, for on reaching her residence, the only remaining symptom of her attack was a little heaviness of the head: she ate well, and had nine hours of very tranquil sleep.

On awaking next morning, she complained of a little heaviness of the head and feelings of numbness; but she breakfasted as usual, and then again started for Saint-Quentin. After accomplishing this walk of three leagues, feeling quite re-established, she left by rail. She had scarcely gone half the journey, when she was seized with symptoms precisely similar to those of the previous evening. At the Creil station she got out of the carriage, when the giddiness was so great, that to avoid falling, she had to lean on the arm of a fellow-traveller. The carriages, she told me, seemed as if they were dancing, as if they were being carried high up into the air and then let fall, to be engulfed in the earth which seemed to open; and she felt herself as if being drawn into the abyss. She continued to experience these sensations for nearly ten minutes; and for a long time afterwards—even at the time she described them to me—she retained the impression of terror which they had originally caused. As on the previous evening, however, she was perfectly aware that the whole was a delusion, and gave clear replies to the questions addressed to her. She returned to the railway carriage, retaining, however, some degree of general discomfort, and from a fear of increasing this uncomfortable state, she remained silent during the remainder of the journey. On her arrival at Paris, two hours afterwards, the giddiness did not return, but the state of general discomfort continued, nor had it left her when she came next day to the Hôtel-Dieu to visit her daughter, who was a patient there. She had then also pain in the head, which she described as resembling a sensation of having her forehead split. In accordance with the recommendation of her daughter, she asked to be admitted as a patient, and was received into our wards.

On examining the abdominal region, I found that there was slight enlargement of the liver; but there was no trace of jaundice, ascites, or anasarca. On making pressure over the pit of the stomach, there was produced an increase of the pain, which the patient said never left her.

Rest, restorative diet, and a methodical use of very simple measures soon caused the symptoms to disappear. To-day, the woman is so thoroughly recovered, that she wishes to return to her home.

What had this patient? You remember, gentlemen, what my diagnosis was from the first. I told you that the singular phenomena which she described to us depended upon the stomach; and that we had to do with that kind of vertigo which I have designated *vertigo a stomacho læso*, the same affection which the old authors denominated *vertigo per consensum ventriculi*, names for which my pupil, Dr. Blondeau, in a memoir on this subject, published at my suggestion, has substituted "*vertigo stomacal*," a term which is less regular but much shorter.*

This is perhaps the most common kind of vertigo: it is at least that for which we are most commonly consulted in private practice. The obstinacy of the symptoms which characterize it, its constantly returning, and its seeming seriousness, strangely torment those affected with it, deceive the persons about the patients, and often mislead even the medical attendants, who, misconceiving its nature, resort to treatment diametrically opposed to that which is appropriate. Giddiness originating in the stomach is often imputed to supposed cerebral congestion, a mistake leading to antiphlogistic treatment by bleeding, leeching, purging, and rigorously low diet—means which, in place of curing the disease, increase its severity; whereas, speedy benefit is obtained by suitable restorative diet and tonics.

The vertiginous phenomena, however variable they may be in their forms, have a something about them which is peculiar; and an experienced, attentive observer will suspect the nature of the symptoms, even when they do not present anything essentially characteristic of the affection of the digestive functions with which they are associated.

The phenomena to which I refer are sensations of emptiness and swimming in the head; or sometimes, the patient feels as if his temples were being tightly clasped by an iron ring. Presently, he experiences a sensation of icy coldness. Some patients tell us that they have a mist before their eyes, and that the objects which they see present a confused diversity of colors: others describe a large black wheel as moving before them with excessive rapidity. But the form of this affection which you will most frequently meet with is that to which the name *gyrosa* has been applied; when the individual is standing, everything about him seems to be whirling round: he is obliged to shut his eyes, and remain absolutely motionless, for he feels his legs tottering and bending under him, as if he were going to fall; and sometimes, indeed, he does fall. If he be lying down, he thinks he sees his bed revolving on an axis passing through his head and feet; or it may be, that the patient sees himself involved in the rotatory movement.

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* Blondeau (Léon): Archives Générales de Médecine for September, 1858.

It is a remarkable and essentially characteristic fact, that to whatever degree these symptoms are present, the patient never loses consciousness of his actions: even when he falls, there is no loss of consciousness, and he never misunderstands the nature of the odd sensations and hallucinations which terrify him. You recollect what we were so often told by our patient of St. Bernard Ward: she said that though it was impossible for her to shake off the feeling of terror at seeing the open abyss at her feet, although the terror was renewed even by the recollection of this sight, she knew perfectly well that it was an illusion of the senses. Other persons have told me of their having had analogous hallucinations, they knowing them at the time to be mere illusions.

The vertiginous phenomena are generally accompanied by squeamishness, which the sufferers compare to sea-sickness. It is indeed *nausea* in the literal acceptation of the word, which is derived from ναῦς, the Greek for a ship.

The slightest cause may bring on these vertiginous affections. The immediate cause of their development may be a trellised wall, a range of bars, or a striped pattern in the hangings of a room. The trellis, the bars, or the stripes look commingled, as if in a sort of mist, and they have a dim appearance. A rather sudden movement, or raising the head, may suffice to induce the phenomena. It is an interesting peculiarity, worthy of being noted, that, in general, the phenomena do not occur when the head is *lowered*, which is the reverse of what occurs when the vertigo depends on congestion of the encephalon.

These phenomena being constantly reproduced by the slightest cause, or even without any immediately exciting cause, get so strong a hold of the minds of the patients, that they, forgetting as it were their other dyspeptic symptoms, only think of, and only mention to the medical man, those which we are now considering. You have observed that the woman in St. Bernard Ward, and two patients in St. Agnes Ward (whose cases I shall have to refer to immediately), spoke only of the vertiginous symptoms, and made absolutely no mention whatever of the signs of disorder in the functions of digestion, to which, nevertheless, they were referable.

The gastric disorder, however, is usually indicated by well-marked symptoms. There are pains in the stomach, which are most violent after the ingestion of food, particularly of some kinds of food: they are increased and propagated to the back by pressure with the hand over the pit of the stomach: there is a feeling of weight, of cramp, of acute pain shooting through the chest and abdomen, extending even to different parts of the body, and accompanied by a sensation of heat or burning in the region of the stomach. There are flatulent symptoms, acid eructations (not inodorous in general), and vomiting of a glairy mucous character, or sometimes of food. Constipation is more common than diarrhœa, although both may alternate in the same individual. These symptoms are absent in a few exceptional cases; but if you interrogate the patients on the subject, you will almost always hear them complain of their digestion being slow and laborious.

But bear in mind, that, generally speaking, the vertiginous symptoms do not occur during, but long after digestion, that is to say, using the vulgar expression, when the stomach is empty. Here let me mention a fact bearing upon the treatment of this singular affection: the administration of a small quantity of food, such for example as a cup of broth, or a biscuit soaked in a little wine, will often prevent the symptoms from supervening, and stop them, should they have set in.

It would, nevertheless, appear, that under certain circumstances, eating may be the determining cause of the symptoms, if we may judge from what we saw in the two patients in St. Agnes Ward, to whom I have just referred.

One of these patients was a young man of twenty-five years of age. He stated that he had generally enjoyed good health, having never had any ailment except an affection which was probably syphilitic, as he had been treated for it in the hospital for venereal cases: beyond this fact, he gave no precise information regarding that illness.

The following is the account he gave of the symptoms for which he came into the hospital. For a month, he had suffered from headache and pain in the right side; these were the symptoms which had chiefly annoyed him. He had abdominal pain, which was increased on pressure; but on making a very careful examination, I was unable to discover any sign of visceral lesion. The peculiarity in the abdominal pain was its subsiding spontaneously, immediately after a repast: but the patient had also cerebral symptoms which made him very anxious. They consisted in dizziness, impaired vision, buzzing in the ears, heaviness of the head, and a peculiar feeling which he compared to incipient drunkenness. He affirmed that he was not in the habit of drinking either wine or any other intoxicating liquors to excess. When he attempted to rise from table his legs bent under him, he experienced a feeling of general discomfort, and felt as if he were going to faint: to save himself from falling he was obliged to lean on whatever was within his reach. He never, however, lost consciousness.

These symptoms, which recurred, I repeat, after the patient had eaten, which recurred several times within the first hour immediately following a meal, were unaccompanied by pain or uneasy sensation in the region of the stomach, and constituted with the gastralgia, or rather enteralgia of which I have spoken, the only symptoms characteristic of the disorder of which the digestive functions were the seat.

It sometimes happens, though not often, that the vertiginous symptoms are not only the predominating morbid phenomena, but are the only ones of which the patients complain, and that because they suffer from none other: the dyspepsia in which they originate does not otherwise show itself. Here is a case in point.

A lady of a certain age came to Paris from Bordeaux to seek medical advice in respect of cerebral symptoms with which she had been distressed for several months, and which consisted in giddiness which scarcely allowed her a moment's tranquillity. This giddiness was brought on by the slightest cause, and proceeded so far as to produce a syncopic state, obliging the patient to keep the recumbent position. The bustle of the streets, the sight of the passers-by, or of a carriage going along at a rather quick pace, so decidedly caused the symptoms to return, that this lady was soon unable to leave her own room. She believed that she was threatened by an attack of apoplexy, and her fears were increased by those around her; their officious advice fanned her morbid fancies. To avoid the congestion which she dreaded, acting in harmony with her own theory that nothing should be done to augment the quantity of blood, she condemned herself to a very severe regimen, and lived exclusively on soups and beef tea. Her appetite decreased; but digestion continued to be properly performed. By pursuing this regimen, she was reduced to a deplorable state of cachexia. When my friend Dr. Lasègue was called in, he was struck by her emaciated appearance and yellow skin. At first, he thought that there was cancer, but minute examination disclosed no sign of any such affection: he soon became convinced that the vertiginous symptoms depended on derangement of the functions of nutrition, and were increased by abstinence. Dr. Lasègue asked me to see the patient with him in consultation: I did so; and entirely concurred with his diagnosis. The result showed that we were right. By the administration of tonics and a restorative diet, in eight days a remarkable change in her state was effected. The attacks of giddiness were much less frequent, and in a short time the patient ceased to be troubled with them. In six weeks she was well and plump.

You perceive, therefore, gentlemen, that vertigo often supervenes in persons whose digestion is not at fault in any way. The appetite is good, the alvine evacuations regular, and there are no acid eructations; but, nevertheless, success attends the employment of treatment directed against dyspepsia. I have frequently asked myself, whether the treatment which in these cases I directed against the affection of the stomach was not, unknown to me, addressed to the nervous system; and whether I had not diagnosed a gastric affection, rather from the effect of treatment than from the symptoms of the disease; whether I had not been led into an error in diagnosis, by obtaining success from treatment usually employed with benefit in dyspepsia?

I know very well that nausea and vomiting often occur in cases of vertigo when the tissue of the stomach is in a perfectly normal state, and when its secretions are exactly what they ought to be; but who is not aware that in numerous affections of the nervous system, vomiting is a common symptom: without speaking of cerebral fever, let me mention sea-sickness, and the dizziness produced by waltzing, in both of which the gastric symptom is that which chiefly occupies the patient, and of which he complains the most bitterly. Now, in the vertigo which is called, rightly or wrongly, stomachic [*stomacal*], the nervous symptoms, and those observed in connection with the stomach, do not, for example, greatly differ from those of vertigo depending on lesions of the labyrinth.

It is right to state, however, that the latter form of vertigo very seldom yields to the treatment which is generally successful in simple vertigo, accompanied only by nausea and vomiting.

Stomachic vertigo occurs pretty frequently during convalescence from long illnesses, such as serious fevers, particularly when they have caused a great disturbance of the functions of nutrition, reacting on the digestive canal, while it still remains in a flagging state.

This occurred in our second patient in St. Agnes Ward.

This man was forty-eight years of age. He was of a robust constitution, and had always been in good health till within fifteen months of his coming into our hands, when he was seized with severe scurvy caused by the miserable diet to which poverty condemned him. He lived in a very unwholesome lodging: his only room was badly ventilated, and being immediately under the roof, was exposed to cold, damp, and all vicissitudes of weather. I had an opportunity of seeing this man in another service in this hospital to which he had been admitted. There were large ecchymotic spots over the whole surface of the body, some of which were as large as the hand: the gums had been destroyed by sanious ulcerations: and there was great diminution of strength. The disease continued two months; or at least at the end of that period, the patient left the Hôtel-Dieu. For more than three months after this, the patient had obstinate lienteric diarrhœa: his appetite, however, remained good, and he ate a large quantity of food, but it was not aliment fitted to restore the waste of the body, being chiefly bread, and soups made without any meat. Digestion was performed badly: two hours after a meal, the alimentary substances were passed by stool almost in the state in which they had been swallowed: the stools were as many as twenty during the twenty-four hours. For five months, the general debility was so great, that the man could not leave his room. From the extreme degree to which emaciation had proceeded, he was unable to sit, and had therefore to remain in bed.

As soon as he was able to leave the house, this unfortunate person resumed his ill-remunerated labor, that he might obtain sustenance for himself and family. Under these circumstances, his food was very inadequate: digestion, too, was imperfectly performed, in consequence of his having lost a great number of his teeth, and those which remained being so loose in their sockets from the scorbutic affection, that he could not masticate his food. His new lodging, though less unwholesome than the former, was still a very insalubrious abode.

Hygienic conditions of so unfavorable a description were not calculated to restore rapidly his lost strength. When he was readmitted to the hospital, he was still complaining of being very weak; but the cause of his coming back to the Hôtel-Dieu was a return of the symptoms from which he had suffered, and which occasioned him great anxiety in consequence of their having become much aggravated during eight preceding days.

I shall now tell you what he complained of. He felt numbness, a sort of paralysis of the muscles which move the jaws, and of the tongue: this interfered with the opening of the mouth, and prevented him from being able to articulate certain words. His voice was hoarse, and he could no longer cry his wares on the streets as a costermonger. When I asked him to open his mouth, he was only able to expand his jaws half way; and that occasioned pain in the temporo-maxillary articulations. His tongue, which he could not protrude beyond the dental arch, obviously deviated to the right. On examining the back part of the mouth, I discovered that there was deformity of the veil of the palate which was not regularly concave: on the left, it was flattened, and the uvula was warped on that side; however, on touching the parts with a bristle, or the nib of a pen, it was seen that motor power remained, and that muscular contractions were excited.

For eight days, the patient suffered from headache, which was uninfluenced by the stomach being full or empty: he compared the headache to that experienced after a drunken debauch. In relation to drinking, he affirmed, that he never took brandy, and very seldom drank alcoholic liquors of any kind. He added, that his brain was disturbed by less than one tumbler of wine, of which, in former times, he could easily have taken three litres without feeling any discomfort.

He slept well, and on awaking his head was free, but after sitting up for some minutes, the pain returned. He felt better when lying down, provided the head were placed rather high. When standing, he experienced tingling sensations in the eyes, and then disturbed vision: he saw ascending and descending sparks, and objects seemed to be turning and dancing around him: ere long, the sight became exceedingly dim, and he felt as if in a mist. To prevent himself from falling, he was obliged to lean on any support which might be nearest. Upon one occasion, he did fall; but there was no loss of consciousness, and, in a few seconds, he rose without assistance. These vertiginous symptoms increased when he raised or lowered his head: but they were less severe when he looked up than when he looked down, which is the reverse of what is usual in such cases. For some time, he tried to serve masons, but was obliged to renounce the attempt, in consequence of inability to mount scaffolding, without feeling as if his head were going round, a symptom to which he had not been subject before his illness.

When the vertigo supervenced, the headache increased, and the patient felt as if his head were splitting: sometimes he had nausea, but he never vomited. He stated, that neither before nor after eating had he ever had what could properly be called pain in the stomach.

The lienteric diarrhœa, by which he had been tormented for more than three months, was succeeded by constipation to such a degree, that he was eight days without a stool. For some time, however, the movements of the bowels had resumed their usual regularity.

In addition to the general debility of which he complained, he had a constant feeling of weight in the lower extremities: neither exercise nor the warmth of bed could warm him. Before he had the scurvy, he perspired easily in the feet; but now his feet remained dry, when all the other parts of the body were covered with sweat.

Rest for a week, good food, and medical treatment, consisting chiefly in the administration of alkalies, sufficed to stop the vertiginous symptoms; and this man felt so well after being ten days in our clinical wards, that he then expressed a wish to return home to his family.

Whatever relations this case may bear to the subject now before us, it is one of considerable complexity, for the vertigo may be considered as the result of profound derangement of the nervous system during a long and serious illness. According to this hypothesis, the symptoms were of the same kind as the paralysis of the veil of the palate, and the paralysis of the tongue, observed, as I have had occasion more than once already to tell you, as a sequel to certain diseases, such as diphtheria and typhoid fever, which produce a great strain upon the system. In this case, however, it was evident that the digestive organs were the organs more particularly involved, as was shown by the long continuance of the lienteric diarrhœa. So dependent were the vertiginous symptoms upon disordered digestion and nutrition, that good alimentation for a week was sufficient to effect a cure.

Nevertheless, gentlemen, this case has not that distinctiveness which characterizes those we most frequently meet with in practice. Some time ago, I was called to attend upon a magistrate, sixty years of age. Consequent upon very assiduous mental work, generally performed after dinner, he had been feeling a weight at the stomach, and frequent acid eructations. The appetite was becoming less and less every day. All at once, when looking up to the ceiling, he became exceedingly giddy, saw objects whirling round him, and felt at the same time transient nausea. Being anxious about himself, he sent for his medical attendant, who prescribed purgatives and sinapised foot-baths. The malady, however, made rapid progress. There was vertigo not only when the patient was sitting or standing, but also when he was lying in bed. He had incessant nausea, which he compared to seasickness.

He was in a state of extreme anxiety, and believed he was threatened with apoplexy. Some physicians thought that there was incipient softening of the brain.

My opinion was that it was a case of stomachic vertigo. The treatment which I instituted consisted in administering alkalies and bitters: in a fortnight, the symptoms had entirely ceased. Some weeks later, they returned; but on a repetition of the treatment, they again disappeared.

In cases in which the vertigo is accompanied or has been preceded by gastric derangement, you will often be justified in instituting the treatment for stomachic vertigo; and it will nearly always be successful. However, you must never omit seeking to discover whether the vertigo be not the result of sympathy with some lesion of the liver, kidneys, bladder, or uterus. As you are aware, hepatic and nephritic colics, as well as uterine pains, are often accompanied by nausea, vomiting, and giddiness.

There is another kind of vertigo which greatly resembles stomachic vertigo, viz., that usually consequent upon lesion of the internal ear, and which may be termed *vertigo ab aure læsa*. When speaking of apoplectic cerebral congestion, I mentioned the able communication on this subject which Ménière laid before the Academy of Medicine.

The connection between vertiginous symptoms and diseases of the internal ear were pointed out, in 1863, by Triquet in his clinical lectures;* and in a manuscript note communicated to me, he attaches special importance to the vertigo and buzzing in the ear, which occur in cases of inflammation of the labyrinth. He claims for Saissy (de Lyon) the credit of having been the first to point out the coexistence of vertigo with diseases of the ear. I cannot indorse this claim, for in the two cases given in Saissy's work, which appeared in 1827,† cases quoted from the work of Dr. Viricel, he speaks of violent pains depending on lesions of the tympanum, demonstrated by necroscopic examination; but neither in the account given of the cases, nor in Saissy's remarks upon them, is there any mention of vertigo.

It is to Ménière, therefore, that we are indebted for our knowledge of the relation which exists between lesions of the labyrinth and the cerebral symptoms which all physicians prior to him had attributed to the stomach, to apoplectic congestion, or to the commencement of very serious affections of the brain. In 1861, Ménière‡ showed that patients affected with inflammation of the labyrinth present an assemblage of symptoms reputed cerebral, such as vertigo, dizziness, unsteady gait, turning round, and falling—symptoms which are accompanied by nausea, vomiting, and sometimes by syncope.

He became convinced by the frequent observation of cases of this description, that all the symptoms (so far from following the usual course of cerebral affections or stomachic vertigo), disappeared after a period variable in duration, leaving in their place deafness rebellious to treatment, and generally incurable.

Ménière, in the work to which I have alluded, mentions the case of a young woman, who, when menstruating, travelled on a winter night on the outside of a stage-coach, and experienced, as a consequence of this exposure to great cold, *a sudden attack of complete deafness.* When received into Chomel's wards, her principal symptom was constant vertigo: vomiting was induced by the slightest attempt to move; and she died on the fifth day. At the autopsy it was found that the brain, cerebellum, and spinal cord, were free from morbid change. As the patient had all at once become deaf, having had her hearing perfectly good up to the date of the attack, Ménière removed the temporal bones, so that he might be able carefully to examine into the cause of the complete deafness which had supervened so rapidly. The semicircular canals were the only parts of the labyrinth which presented any abnormal appearance: they contained a reddish-colored plastic lymph, instead of the fluid of Cotugno.

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* TRIQUET: Leçons Cliniques sur les Maladies de l'Oreille, p. 113. Paris, 1863.

† SAISSY: Essai sur les Maladies de l'Oreille Interne. Lyon, 1827.

‡ MÉNIERE: Bulletin de l'Académie de Médecine, vol. xxvi, p. 241.

In the cases of Saissy and Triquet, there was found, on necroscopic examination, a similar exudation of a reddish plastic matter, and also thickening of the nervous membrane which lines the semicircular canals. There is, therefore, ground for supposing, that possibly, had attention been called to the question, there might have been observed during life, symptoms similar to those observed in Ménière's case.

No doubt, gentlemen, many of you remember the woman, who lay in bed 25 of St. Bernard Ward. She was almost absolutely deaf: whenever she was interrogated in a voice somewhat too loud, her countenance indicated acute suffering, and she complained of great pain in the head, and insupportable noises in the ears, while she was also at the same time seized with vertigo, and held her head between her hands, as if for the purpose of shutting out all external noise. Everything about her, she said, seemed to be turning round. If she were addressed, when standing, in too loud a voice, she would lay hold of the bars of her bed to save herself from falling. She told us, that for a long period, without any appreciable cause, she had suffered from vertigo, which, day by day, was increasing, and had reached such a degree, that she could no longer walk alone in the streets, the noise of the carriages being insupportable, and bringing on the giddiness. She also mentioned that she often felt herself pushed from *left to right;* and that when walking on the pavement, she was always careful to take the right, being afraid of falling upon the causeway. Bear in mind, gentlemen, that the deafness was greatest on the right side, and that it was on the same side that noise produced a painful impression. The patient frequently had nausea and loss of appetite, although she was not feverish, and had a tongue which did not indicate indigestion. There was no appreciable loss of flesh: she never had had anything wrong in respect of the hepatic and renal secretions, nor the menstrual flux. The cause of the vertiginous symptoms was an affection of the auditory nerve: the nearly constant buzzing in the ear, the almost complete deafness, the exacerbation of the buzzing and pain whenever a noise was made near the patient, are facts corroborative of this opinion. On examining the external auditory canal, the membrane of the tympanum was seen to be depressed towards the centre, and to present at that point a sinking down which Triquet attributes to the welding of the ossicula; but this depression of the tympanum, which indicated former inflammation of the middle ear, existed only on the left side, and established a contiguity of pain between the membrana tympani and the fenestra ovalis.

There never occurred in this woman loss of consciousness, convulsions, nor paralysis, and her intellectual faculties remained intact: it was, therefore, scarcely possible to be satisfied with the hypothesis that there was a lesion of the cerebrum or cerebellum: her sight was good, and she never had strabismus. But on the one hand, on considering the experiments of Flourens, Brown-Séquard, and Vulpian in connection with conclusions arrived at by Ménière, and on the other hand, considering the symptoms of our patient, viz., the deafness, buzzing in the ear, impulse to turn to the right, and vertigo, it was natural to suppose that the semicircular canals were the seat of a morbid change accounting for all the symptoms we noted. Again, the lesion of the labyrinth, though existing on both sides, was most marked on the right side, the pain was most intense on that side, and the impulse to turn was from *left to right.*

Gentlemen, I cannot upon this occasion set before you in detail the result of each individual experiment of Flourens,[*] results completely confirmed by the researches of Brown-Séquard and Vulpian: I shall only say, that it is now generally admitted by physiologists, that the simultaneous lesion of the semicircular canals of both sides gives rise to forward movement, or backward movement (a tumble head-over-heels), according to the situation of the injured canals; and that when there is lesion on one side only, the propulsion is always to that side. Here, too, pathological anatomy confirms the results of experimental physiology. In 1861, MM. Signol and Vulpian communicated to the Société de Biologie[†] the case of a cock which in fighting received a violent stroke on the head from the beak of another cock. The animal was at once stunned, but was soon seen, with head drooping forwards, to turn himself *from left to right*, when attempting to walk. He became blind. When he died, six weeks after receiving the wound, a great part of the right temporal bone was found to be necrosed: the whole of the portion of the bone containing the semicircular canals was isolated by a newly formed membrane, and it was impossible to discover any trace of the semicircular canals on the right side.

If there really existed in our patient, as I suppose, a lesion of the semicircular canals, the propulsion from left to right would be explained by the morbid change in the canals being greatest on the right side. As for the other symptoms—the vertigo, pain in the head, and nausea—they are sufficiently explained by recollecting that any violent shock imparted to the membrana tympani by a probe or an injection is sufficient to produce buzzing in the ear, vertigo, and nausea. In these cases, the shock is probably transmitted to the fenestra ovalis by the chain of ossicula, and thence to the internal ear, the lesions of which produce the giddiness, as is proved by the case of the young woman reported in Ménière's memoir.

I believe, then, that there was lesion of the semicircular canals in our patient: but if physiology, combined with the clinical and necroscopic study of the cases reported by Ménière, justify our adopting this conclusion in respect of the seat of the lesion, the cause of the lesion remains unexplained. Saissy and Triquet attribute a large share in the etiology to the rheumatic diathesis, and to catarrhal constitutions of the atmosphere: we have seen in the case described by Ménière, how much may depend on exposure to cold and the suppression of the catamenia. In our case, we cannot attribute anything to these causes: because the existence of the rheumatic diathesis was not ascertained, and the patient had not had suppression of the menses.

In conjunction with the case which I have now recapitulated, it will be well to lay two others before you: one of them is detailed by Dr. Burrgraeve,[‡] and the other by Dr. Hillairet.[§] In Dr. Burrgraeve's case, there supervened, consequent upon a chill, inflammation of the internal ear, with perforation of the tympanum and discharge of sanguinolent pus from the auditory canal. Upon the sudden suppression of the discharge, the gait became unsteady, the patient being frequently unable to maintain his equilibrium. He soon experienced vertigo, and was obliged to remain close to his bed, to prevent himself from obeying the tendency to turn round. His head was violently drawn from right to left, and from left to right: he felt it impossible to walk, from a feeling that the floor was not firm footing: he felt as if on the deck of a ship rolling in a heavy sea: he experienced retching and vomiting, having in fact veritable sea-sickness. To quote the patient's own words, as reported by Dr. Burrgraeve: "When the position of my head was unsettled by my turning it quickly, or by blowing my nose, my legs gave way, and I fell as if struck down by a thunderbolt." It is a circumstance worthy of attention in this case, that the superior extremities did not participate in the incoördination of the inferior, and that they retained precision of movement during the whole course of the disease. The head was perfectly free: there was nothing abnormal in the sight, smell, or taste: nor was there anything morbid in the sense of hearing, except a disagreeable buzzing and singing in the affected ear. The author states that these symptoms in a great measure ceased when the discharge reappeared; but for more than a month afterwards, there remained a certain amount of indecision in the movements.

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[*] FLOURENS: Recherches Expérimentales sur les Propriétés et les Fonctions du Système Nerveux. 2de edition (1842), p. 442 et seq.

[†] J. SIGNOL et A. VULPIAN: Note sur un Cas de Nécrose d'une portion du Diploé Cranien chez un Coq. Altération profonde de l'Appareil Auditif: Phénomènes Symptomatiques semblables à ceux que produit la Section des Canaux Semicirculaires. [*Comptes Rendus des Seancés de la Société de Biologie*, 1861, p. 135. Paris, 1862]

[‡] BURRGRAEVE: Gazette Médicale de Paris, 1842, quoted from Annales et Bulletin de la Société de Médecine de Gand, 1841.

[§] HILLAIRET: Lésions de l'Oreille Interne: Action Réflexe sur le Cervelet et les Pédoncles. [*Comptes Rendus et Mémoires de la Société de Biologie.* Third Series, vol iii, p. 148, for the year 1861. Paris, 1862.]

It is evident that all the symptoms which Ménière has referred to lesions of the labyrinth were present in this case,—the vertigo with buzzing in the ear, the nausea, the vomiting, the irresistible tendency to turn round in a determinate sense, the characteristic symptoms, as I have said, of lesions of the semicircular canals.

The case communicated to the Société de Biologie by Dr. Hillairet is not less interesting than that described by Dr. Burrgraeve. After a chill, which occasioned acute and long-continued pain in both ears, there supervened a purulent discharge from the right ear. From the time at which this running began, the attacks of pain became less violent, and much less frequent. But the chronic inflammation within the petrous portion of the temporal bone soon manifested itself by numerous fleshy granulations appearing in the external auditory canal in the form of polypus. Subsequently, this polypus, by preventing the pus from escaping, caused the pains to return with their original characters: and in addition, there were buzzing in the affected ear, vertigo, a tendency to vomit, fidgets, feebleness of the inferior extremities, a disposition to stoop, and to turn to the side opposite to that which was the seat of the lesion. Removal of the polypus relieved the patient by giving easy exit to the purulent matter. By and by, the discharge gradually dried up under the influence of local and general treatment, whereupon there was an end of the nervous phenomena.

Dr. Hillairet without hesitation connected all these nervous phenomena observed in his patient with a lesion of the semicircular canals: he then, from physiological experiments and from the case so well analyzed by MM. Vulpian and Signol, came to the conclusion that the nervous phenomena were probably the result of lesion of the internal ear.

Grant that all these facts are well established, we shall still have to explain how lesions of the semicircular canals produce disturbance of the brain, that is to say, such symptoms as vertigo, titubation, an irresistible tendency to fall, or to turn round in a determinate direction. We know that all these phenomena may occur when there is lesion of certain parts of the encephalon: but in the cases now under analysis, we must reject the hypothesis of a lesion propagated to the brain by contiguity of the internal ear, inasmuch as the integrity of the cerebral substance is established by the results of clinical study, and by the autopsies made by Ménière, Viricel, and Vulpian.

You know, gentlemen, the important part now attributed to reflex action in the production of physiological and morbid occurrences: you know that the spinal marrow incited by the sensitive nerve of animal or organic life is a centre of manifold reflex actions: the brain also participates in this action whenever a cranial nerve of general or special sensation is excited in a particular way. Irritation of the ophthalmic branch, without the individual being aware of it, is reflected in a special manner by the brain, and as consequences of this reflection there occur a flow of tears and injection of the conjunctiva: in the same way, when the retina is excited, there is observed, in virtue of similar reflex action, winking of the eyelids, epiphora, and contraction of the pupils. You are likewise aware of the fact, that the operation for cataract by depression induces vertigo and retching.

Analogous phenomena occur in acute earache. I could multiply examples of this reflex action of the brain. But for the present, let it suffice to remark, that Brown-Séquard in his lectures on the nervous system* has announced as his conclusion, that irritation of the auditory nerve, of the optic nerve, and of every sensitive nerve, may, by reflex action, produce convulsions, vertigo, and other symptoms of encephalic disturbance. And, as reflex action may produce its results upon the vaso-motory nervous system, as well as on the nerves of motion and sensation, it is a legitimate supposition that in lesion of the semicircular canals, reflex action influences the *nervous system of the brain*, in such a way as to produce cerebral anæmia and consequently many of the symptoms of that form of anæmia, such as giddiness, nausea, and a tendency to syncope.

This last remark brings me to enunciate the hypothesis that stomachic vertigo is perhaps simply the result of a reflex action upon the cerebral circulation, which reflex action has the seat of its incitation in the stomach.

However we may interpret the vertiginous phenomena connected with lesion of the labyrinth or with stomachic derangement, their existence is now quite recognized: you may always refer them to an affection of the organ of hearing, whenever they are accompanied by continuous buzzing in the ear, and followed by deafness.

Gentlemen, it very often happens that stomachic vertigo, particularly when it occurs in the aged, or in those who are verging upon old age, is a prelude to very serious cerebral lesions, such as apoplexy and softening of the brain. Although in the great majority of cases, the vertiginous symptoms which I have described to you leave only a fleeting impression on the brain, and seem to have their origin in the organs of digestion; and although I do not hesitate to employ the treatment which I am going to point out, and which cures the patients—some temporarily (as in the greater number of cases), and others permanently, I am not the less reserved in my statements, knowing that in a few exceptional cases, a simple vertigo is followed by formidable cerebral symptoms too evidently due to very serious encephalic lesion.

To stomachic vertigo assuredly belong the cerebral phenomena which accompany indigestion, gastric discomfort, a simple state of plenitude of the organ, such as occurs after a meal more than usually ample, or after eating some particular articles of food.

These vertiginous symptoms, accompanied by a feeling of weight and dull headache, by ringing and buzzing sounds in the ear, bear a much nearer resemblance than the preceding to the forms of vertigo regarded as depending on cerebral congestion. Even when they are the sole indications of discomfort in the stomach, the circumstances under which they arise prevent a mistake as to their nature. They are phenomena as transient as the causes which produce them, and they yield with the greatest ease. It is not, indeed, necessary to enlarge on this point, any more than upon the proper treatment.

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* BROWN-SÉQUARD: Lectures on the Physiology and Pathology of the Central Nervous System, p. 195 et seq. London, 1860.

It is otherwise, however, with the symptoms to which we are now giving our special attention. The ingestion of a small quantity of aliment, of broth, of a little generous wine, or particularly of alcoholic and aromatic liquors, will frequently calm them: but to prevent their return, to cure the disease, it is necessary to institute, and perseveringly follow up, a treatment directed against a pathological condition more or less of old standing, and more or less persistent.

There is a plan of treatment which has rendered me great services: I owe my knowledge of it to Bretonneau, my venerated master. You have seen me employ it in our cases, so that you know that it consists in administering bitters and alkalies. The following is the programme.

Every morning, the patient takes an infusion of two grammes of shavings of the wood of quassia amara. The shavings are macerated for twelve hours in a teacupful of cold water. A similar dose may be obtained by allowing the same quantity of water to remain in a goblet made of this bitter wood.

After each of the principal repasts, as well as on going to bed for the night, the patient takes a powder, for which the following is the prescription:

Bicarbonate of soda, ...	1 gramme.
Prepared chalk, ...	2 grammes.
Magnesia, ...	1 gramme.

Mix, and divide into three packets, which are to be stirred in half a tumbler of sugared water just before being taken. These alkaline powders ought to be taken for five or six consecutive days, then discontinued, to be resumed after eight or ten days. During the intermission of the powders, the patient ought to drink such natural mineral waters as those of Vichy, Pougues, Vals, or Ems, which seem to act chiefly in virtue of their alkaline ingredients; more than two tumblers for a daily dose must not be taken: or, recourse may be had to the waters of Bussang, Spa, or Schwalbach, which also partly owe their medicinal action to the small quantities of alkalies which they contain, but principally to their ferruginous ingredients.

When the appetite is languid it will be useful to give preparations of the strychnia family of plants to rouse the sensibility of the mucous membrane of the stomach, and the contractility of its muscular coat. These medicines likewise act as excellent tonics. I often prescribe from one to four of the bitter drops of Baumé to be taken at the commencement of meals; or, I order from five to ten drops of the tincture of nux vomica; or the extract of nux vomica in pills, each containing five centigrammes of the extract.

Beyond every other measure, it is essential to insist on a tonic and substantial regimen, assisted by moderate exercise. In a word, the treatment of stomachic vertigo is the treatment of dyspepsia, a subject into which I shall have to enter very fully upon another occasion.

LECTURE LXVIII.

DYSPEPSIA.

Dyspepsia is not so much a Disease as a Phenomenon common to many Diseases.—In the cases in which, from its predominance, it seems to constitute a Morbid Species, it is subordinate to numerous different conditions.—General Considerations upon Aptitudes of the Organism, and Manner in which particular Organs accommodate themselves to the Stimulants which act upon them.—Application of this fact to the question of Dyspepsia.—Dyspepsia, the consequence of Increased Excitation of the Gastric Secretions and Muscular Movements of the Stomach.—Reflections upon that Neurosis which I have called "exhaustion of incitability" [épuisement de l'incitabilité].—Asthenia consecutive upon very prolonged excitation.—Dyspepsia the result of Sympathy with Diseases of the Liver, Stomach, Intestines, and other organs.

GENTLEMEN: We nearly always have some dyspeptic patients in the clinical wards. You sometimes see me prescribe alkalies, and at other times acids, to relieve the symptoms from which these persons are suffering. There are also cases in which I order preparations of cinchona, quassia, or strychnine; and there are others, in which I order opium, belladonna, and antispasmodics. In fact, I vary my treatment in an infinity of ways.

The reason of my thus acting, as if I had no fixed rules to guide me, really arises from there being nothing determinate in dyspepsia itself. In it, more than in any other morbid condition, the physician, free to act on the suggestions of the moment as they arise, is forced to feel his way as he proceeds, inquiring into the indications, which vary with the case, with the person, and which may also differ in the same person at different times. There is nothing surprising in this, when it is remembered that *difficulty of digestion*—for that is the etymological meaning of the word *dyspepsia*, derived from the Greek word *δυσπεψία*—is a symptom common to a host of acute and chronic diseases; and that it is subordinate to morbid conditions differing very much from one another, even when it is so predominant a feature as to seem to be entitled to be regarded as a pathological species.

I take up this subject to-day, without the least intention of attempting to give you a complete account of dyspepsia. I only propose to enter into some general considerations, to lay before you some of the cases which I have seen, and to formulate some of the indications which most frequently present themselves at the bedside. I shall not shrink from going into details, which, in a subject so vast and so obscure, appear to me to be of greater practical utility than a dogmatic description, however elaborate.

A short preliminary excursion into the domain of normal physiology is requisite, to enable you to understand the facts which I shall have to lay before you.

There are three things which have to be considered in the performance of every function, as has been said by Professor Récamier, one of my most illustrious predecessors in this chair; viz., the *stimulus*, the *support of the* *stimulus*, and that which he called *reciprocal capacity* [*capacité réciproque*]. This last expression is not, perhaps, very clear; and I shall, therefore, substitute for it *functional relation* [*relation fonctionelle*], which is more intelligible. By "support of the stimulus," Récamier understood the organ in its totality with its anatomical and physiological accessories, the functional apparatus which ought to be in communication with its physiological excitant, its stimulus, the excitant of the stomach, which is all that brings the support into operation. Aliment is the stimulus, the excitant of the stomach which is the support of the stimulus: and light is the normal excitant of the eye. The "reciprocal capacity," which I propose to call the "functional relation," is the mutual bearing on each other of the support of the stimulus and of the stimulus itself; and it is from this mutual bearing that there results the normal performance of the function.

Having made good this position, let us endeavor to study the different modifications which may take place in the support, and in the stimulus; let us see what are the results and modifications in relation to the function.

Suppose, for a moment, that the support of the stimulus is normal, and the organ healthy; and suppose, at the same time, that the stimulus is abnormal, it is evident that there will be disturbance of the function. Take the eye, for example, and apply to it light differing in quality and in quantity from that which it habitually supports, and you will produce functional disturbance of vision. Or make your experiment on the stomach—give it aliment of abnormal quantity or quality—and you will induce a perturbation of its functions.

Suppose, on the contrary, that there is an abnormal disposition of the normal stimulant, and also of the support of the stimulant. In the case of the eye, suppose the light, sufficient in quantity and normal in quality, acting upon the morbid organ in one or another mode: in the case of the stomach, suppose food to be given normal in quantity and quality, but that the organ from some cause or another is not in a proper state to receive it, and the functional relation no longer existing, the physiological action of the organs will be—as in the first hypothesis—necessarily disturbed.

It may happen, however, that although both the stimulus and its support are in an abnormal state, the functional relation may remain in a regular state up to a certain point. This is what I have called the accidental or fortuitous functional relation [*relation fonctionelle accidentelle, fortuite*] in diseases. For example, it may happen that the eye may be to a certain extent in a morbid state, and that the light may likewise reach it in a form and in quantity which are not normal: under such circumstances, the light becomes adapted [*coadaptée*], if I may use the expression, to the morbid state of the eye, and thus vision is accomplished. Let us apply this illustration to the stomach. If we administer to the stomach when in an abnormal state, an aliment which is to a certain extent abnormal in respect of quantity and quality, the functional relation will be established accidentally and fortuitously. Digestion will be performed in a nearly normal manner, although neither the stimulus nor its support are in a quite regular state. In virtue of this accidental functional relation, some empirical methods of treatment prove successful in certain diseases of the stomach in which the food administered to the morbid organ is perfectly well borne by it, which would not have been the case had the organ been in its normal physiological condition.

Man is unquestionably the animal capable of becoming most easily adapted to diversities in the external circumstances necessary for maintaining life. The first individual of our species did not assuredly come into existence in lat. 50°: his body not being protected by hair or feathers, like other animals of the higher classes, proves that the Creator called him into being in a climate sufficiently mild to enable him to dispense with clothes, which in our climate are absolute necessities. However, the territory of man's origin becoming overcrowded, emigration took place to other regions. Covering the surface of the globe from north to south, and from east to west, everywhere adapting itself to new climatological conditions, the human race became able at last to live as well in polar as in equatorial regions. This adaptation, however, to the greatest possible diversity of climate is perhaps less remarkable than the adaptation of man to great diversity of food. Advancing from the simplest possible regimen, consisting principally of slender rations of vegetables such as rice, with water and a little milk for drink, a regimen similar to that which the Indians and other peoples still subsist on, man has reached that generous fare of northern nations which contains so large a proportion of animal food. His organization has become habituated to conditions totally different from those in which he was originally placed: it has well adapted itself to them, and has by means of the new regimen, made the man of the north, a much more vigorous man than the man of the equator.

This wonderful faculty of adaptation to circumstances, belongs not only to individuals of the same species taken separately, but also to different organs of the economy.

The support of the stimulus, the organ remaining in a normal condition, becomes at last adapted to the action of an abnormal stimulus. It is true that, at first, the result is a certain amount of morbid action; but in virtue of the aptitude to accommodate itself to a change of circumstances, the economy is modified and rearranged in harmony with this new impression; and after some time, the organs getting into tune with the new stimulus, the functional relation is established with regularity.

Under certain circumstances, the physician, assisting nature, may contribute to place persons in favorable, accommodating conditions: he may do this by means of employing pharmaceutical and physiological alteratives: he may be able to establish, for a longer or shorter period, accidental functional relations. He may be able to accomplish for particular organs, the same that he can effect for the entire economy. For, example, in respect of the stomach, the organ with which at present we are more particularly engaged, it may be placed by the physician in the conditions specially required to regulate the acts which it is destined to fulfil.

Let me now deal with my subject in a more direct manner. In relation to the stomach considered as a support of the stimulus, we must take into account its anatomical structure, or in other words, its muscular and mucous coats, its glandular, circulatory, and nervous systems: we must also consider its movements and its secretions: we shall then see what are the modifications, organic and functional, which produce that condition which we call *dyspepsia*.

First, then, gentlemen, in what manner, and under what influences are the secretions of the stomach modified? They are modified by excess or insufficiency of a stimulus.

In an animal into whose stomach a fistulous opening has been formed, a great quantity of gastric juice can be caused to flow by merely exciting the mucous membrane, by introducing a glass tube into the stomach through the artificial opening. By this excitation, there is produced a secretion which in respect of quantity is extra-physiological, but, in respect of quality, is quite normal. Should the excitation, however, be increased beyond a certain degree, it becomes inflammation, the secretion of gastric juice ceases, and from the fistula there flows only mucus.

Similar disturbances occur apart from any mechanical excitation. Under the influence of fever, which is perhaps nothing more than a great modification of the functions of innervation of organic life, the secretion of gastric juice is disturbed and arrested. This experiment has been made not merely once, but many times, by Professor Claude Bernard.* By producing fever at pleasure, in animals, in which he studied the phenomenon, he was able to suspend the secretion of the gastric juice, although there was no inflammation of the mucous membrane of the stomach—although, consequently, there was no trace of gastritis to explain the occurrence.

And, gentlemen, observe, that what takes place in respect of the stomach is not at all different from what we see elsewhere every day. When the first phenomena of inflammation have disappeared after the occurrence of a traumatic lesion, and the condition is progressing to resolution, we may regard every cell of adventitious tissue as a little stomach into which the arteries pour the food, and from which the veins and lymphatics carry away the residuum, after the functional plastic exudation has taken place. In the particular case, the functional exudation, accidentally normal, consists of plastic lymph and pus. Should fever light up, we see the secretion from the cellular tissue become modified, and the tendency to consolidation become arrested—the semi-cicatrized wounds reopen, and secrete an ill-conditioned ichor, as different from plastic lymph and normal pus, as the mucus of the stomach is different from gastric juice.

You know, gentlemen—and it is again to Claude Bernard that you are indebted for the information—that section of the pneumogastric nerves causes an immediate suspension of the movements of the stomach, and a diminution in the secretion of gastric juice. You know also, that when, in our experiments on animals, we irritate the ganglia of the great sympathetic, which send nervous filaments to the stomach, energetic contractions of the stomach are produced, and the gastric secretion becomes more abundant. We thus obtain a demonstration of the changes which take place in the stomach when the cerebro-spinal and sympathetic systems are acted upon.

To come closer to a class of facts which are more clinical in their bearing, I ask: Who has not seen the influence on digestion of deranged innervation? Who does not know that great mental emotion suspends digestion, and induces indigestion? Who does not know that prolonged anxiety produces a very injurious influence on the digestive organs, and is a frequent cause of dyspepsia?

Local pains, neuralgia of the stomach and intestines, likewise disturb the secretions of the digestive organs. The same takes place in respect of them, as in respect of other organs. Neuralgia of the eye brings on a more or less violent congestion of the parts, raises their temperature, and increases the secretion of tears: in the same way, neuralgia of the stomach produces analogous effects upon that organ, augmenting its secretions to such a degree that they are poured forth, not only when food is ingested, but, also, irrespective altogether of digestion.

These are some of the effects consequent upon augmented normal excitation; and we shall now see what the results are of this same augmented excitation when it has been too long continued. I have often spoken to you of the effects produced on the economy by the abuse of excitants: I have told you that, if an organ is subjected to excitation repeated too often, or pushed too far, it ceases to respond to its stimulus, and that that state is soon induced which Brown called "asthenia." What was his explanation of asthenia? Let me now repeat what I have so often impressed upon you on other occasions; and I make this repetition without any scruple, for it is most important that you should have a correct understanding of these views, the only opinions, perhaps, really judicious and practical which are embraced in the great theory of the Scottish physician.

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* CLAUDE BERNARD: Leçons sur la Physiologie et la Pathologie du Système Nerveux. Second vol., p. 374. Paris, 1858.

Brown, convinced that life was maintained solely by excitants (an opinion nearly the same as that afterwards taught by Broussais), thought that every organ was endowed with a peculiar capacity for excitation, to which he gave the name of "excitability;" and he believed that this excitability was exhausted by being merely brought into exercise. He said, for example, that the brain, the spinal marrow, and the muscles, have an aptitude to enter into simultaneous action, to execute the function of locomotion. Now, if the excitation exercised by the mind upon the muscles through the medium of the spinal nervous system, which it commands, is exercised for too long a time, the nervous system and muscular apparatus will at last cease to respond to the cerebral excitation, whereupon they will lose their capacity of being excited—their excitability—and will fall into a state of *asthenia*, which may here be interpreted *powerlessness*. According to Brown, it was only by means of repose that the muscles and the nervous system could regain their lost capacity for excitability. But if excitation is continuously carried beyond its normal limits, the excitability exhausts itself in a proportion greater than that which can be restored by repose; so that the being habitually excited will deprive the organs of the power of bringing into play the normal stimulus to which they formerly responded, and will cause them to require a more powerful stimulus. Let me give you an example: The eye accustoms itself to support light in certain proportions, regular in respect of quantity and quality. Suppose that we represent the quantity by the figure 10, and assume that the visual apparatus is in a normal physiological state: let us assume that this quantity of light (the eye remaining in its previous condition) is suddenly increased to 20, the result will be the production of that peculiar abnormal phenomenon called *dazzling*. It is not asthenia; for if only 10 degrees of light are again afforded to the eye, it will regain the regularity of its functions which were temporarily disturbed. But if, in place of quickly and temporarily augmenting the stimulus, it is gradually augmented—if, day by day, the eye is accustomed to a stronger and stronger light—the time will come when the visual functions will be performed under the influence of that strong light, exactly as they were originally performed with a much feebler light: moreover, the time will come when vision will be impossible unless the eye receive an amount of stimulus in excess of the originally required quantity. In an individual, therefore, who has been accustomed for six months or a year to 20 degrees of light, and all at once receives only 10, the excitation of the retina produced by this diminished quantity of stimulus will no longer be sufficient to bring the visual functions into play. Under the influence of an excitation greater in amount and constantly repeated, the excitability of the organ is exhausted, and asthenia is produced: the result is inability of the eye to perform its function, unless it have a quantity of stimulus twice as great as that which originally sufficed.

Similar results follow similar causes in the stomach. An individual, for example, lives on plain fare, partakes very sparingly of spiced dishes, abstains from condiments, and drinks alcoholic liquors in small quantity: the stimulus and its organic support—that is to say, the food and the stomach—are in a state of functional relation which is perfectly sufficient and normal, so that digestion is performed with regularity and ease. But the individual, little by little, increases the quantity of spice taken with his food; day by day, he uses a little more alcoholic drink; and, in a word, he takes more excitants. He may, on the first day, have suffered from this change of regimen, but he soon becomes accustomed to it, and in proportion to the gradual augmentation of the stimulus, there is, on the part of the stomach, a gradual adaptation to the new impressions. What would take place if this individual were abruptly to resume his former style of feeding? The stomach being imperfectly excited, would not yield its necessary secretions, and difficult digestion would be the consequence. To combat this dyspepsia, you would then be obliged, either to act in accordance with Brown's recommendation, and keep the organ at rest for a certain time, to enable it to regain its original excitability, lost through abuse of excitation, or, else, be forced to resume the excitants to which the stomach had become habituated, and even to have recourse to other excitants more and more energetic.

In the remarks which I have now been making, I have only had in view what takes place in respect of the secretions of the stomach; but it is also very necessary to take greatly into account the muscular apparatus of the stomach, which is as essential as the secretory apparatus, for the performance of the function of the organ. Unless the movements of the stomach and intestines take place with perfect regularity, digestion cannot be accomplished in a normal manner. Now, there are different ways in which the gastro-intestinal muscular system may be disturbed. Its excitability may be diminished, enfeebled, when the individual will digest badly, because (if I may so express myself) there is an arrest in the contractions of the stomach: on the other hand, the excitability may be increased, when the individual will no longer digest rightly, because the contractions of the stomach are too frequent and too energetic.

In the latter case, the alimentary substances will be propelled quickly from the stomach into the duodenum, where they will arrive imperfectly chymified or not chymified at all. Being insufficiently prepared for the new process to which they are submitted in the first portion of the intestine, their digestion will go on badly, and dyspepsia will be the result.

Just as we have seen in respect of the gastric secretions, the increased contractility of the stomach may depend upon the supervention of a disturbance of the nervous system—of the cerebro-spinal system, consequent, for example, upon mental emotion—or of the ganglionic system. Likewise, also, the cause may be the abuse of excitants taken into the stomach, they acting more or less directly upon its contractile apparatus.

In the same way, also, that increased and long-continued excitation of the secretory system leads to asthenia of that system, so does long-continued augmented contractility produce asthenia of the muscular apparatus. This asthenia has also, however, other causes, and one of them, which is not unusual, is taking food in too large quantities, so as to cause abnormal distension of the stomach. There then occurs in respect of the stomach, what occurs in respect of the bladder when, after prolonged distension from retention of urine, it becomes paralyzed, in consequence of forcible distension having annihilated the muscular tonicity. The same thing takes place in the other hollow organs.

As I have been saying, this is not an unusual cause of asthenia of the stomach. It is observed in great eaters, like those whose histories you will find in books, who devour from sixty to eighty pounds of food in twenty-four hours. In these persons, the stomach becomes distended to such a degree as to assume the capacity of the rumen of an ox. You can understand that the organ, from being thus distended, will lose its muscular tonicity, and that after a certain time, to revive it, it will be necessary to have recourse to the use of artificial excitants, the energy of which will have to be increased in proportion to the deficiency of contractility, to the asthenia, which is progressing day by day.

This muscular asthenia of the stomach, provided the exhaustion of excitability depend on that species of paralysis which is produced by forcible distension of the organ, like secretory asthenia, is the immediate cause of the dyspepsia to which great drinkers and large eaters are subject. We shall see how such patients have to be treated: we shall see that they require particular treatment, which is much more efficacious in this than in other kinds of dyspepsia.

There is another form of asthenia which has strange characteristics of its own: it is observed in the muscular apparatus of animal life, and probably has its analogue in the muscular system of organic life. This singular affection, to which I have long directed my attention, has certainly been seen by a very large number of practitioners, and yet it has never been studied in relation to its special character.

The name which I have given to this neurosis is "exhaustion of incitability" [*épuisement de l'incitabilité*]. Let me give you an illustrative case.

At Tours, ten years ago, I saw a young, newly-married lady, who had nothing the matter with her health, except the strange nervous affection of which I am now going to try to sketch the picture. She described herself as being paralyzed: but upon examination, it was found that the powers of motion and sensation were intact. When the patient was asked to rise and walk, she did so in a deliberate manner, and with perfect precision and exactitude. Scarcely, however, had she proceeded fifteen paces, when she was observed to walk with less confidence, and after a few hesitating steps, she sunk down, and was unable to proceed another yard. I then caused her to be seated; and before a quarter of an hour had elapsed, she had regained her powers, and was once more able to walk the same short distance which she had previously accomplished. When interrogated as to the nature of her sensations, she replied, that after walking some steps, she felt such an extreme degree of fatigue, as to be unable to proceed any further: she compared the sensations which she experienced to feelings she had sometimes had when in health after a very long walk. The condition, therefore, was really not one of paralysis, but of exhausted excitability. Since the occurrence of that case, I have met with many persons presenting exactly similar symptoms. They all recovered—the majority under hydropathy and sea-bathing—some under electricity—and others under treatment by preparations of nux vomica.

Let us now return to the subject of dyspepsia.

Hitherto, gentlemen, I have only referred to what is called in scholastic language, *idiopathic* dyspepsia, that is to say, dyspepsia in which the cause is directly referable to the stomach as the seat of the disorder—dependent upon the state of its own internal organization, or dependent upon implication of that portion of the cerebro-spinal or ganglionic system which presides over its muscular movements and secretions. I have now to speak of those forms of dyspepsia which may be called *symptomatic*, those in which disorder of the function of digestion is merely the influence on the stomach of disturbance which has supervened in other organs with which the stomach has more or less intimate relations. The forms of symptomatic dyspepsia to which I allude, or these sympathies, if you prefer that expression, demand our serious consideration.

Disorders of the intestines, particularly constipation, here occupy a prominent place. It is a striking fact, that in the majority of dyspeptic patients, the bowels are moved seldom, and with difficulty. Is this the cause or the consequence of the dyspepsia? We can understand that from the mere circumstance of an individual eating little, the fæces will be less in quantity, and that in this sense, dyspepsia may be said to produce constipation; and we can also understand that the constipation may lead to disorder of the digestion. When an opportunity occurs for my addressing you on the subject of diarrhœa, I will show you that affections of the large intestine, that irritation of the very lowest part of the digestive canal suffice to excite a flux from the ileum—anal irritation producing its influence upon the small intestine. This sympathy between the large intestine and the other parts of the intestinal canal, is illustrated by the most common everyday experience: for example, indigestion will be produced by a lavement taken immediately after a meal by one unaccustomed to such a proceeding. If in place of taking a lavement, the individual introduces a suppository, a similar result is produced, or at least there pass stools, which at first are solid, being the contents of the rectum and colon, and are afterwards liquid or semi-liquid, formed by the contents of the cæcum and lower portion of the small intestine. Notwithstanding such decided effects, the suppository need not have penetrated more than four or five centimetres within the anus: but this local limited irritation is propagated much farther by sympathy. Any irritation of the lower portion of the large intestine will act in the same way, and will not only produce semi-liquid evacuations, frequent desire to go to stool, and tenesmus of the rectum from local irritation, but will likewise give rise to liquid excretions, an abnormal liquid secretion from the intestines, a diarrhœa often profuse and intractable, in consequence of the local irritation which causes it being permanent and not temporary like the suppository.

It is evident, therefore, that there exists a synergy in the different portions of the muscular apparatus of the digestive canal, in virtue of which the large intestine exercises a sympathetic influence upon the stomach and small intestine, in the same way that the stomach and small intestine may act on them; so that the regularity of the contractions in one part are dependent upon the regularity of the contractions in the other.

You can now understand how constipation may be a cause of dyspepsia. The large intestine being sluggish, that is to say, its muscular apparatus contracting badly, that of the digestive canal, and that of the stomach, likewise relax their movements, so that digestion becomes slow and difficult. There then occurs a state the opposite of that which I described in relation to diarrhœa. So true is this, that there are some patients in whom nothing more is required for the cure of the dyspeptic symptoms than to induce regular action of the bowels by rousing the muscular synergy of the intestines by administering the ascending douche or simply by giving lavements.

Here, gentlemen, we have to consider a matter of detail in the differential diagnosis. Pains in the transverse portion of the colon are often mistaken for pains in the stomach. It is no exaggeration to say, that perhaps in half the cases which are called gastralgia—particularly in old and elderly men, and in a great many young women—the affection is nothing more than colalgia. That such a mistake should be made is not remarkable, when we consider the anatomical relations of the transverse portion of the colon, which is situated in the epigastric region, contiguous to the great curvature of the stomach. The pain felt by the patient may then be attributed not only by him but also by his physician, to the stomach, in the same way that pain developed in one of the hypochondria, and having its seat in the ascending or descending colon, is often supposed to be hepatic or splenic pains, merely from the relations which these parts of the intestine have at certain points in their course, one with the liver, and the other with the spleen. Upon carefully questioning the patients, it is found, that the so-called gastric pains supervene, not during the first stage, but in the latter hours of digestion; that they are coincident with obstinate constipation, or sometimes, are followed by diarrhœa accompanied by a more or less profuse excretion of mucus, with which the fæces are covered; and that sometimes also, when the alvine evacuations have been kept waiting, or are passed with pain, they have the appearance of bands or white ribands, which have been compared to pieces of macaroni. These mucous excretions are often mistaken for fragments of tapeworm; and every practitioner has had to correct mistakes of this kind, and to reassure patients laboring under the belief that they had passed fragments of a parasite which they then were exhibiting to their medical adviser. Be that as it may, obstinate constipation may become the cause of real inflammation of the colon, accompanied by enteralgia which may be taken for gastralgia, although there is nothing wrong with the stomach. I repeat, however, that functional disorder of the large intestine very frequently leads to functional disorder of the stomach, so becoming the starting-point of dyspepsia.

Dyspepsia is a very usual epiphenomenon of disease of the liver: and it is easy to understand, that an organ which is the largest gland in the body, is immediately related to the stomach, performs a very important part in digestion, has exceedingly intimate sympathies with the other parts of the gastro-intestinal apparatus—it is easy, I say, to understand, that the disturbed action of which it is the seat reacts more or less powerfully upon the functions of the stomach. Hepatic, will also often be taken for gastric pains. We shall have occasion to revert to this fact when we come to study hepatic colic. The occurrence of errors in diagnosis are quite accounted for by what we know of the relations of contiguity between the stomach and liver.

Renal affections, or to speak more correctly, affections of the urinary apparatus, are likewise frequent causes of dyspepsia, particularly in old men. When you are consulted by elderly patients, who complain of impaired digestion, loss of appetite, gastric pains, belching, and vomiting, direct your attention to the state of the kidneys and bladder, and you will often find that the only urinary symptom complained of is habitual retention of urine.

The relation which diseases of the uterus bears to the development of dyspepsia is not less remarkable. We ought not, however, to be surprised at this, if we bear in mind the important part which the function of generation performs in the female economy, and recollect the powerful effect produced upon the whole system—particularly local and general effects on the nervous system—by the mere physiological modification of the state of the reproductive organs. These disorders of innervation, which show themselves, moreover, by an assemblage of symptoms (not necessary to be now described) suffice to explain the derangement of the digestive functions which frequently supervene. Every one knows, that in many women menstruation is accompanied by gastric disturbance. Does not this gastric disturbance, of which vomiting is one of the most marked symptoms, sometimes become very serious during pregnancy? It is not surprising, then, that pathological changes in the uterus of a more or less serious character, should act in a manner similar to the physiological changes which the organ undergoes. Dyspepsia is almost the necessary concomitant of chronic affections of the womb characterized by catarrhal discharges, by leucorrhœa, and by other local symptoms with which you are acquainted. It must not, however, gentlemen, be forgotten, that leucorrhœa is often not the cause, but the effect of the dyspepsia by which the menstrual function is disturbed and the catamenia suppressed: it must not be forgotten, that many patients attribute to the leucorrhœa by which they are tormented, the stomachic symptoms from which they suffer, while, in reality, the stomachic is the starting-point of the uterine disorder.

Among the exciting causes of dyspepsia must be reckoned diseases of the heart, nearly all of which in their latter stages are accompanied by a disturbance of the digestive functions tending to accelerate the fatal issue.

The disorders of the digestive functions so commonly associated with the different cachexiæ, are more frequently met with in patients of tubercular diathesis than in any other class. Dyspepsia not only shows itself in the last stage of pulmonary phthisis, but in some cases, it also supervenes as one of the symptoms of the incipient malady before the other signs of the thoracic disease are in any way declared. Under such circumstances, it often misleads the physician, who, although he take every means of detecting them, frequently fails to detect any material lesions, and concludes that the case is one of idiopathic dyspepsia, although, in reality, the disturbance of the digestive functions is only the indication of an organic disease which will burst forth at a given moment, exhibiting its peculiar characteristics.

There is also another class of causes of dyspepsia to which I must solicit your attention. I do not at present mean to refer to those forms of dyspepsia which are associated with the gouty and rheumatic diathesis, for I shall afterwards speak of them in a more special manner, when I lay before you the history of gout: at present, I propose only to speak of those dyspeptic affections which are dependent upon the herpetic diathesis [*diathèse dartreuse*]. The stomachic affections which are coincident with cutaneous affections, and often are, nay most frequently are, coincident with the disappearance of chronic eruptions, or alternate with them, have always been recognized by observers. Apart altogether from the humoral hypothesis, the coincidence and alternation to which I refer are explained by the synergic connection of the external and internal tegumentary membranes. As was said by Lorry: *Primarium cum cute condensum habet ventriculus.*

Gentlemen, I must enumerate to you the different kinds of causes by which dyspepsia is produced, for although they are all ultimately resolved into that increase or diminution of the activity of the gastric secretions which I have stated to be the proximate cause of dyspepsia, it is essential to establish the differences which exist between that dyspepsia which is symptomatic or sympathetic, and that which is idiopathic. In a nosological point of view this is necessary, but in respect of treatment, it is still more requisite to do this. To combat the one, we must direct our measures directly to the stomach; but to combat the other, we must first of all combat remote causes, that is to say, organic affections, diseases which have in the first instance attacked another apparatus.

Forms of Dyspepsia.—Dyspepsia associated with Chronic Gastritis.—Boulimic Dyspepsia,—Flatulent Dyspepsia.—Acid Dyspepsia.—General Disturbance of the System caused by Dyspepsia, such as Anæsthesia, partial Analgesia, Neuralgia, and Disturbance of the Intellectual Faculties.—Disturbance of the Circulation.—Anæmia.

Before entering upon the question of treatment, there is another point which we have to study. Dyspepsia, whatever its nature may be, does not always present the same characters. Let us attend to its principal forms.

One of its forms is associated with chronic gastritis. In inflammation of the stomach, its muscular fibres lose the normal regularity of their movements: and the secreting function of the organ is disturbed. This kind of dyspepsia is accompanied by loss of appetite, and a bitter taste in the mouth. The tongue is generally covered with sordes. Nausea, vomiting, and retching commonly occur. There is frequently vomiting of food, preceded or followed by vomiting of a glairy matter, usually called "phlegm" [*pituite*]. Sometimes, though rarely, the ejecta are acid. Frequently, there are corrupt-smelling eructations, tasting of hydrosulphurous acid gas, or as the patients say "tasting of rotten eggs."

It will generally be easy for you to ascertain the cause, when dyspepsia presents itself under this form. Temporary irritation, such as is occasioned by a fit of indigestion, will be the starting-point of the symptoms, which will be as transient as the affection originating them; but in other cases, when the dyspepsia has become chronic, you will find that it is dependent upon permanent irritation of the stomach, upon chronic gastritis, an affection the existence of which has been denied with far too much dogmatism in recent times.

Gentlemen, every one of you knows how leading a part has been played in pathology during the last fifty years by gastritis, acute and chronic: or at least, how great a part has been assigned to it by Broussaís and his followers. Exaggerating the import of the facts which he observed, and reverting I may say to the theories of Van Helmont, who placed in the epigastric centre the principal *archæus*,* to the empire of which he held that the whole economy is subservient—going even beyond these theories, Broussais maintained that the cause of all diseases is in the mucous membrane of the stomach. He maintained that inflammation of that mucous membrane was the source, not only of all phlegmasiæ and pyrexiæ, but likewise of nearly every morbid affection, both chronic and acute. The din of the famous controversies of that period, in which the doctrines of the Val-de-Grâce were supported and opposed, has been heard even in your time. Though the celebrated chief of the school of Physiological Medicine pushed his principles to excessive extremes, we seem, in the present day, to have fallen into opposite extremes, and, to avoid being reproached with the extravagancies for which Broussais is justly blamed, we are ready to argue that gastritis never exists.

There is a disposition to hold that the internal coat of the stomach is proof against inflammation, though no one denies that every other mucous membrane is subject to it. It is admitted that the mucous membranes of the nasal fossæ, pharynx, trachea, bronchial tubes, uterus, vagina, and even the mucous membrane of the intestine itself, are subject to inflammation; but it is alleged that the stomach is not under that liability. Is it not so in everything? The fear of falling into one particular evil, causes us to fall into another:

> In vitium ducit culpæ fuga, si caret arte,

as Horace said. In medicine, as in everything else, we hardly know how to keep ourselves within correct limits: for example, medical men, after believing that they saw gastritis in all diseases, now deny that it ever exists.

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* Basil Valentine invented the word *archæus* (French, *archée*) from αρχη, the beginning: it was afterwards adopted by Paracelsus and Van Helmont. By Van Helmont the chief *archæus* was regarded as an immaterial principle existing in the seed prior to fecundation, presiding over the development of the body, and over all organic phenomena. He placed this chief "archæus" in the upper orifice of the stomach: he said that besides it there are others subordinate to it, situated in different organs.—TRANSLATOR.

Nevertheless, it does exist. Acute gastritis is a rare affection; but it is sometimes observed, and cases as to which there can be no doubt may be cited. Chronic gastritis is frequently met with. It is true that it often remains masked; but on making a study of the patient, we soon detect, under the veil which covers it, the gastritis which causes the more or less serious disturbance of digestion.

There is another form of dyspepsia in which *bulimia* takes the place of loss of appetite. The patient has a constant feeling of emptiness in the stomach: two hours after eating, or it may be only one hour after his repast, there is a keen renewal of the appetite, which perhaps is not a real appetite, but only a craving for food. This hunger, even when it is satisfied, is accompanied by a great feeling of weakness, particularly in gastralgic women.

In this form of dyspepsia, neither eructation, flatulence, nor vomiting occur, as in that which I have just described. Constipation is one of its usual symptoms: sometimes, however, there is diarrhœa, the result of the food being too rapidly propelled from the stomach into the duodenum before there has been time for the accomplishment of the first part of the process of digestion—gastric digestion. I shall not upon this occasion repeat my previous remarks on the mechanism by which this is produced; and I shall also reserve some additional observations till I have to speak upon diarrhœa, when the details of the pathological physiology of this subject will be more in place. When we come to consider the treatment of that form of dyspepsia, we shall see that the diarrhœa may be combated by very simple measures, and that we are the more easily able to master it, the more directly we attack its cause.

Flatulent dyspepsia is characterized by the secretion in excessive quantity of the gases which are normally developed in the intestinal canal. Immediately after the ingestion of food, these gases are produced, more or less abundantly, in the stomach and intestines, which they distend; and this distension leads to such increase in the size of the abdomen as obliges the patients to loosen their clothes from inability to bear their tightness. An attempt has been made to explain this phenomenon by supposing that there takes place a rapid fermentation of the ingested feculent substances—that there is a production of carbonic acid gas, the result of a fermentation in the digestive canal exactly similar to that which occurs in the wine-maker's mashtub. Matters do not, however, proceed after that fashion. As Graves remarks, persons subject to flatulence have gas developed in the intestinal canal with almost equal rapidity, whether they eat food which can ferment or whether they confine themselves almost exclusively to animal aliment. In the latter case, one cannot say that there has been fermentation. That some gas is always produced from the alimentary mass during digestion is, however, a fact which is certain; but the principal source of the gas is secretion from the intestinal canal. A proof that this secretion is independent of the *coction* of the aliments is afforded by the fact than an hysterical woman will sometimes become tympanitic in ten minutes: under our very eyes and hands we see and feel the abdomen attain a great size. That certainly could not be explained upon the fermentation hypothesis; for I am supposing the patient to be seen before or after a meal at a time when there could be no food in the digestive canal: but, granting that alimentary substances are present in the canal, it is impossible to admit that the fermentation takes place with such rapidity. Consequently, the formation of gas is the result of disturbance of the nervous system: an increased secretion of gas takes place exactly in the same way that there is under a similar influence an increased secretion of tears, saliva, or urine. This remark is important; for if, on meeting with such cases as I have now alluded to, you reason after the manner of the chemists who regard the stomach in the same light as the glass vessels in which they experiment; if you say that there is an excess of carbonic acid gas, and, that this being the result of fermentation, your business is to put a stop to that fermentation by the means which chemistry supplies for accomplishing that end—by acting thus, you will be mistaken if you suppose that you are curing the malady: for you do not really obtain any good result. If, on the other hand, you act the part of the physician, and have recourse to baths, cold affusions, and the administration of a few drops of ether; or if you employ any other treatment the good results of which you have learned from experience, your intervention will be useful. The flatulence, which is characteristic of the form of dyspepsia of which I am now speaking, requires to be treated by remedies with which I shall make you acquainted.

There are cases in which the acids of the stomach are generated in large quantities. Almost as soon as the patients have swallowed their food, they have sour eructations; and, after meals, they sometimes bring up acid matters in greater or less abundance. So great occasionally is the acidity of these matters, that without exaggeration it may be described as setting the teeth on edge like currant or lemon-juice; and, when received in copper vessels, they cover them almost instantaneously with a green coating of lactate of copper. This you have had frequent opportunities of seeing at the bedside of the young girl of bed 27, St. Bernard Ward, who has every day been vomiting an acid fluid such as I am now speaking of. A chemical explanation of what takes place in such circumstances has not been wanting. It has been said that the glucose contained in the food, having undergone digestion in the stomach, is transformed into sugar, which sugar is transformed into alcohol: but here chemistry is again at fault, for the formation of acid products is often more abundant when the patients are fed on animal food, than when their diet consists of starchy substances. The very opposite of this, however, sometimes occurs. Nevertheless, it is sufficient in some cases similar to the first to have clear proof that the acids of the stomach are the result of a peculiar secretion, and not of a mere chemical decomposition, as some allege. Graves taught in 1828, and Berzelius repeated the lesson seven years later, that the acid secretion owes its acidity to lactic acid. The abundance of this secretion in the form of dyspepsia now under consideration is due to a peculiar excitation of the gastric mucous membrane, an excitation wholly under the influence of the nervous system, which presides over the secreting organs.

Whatever forms they may assume, the disorders of the digestive functions have an influence upon the whole economy, the effects of which, however, are chiefly seen in the nervous system, in the moral powers, and in the constitution of the blood, thus giving rise to what Beau has called the secondary symptoms of dyspepsia.* So great is this influence, according to my honorable colleague of the Hôpital de la Charité, that certain diseases are sometimes purely symptomatic of the gastric affection. It is so not only in hypochondria, which, according to many physicians, is related to dyspepsia, but also in other diseases, among which hysteria may be mentioned. Without adopting this view of the matter, which seems to me to be rather far-fetched and calculated to lead us back in some degree to the theories and doctrines of Van Helmont, I consider that it has the merit of keeping prominently in view an important element, a serious complication of these diseases, which the talented observer whom I have named erroneously regarded as caused by dyspepsia, while they are only aggravated by it.

In relation to the effect which dyspepsia produces upon the nervous system, Beau has laid great stress upon the fact that nearly all dyspeptic persons labor under nervous symptoms analogous to those from which hysterical women suffer. He says that nearly always in both classes of patients there are peculiar disorders of sensation, symptoms of analgesia and of partial anæsthesia occupying sometimes one point and sometimes another point in the skin, in the hands, arms (particularly the internal surface of the forearms), the trunk, or the face. This paralysis of sensation is sometimes so decided, that one may pinch in the most vigorous manner, prick the skin, and even transfix it with a needle, without the patient feeling what is being done. I have on many occasions repeated this experiment in your presence, so that from your own observation you can verify the statement I have now made. Sometimes patients, while they lose the sense of pain, retain tactile sensation: they distinctly feel when they are pricked, pinched, or touched: they tell you even when you prick them or pinch them, and yet they experience no pain.

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* Beau: Traité de la Dyspepsie. Paris, 1866.

Along with this analgesia and anæsthesia there are often symptoms of local neuralgia—neuralgia in the neighborhood of the parts struck with paralysis of tactile sensation.

The influence of dyspepsia upon the nervous system extends to the intellectual and moral faculties. You all know, and some of you perhaps know from sad personal experience, that difficult digestion greatly interferes with intellectual work, impedes the expression of thought, and that, when the difficulty is habitual, the disturbance of the gastric functions assumes the character of melancholy and hypochondria. You will often meet in dyspeptic persons with great mental lethargy, showing itself in an inaptitude for work, sometimes in an impossibility of forming or clearly expressing ideas. Some tell you, that their memory is gone: and many complain of pains and weight in the head, accompanied by a very distressing feeling of emptiness. It is in such cases that there supervenes *vertigo a stomacho læso*, an affection regarding which I recently addressed you in a special lecture.* After meals, the patients experience an invincible tendency to sleep, a sort of torpor, or at least an insurmountable repugnance to move: their sleep is disturbed by agitating dreams, and nightmares. Generally, the persons thus affected have an excessive degree of nervous irritability. They are melancholy, morose, exceedingly pusillanimous, and so irascible that they cannot bear to be thwarted in the slightest degree either by word or deed.

When the disease goes on for a certain time, the influence which it exerts upon the constitution of the blood is shown by symptoms to which Dr. Beau has given the name of "aglobulie." This diminution in the quantity of red globules along with an increase in the normal quantity of the serum, is characterized by phenomena which it is hardly necessary to describe in this place. The integuments have lost their color, and probably present that pale yellow hue which is met with in anæmic subjects. The patients are liable to buzzings in the ear, disturbed vision, and palpitation of the heart. On auscultation of the heart, an anæmic blowing sound is heard at the base, which is prolonged into the cervical vessels. Ultimately, there is met with the entire series of nervous symptoms which peculiarly belong to individuals whose blood is impoverished. When this impoverished condition of the blood proceeds to an extreme degree, the disorders of the circulation may occasion œdema of the extremities and anasarca, although it has been alleged that the latter is not met with in the circumstances now described. Sometimes there is even slight passive interstitial hemorrhage, spots of purpura, for example, appearing on different parts of the skin.

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* See page 355.

The emaciated condition of the patients shows the greater or less disturbance of the nutritive functions. But the disturbance of nutrition is characterized by a special sign, to which Beau was the first to call attention. It is the *ungual furrow*. This ungual furrow is a transverse groove in the nail, such as would result from a loss of substance in the external layer. This appearance admits of being remarkably well studied in the nail of the thumb: from the thickness of this nail, the furrow is more marked. The ungual furrow met with in diseases of long duration, such as serious fevers, is hollowed out more or less deeply and is more or less wide. It often happens that there are several furrows arranged in series and separated the one from the other by spaces in which the surface of the nail is uneven, rough, and sensibly less elevated than the rest, presenting sometimes a milky color, and on pressure showing an evident diminution of thickness.

The cachectic condition into which patients fall who have been long subject to dyspepsia, frequently misleads the physician and induces the belief that there exists a bad diathesis. The idea suggests itself that there is pulmonary phthisis, a supposition all the more natural, that there exists cough, the frequent accompaniment of gastric disturbance. This cough, the cough which so often accompanies gastric disturbance, is dry, coming on in isolated attacks or in urgent fits, accompanied by a very painful feeling of strangulation and angina, occurring periodically in paroxysms at certain hours, particularly in the evening. This stomach cough excites serious anxiety as to the state of the chest, which does not always upon examination dissipate the fears which have been formed: it is in some cases only after repeated examinations that it is admitted that no signs of tuberculization exist. This supposition that there is tubercle has all the more appearance of being correct, from cough, emaciation, and debility being present, frequently coexisting moreover with neuralgic pains in the walls of the chest, particularly in the back, whence they shoot into the sides. Although the absence of the tuberculous diathesis diminishes the gravity of the prognosis in these cases, it is necessary to be aware of the fact that dyspepsia in this extreme stage, and presenting all the characters to which English physicians have applied the name of "dyspeptic phthisis," is in truth a dangerous malady.

Gentlemen, if for convenience of description, and with the view of the better adapting them to their appointed places in nosological tables, we isolate from one another the different forms of the same disease, if we divide one malady into genera and species, in imitation of the methods adopted in the study of the natural sciences, it will be found that such classifications are seldom suitable in medicine. If, with a view to render our views more precise to those to whom they are addressed in lectures or in books, we are obliged to unite, to group together certain facts, in such a way as to form a more or less complete picture, we ought to recognize the fact, that all such classifications are artificial, and contain nothing positive, when tested by a comparison with the reality. In natural history, and in botany, the species have a certain number of characters, which are invariable and immutable, and which enable us to distinguish the one species from the other. This is not the case in pathology. The same disease is far from presenting immutable phenomena exclusively belonging to it: different species have characters in common, which commingle and blend with one another in such a manner, that the nosologist has often a difficulty in assigning to them a place in the classification which he has drawn up. This is particularly the case in respect of dyspepsia. Although we distinguish many species of dyspepsia by resting their distinctive characters upon the predominance of one or of several morbid phenomena which appear to characterize them, these species often blend into one another, their reputed characteristic symptoms commingling, and alternately assuming the leading place. It is necessary to make this remark; for, hearing me speak of the different forms of dyspepsia, and seeing them formulated with so much precision by certain authors, you might suppose that nothing was easier than to distinguish them from each other; and when you found yourselves alone at the bedside of the patient, you would feel yourselves peculiarly at a loss to be no longer able to recognize what appeared to you so plain and simple in the lecture-room. You would experience great embarrassment in deciding upon the appropriate treatment, and in vain would you look out for the indications which you had imagined were always to be met with. Proceeding consequently at haphazard, you would fall into serious therapeutic mistakes, mistakes which would lead you to become unbelievers in medicine. On the other hand, if you remember, that there is a possibility of this commingling of the different forms of dyspepsia, you will be enabled, when you meet with it, to adapt your treatment to the actual nature of the case, to watch its manifestations, and combat its individual symptoms by different means, in place of resorting to one uniform mode of treatment. You will have recourse to mixed methods of medication, applicable to the different symptoms which in the aggregate constitute the disease with which you have to deal. It is, gentlemen, a general rule in medicine, that, except in a few exceptional cases, when a specific disease has to be treated by a remedy which is also specific, we are obliged to attack the different elements of disease by following the indications which they severally present.

Treatment of Dyspepsia.—The most important part of the Treatment is the Regimen.—The best Regimen is that which the patient has learned by experience agrees best with him.—The Specific Character of the Phlegmasia must be taken into account.—Connection of Dyspepsia with the Herpetic Diathesis.—Remedies which produce a Local Modification of the Gastric Inflammation, such as Emetics, Purgatives, Mercurials, Subnitrate of Bismuth, Precipitated Chalk, Alkalies, Lactic Acid, and Hydrochloric Acid.—In Bulimic Dyspepsia, are given Opium and Belladonna in small doses, Zinc, and Antispasmodics.—In Acid Dyspepsia, both Acids and Alkalies available, as they do not act as Chemical Remedies; Narcotics, Mineral Waters.—In Flatulent Dyspepsia, use of Alkalies: Bitters, Quassia, &c.: Tonics, Cinchona, &c.: Aromatics: Mineral Waters, containing Chlorides of Soda: Hydrotherapy: Sea-bathing.—In Dyspepsia connected with Diseased Liver, use of Alkalies, Alkaline Mineral Waters: sometimes, Acids.—Acids particularly indicated in Dyspepsia associated with a Chronic Morbid Diathesis, particularly in fully declared Phthisis.—In Dyspepsia connected with Marsh Cachexia, Alkaline Mineral Waters and other weak Mineral Waters are of great use.—Dyspepsia connected with affections of the Uterus is beneficially treated by the Local Treatment suitable to such affections, and also by General Treatment, particularly by Sea-bathing and Hydrotherapy.—In Dyspepsia resulting from Habitual Constipation, advantage derived from Belladonna, certain Purgatives, Mineral Waters containing Sulphate of Magnesia and other Sulphates.—In certain severe cases of Dyspepsia, the Inhalation of Oxygen Gas is resorted to.

Having made these preliminary remarks, I am now in a position to enter upon the question, so difficult and so complicated,—the *treatment of dyspepsia.*

From what I have said to you, you will perceive, that it is impossible to formulate precise rules; and that I must confine myself to mentioning a series of measures, applicable only to a limited number of cases, in which the disease has well-defined characters, and useless in the majority of cases, unless combined with other remedies selected according to the special indications.

When dyspepsia is associated with well-marked chronic gastritis, its treatment is subordinate to the treatment of that affection, and consequently consists in the use of remedies for inflammation of the stomach. In this, as in every form of dyspepsia, regimen constitutes the most important part of the treatment. The first requisite is to reduce the quantity of food taken, so as to render it proportionate to the aptitude of the stomach: this does not imply the necessity of putting the patient on low diet. The selection of the particular kind of food which ought to be prescribed is found, by the majority of physicians, to be a great difficulty. We doctors have all a strange manner of advising our patients on the subject of diet. If we ourselves are fond of tea or coffee, we are indulgent to those who use them habitually or even immoderately. If we prefer this or that kind of wine, if, for example, we prefer Bordeaux to Burgundy, we prescribe Bordeaux to the exclusion of the latter: if we have a fancy for strong meat—beef, mutton, or game—we prescribe strong meat for patients with bad digestion: if we order our patients to eat the flesh of young animals—veal or chicken—or if we advise them to take fish, it is because we ourselves like to eat this kind of food. In fact, it is not unusual for all the clients of a physician to be placed by the physician on the same diet as he himself adopts.

The law by which we ought to be guided in regulating the regimen of a patient is to recommend the food which the patient has found to agree best with him. This is the only really good and reliable rule to follow. The physician, therefore, ought at once to inquire into this matter. Should a person tell you that milk acts on him like a purgative, you will avoid ordering him to take milk, although it is perfectly well digested by you as by most other persons—you will avoid ordering an article of food which might induce vomiting, diarrhœa, and absolute indigestion. Nevertheless, how many physicians, without considering individual peculiarities, invariably order milk diet in chronic affections of the stomach! Therefore, interrogate your patients carefully, so as to ascertain exactly their dietetic aptitudes, and find out even their *fancies*, if you will allow me to use the expression, which vary with the person's state of health, and still more, perhaps, with the state of his disease. A man who has been suffering for some time from dyspepsia has a wonderfully correct knowledge of the aliments which will best agree with him: find out what they are, and recommend him to use them, even though they should seem preposterously unsuitable, and though personally you should have an antipathy to them.

I must add, however, that there are certain ordinary rules, which ought not to be neglected. Taking into account individual peculiarities, it may be stated, as a proposition which generally holds good, that light soups (made with or without animal food), poultry, fish, and non-farinaceous vegetables suit cases in which there is chronic inflammation of the stomach. Such is the regimen which ought to be prescribed by you in this class of cases, provided the articles which compose it have not already been found improper by the individual's own experience.

The same remarks which I have made on food are equally applicable to drink. Always making due allowance for individual idiosyncrasies, the general rule is to allow only a very small quantity of fluid to be taken, and to recommend fermented drinks, wine, or sometimes beer, diluted with water.

Regularity in the hours of meals is a point of no inconsiderable importance. Here, let me mention a matter of detail. It not unfrequently happens that dyspepsia, and the chronic irritation of the stomach on which it depends, arise solely from imperfect mastication, caused by loss of teeth, or by the patient swallowing his unchewed food. In such cases, to indicate the cause of the malady, is also to indicate the means of cure.

The question of regimen, I repeat, takes the most important place in the treatment of dyspepsia. Under a well-appointed regimen, without the use of any other means, the symptoms will disappear in a great many cases. The reason is obvious: a daily succession of fits of indigestion will be avoided, which would have been produced by unsuitable food, and by which the disease would have been kept up, just as a pulmonary catarrh will remain uncured so long as the patient is subjected to the evil influences by which it was originally occasioned.

Generally, however, dyspepsia is not cured by a return to regular habits and a judicious system of alimentation. The gastric symptoms continue with inveterate obstinacy, dependent upon the deepseated character of the chronic inflammation, which is characteristic of chronic inflammation of all organs. The obstinacy of the disease may also, to a certain extent, be due to the inflammation having the stamp of a special diathesis.

This remark renders it incumbent upon me to revert for a moment to what I explained to you in one of my previous lectures. When speaking of the sudoral exanthemata* I recalled to your recollection the fact that diathesic manifestations may declare themselves in internal organs, as well as in parts accessible to direct examination. Taking the herpetic diathesis as an example, I stated that the mucous membranes were very often the seat of its manifestations: and, with a view to show the transition of herpetic affections from the external to the internal integument, I asked, if we did not every day see individuals under the influence of this diathesis, take consecutively eczema of the face, occupying the upper lip or the external orifice of the nares, and very obstinate chronic coryza? In another individual a granular sore throat will supervene: in a third there will be deafness, occasioned by the extension of the irritation from the nasal fossæ and pharynx, to the mucous lining of the Eustachian tube. In women, certain uterine affections, certain leucorrhœal discharges are simply the result of an extension to the internal genital organs of an herpetic affection of the external parts.

In these cases, in which we have as it were the opportunity of following the affection step by step, as it progressively advances from without inwards, no one will deny the nature of the affection, be it coryza, sore throat, or uterine inflammation; but some physicians still refuse to admit, that these affections of the mucous membranes may be the sole manifestations of the diathesis, that they may have supervened consecutively upon the spontaneous or artificial disappearance of similar affections which had for a long time previously occupied a more or less considerable extent of the skin. Nevertheless, gentlemen, clinical experience demonstrates beyond the possibility of doubt that such metastases, such repercussions, to use the old phraseology, do occur. Experience tells us, that herpetic affections may not only invade the mucous membranes of the nose, larynx, and uterus, which are continuous with the external integument, and within range of visual observation, but that they may also invade more deeply seated organs. How frequently do attacks of bronchitis and diarrhœa, and, to return to our immediate subject, how often does dyspepsia depend upon an herpetic affection of the bronchial, intestinal, or gastric mucous membrane! The occurrence of such cases did not escape the observation of our predecessors; and it would not be difficult to collect from their writings a goodly number of cases similar to that described by Schmidtmann, of cardialgic dyspepsia alternating with eczema of the face, so that, when the eruption disappeared from the face, the patient experienced gastric symptoms, which did not subside till there was a re-establishment of the cutaneous disease. Your teachers, my honorable colleagues of the Hôpital Saint-Louis, intrusted with the wards specially reserved for diseases of the skin, have taught you this fact, which I have now pointed out to you as resting upon my own personal observation. Few weeks, indeed few days pass, in which I am not consulted by patients affected with dyspepsia, evidently dependent upon an herpetic diathesis. This diathesis imparts to the visceral affections which it produces, that characteristic obstinacy which belongs to it, just as it similarly impresses with the stamp of obstinacy every affection, acute or chronic, occurring in persons under its dominion. This specific character of the gastric affection ought therefore to occupy a leading place, when we come to consider the question of the treatment of dyspepsia. But, leaving out of consideration for the present this specific element of the chronic inflammation, let us inquire, how we are to modify the inflammation, independently of diathesis. Here, it must be admitted, that our available means are limited. In fact, we can do little more than remove causes, which is not always in itself sufficient, or resort to the employment of certain modifying topical agencies. When the affected parts are situated upon the exterior of the body, so as to enable us to apply directly our remedies, intervention is more easy, and is likewise more efficacious. In chronic ophthalmia, for example, it is easy to apply to the eye, different liquid collyria or powders—solutions of sulphate of copper, zinc, or nitrate of silver, or, in the form of powder, calomel, or oxide of zinc. If the inflammation be situated principally in the eyelids, we may use greasy applications: the pomade of Régent, into the composition of which enter the red oxide of mercury, crystallized acetate of lead, and camphor; the pomade of Desault (de Lyon), and many other unctuous applications of a like nature. In chronic inflammation of the nasal mucous membrane, in ozæna, we may cause the patients to snuff up mercurial powders, and inject caustic solutions, which are also peculiarly suitable in pharyngeal sore throat, and in vaginal and uterine inflammations. In a word, we may attack directly these inflammations, by modifying agents with which we are acquainted, and the action of which may be assisted by the use of remedies directed to the diathesis, or rather to the general state of the system upon which the local affections depend.

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* See vol. i, page 224.

We act with much less certainty by means of topical agents, in inflammatory affections of the stomach. However, when dyspepsia is dependent upon a chronic inflammation, which has retained to a certain extent its acute character, topical modifying agents, substitutive remedies, are indicated. Among them, emetics hold the first place. Their part does not consist in freeing the stomach from the saburral matter or bile which load it; for after the ingestion of aliment a part of this saburral matter or bile is evacuated; however, the mucous membrane thus cleansed, if one may use such an expression, remains inflamed as much as ever, and continues more or less to produce morbid secretions. To seek merely to evacuate these secretions would be as useless as to sweep away the morbid secretions which cover the skin affected by eczema. Here the abnormal secretions do not continue the less, and are scarcely removed from the surfaces which they pollute, when they are reproduced. The same thing happens in respect of morbid secretions of the stomach. Though in a case of poisoning the action of an emetic is mechanical, by causing violent expulsion of the deleterious agent which has been ingested, its operation is of a totally different nature in dyspepsia. In dyspepsia emetics act as substitutive agencies, as modifying powers, as I shall now endeavor to explain.

Tartar emetic, for example, when brought into contact with a mucous membrane, acts in the same way as upon the skin, that is, by determining violent inflammation; but this inflammation, subordinate to the quantity of the agent by which it is excited, undergoes spontaneous cure; and this occurs more quickly when the tartar emetic has been given in suitably graduated doses. The inflammation, therefore, is transient, and that is the characteristic of every inflammation excited to produce a therapeutic result. We may say the same of sulphate of copper, a topical irritant, quite as irritating to the gastric mucous membrane as to the mucous membranes of the eye or nose. When, therefore, we administer an emetic—tartar emetic or ipecacuan, polygala or veratrum album, sulphate of copper or sulphate of zinc—we substitute for the pre-existing gastric inflammation another kind of inflammation, transient in its character, and which will cease spontaneously. We act absolutely in the same manner as when we employ irritant collyria to combat inflammation of the ocular mucous membrane—in the same manner as when we treat by caustic injections the catarrhal affection of the urethra called blennorrhagia. Exactly in the same way emetics act beneficially in the treatment of dyspepsia. It is also by modifying the gastric inflammation, and not by causing evacuation by stool of the saburral matter, the bile, and the morbid secretions of the stomach, that calomel, gray powder, blue pill, and other mercurials prove useful in numerous cases.

These modifying remedies, however—emetics or purgatives—must be cautiously administered, for we cannot with impunity induce frequent vomiting in a dyspeptic subject. We might run the risk of going beyond the limits proposed; and the therapeutic action of the remedies being exceeded, we might see, in place of the chronic inflammation which we wished to supersede, a very violent inflammation, not at all of a transient nature, and calculated to induce serious symptoms.

In employing these remedies, having first of all found that they are indicated, it will be necessary to substitute other modifying agents, which, whilst they must be less energetic and less rapid in their effects, will at the same time not be less active. Such remedies are the subnitrate of bismuth and precipitated chalk. Employed daily as topical means, in certain cutaneous affections (as in the chafing of the skin of infants) these remedies are useful; their efficacy is likewise incontestable in certain chronic inflammations of the large intestine. My friend, Dr. Lasègue, has made known the beneficial results which he obtained in such cases, both in adults and in children, by the employment of injections containing subnitrate of bismuth and chalk. Their utility is not less in the dyspepsia of chronic gastritis. They ought to be given in large doses: from five to ten grammes of the chalk may be administered in the course of the twenty-four hours, mixed with an equal quantity of subnitrate of bismuth, and divided into packets containing from two to four grammes. These powders ought to be taken (as a general rule) before meals.

The secretions of the stomach resume their normal character on the cessation of the inflammation of the gastric mucous membrane. It is necessary, however, in some cases, to give special aid to the secretory functions, which have got into a state of greater or less disorder. Certain acids, such, for example, as lactic and acetic, or, better still, hydrochloric acid, which you have often seen me prescribe, are excellent remedies in the dyspepsia of chronic gastritis. At the same time—and the fact is remarkable—while some persons are benefited by acids, others derive no good from them: to such it is necessary to administer alkalies. It is difficult to determine which class of remedies will prove most suitable; and it is also difficult to state the manner in which acids and alkalies act. The chemical explanations which have been given are open to great objection, particularly as we see acids and alkalies produce equally beneficial effects in different individuals, whose cases are, apparently, exactly similar. Without stopping to consider the interpretation which chemists have given of the manner in which these medicines operate, let us for the present be satisfied with the knowledge we derive from clinical experience. We know that in chronic affections of the stomach, when the patient, after having been subjected to the previous treatment of which we have just been speaking, retains difficulty of digestion, sometimes alkaline mineral waters, and sometimes, though not so frequently, acid mineral waters are administered with success. When I come to speak of acid dyspepsia, I shall return to this point: I shall then discuss the probable manner in which acids and alkalies act. For the present, let it suffice to call your attention to this subject, reserving for a future occasion the remarks which I have to make upon the circumstances which seem specially to indicate one or other class of remedies.

Gentlemen, in pointing out the different forms which dyspepsia may assume, I have told you that there is one form of the affection accompanied by bulimia, or to speak with more precision, by a feeling of emptiness of the stomach soon after eating. I told you that in this form of dyspepsia the disorder of the digestive functions was characterized by diarrhœa, supervening almost immediately after eating. Patients who are thus affected will tell you that they digest very rapidly—that their food is not heavy on the stomach—that their stomach is in excellent order—and that the disorder is only in the intestines. I have told you the way in which I explain the cause of the symptoms in cases of this description; and I shall enter more into details when I come to treat in a special manner of diarrhœa. This, however, is the suitable occasion to state the means at our disposal for the treatment of these cases.

I begin by speaking of opium. This medicine, although sometimes deplorably misapplied in the treatment of diseases of the digestive organs, is, in the class of cases now before us, more useful than any other remedy. To derive from it, however, all the benefit it is capable of imparting, it requires to be given with the greatest circumspection. It is impossible for me to tell you the exact doses in which it ought to be administered. In each particular case, the physician must decide this question by considering the tolerance of the individual for opium. There exists great diversity in this respect, not merely in the differences of tolerance in individuals, but also in the difference between the degree of tolerance which the same person has at different times, according to the varying circumstances in which he may happen to be placed. Some persons can bear enormous quantities of opium; and I mentioned remarkable examples of this peculiarity when lecturing upon epileptiform neuralgia. Others are affected by a single drop of laudanum: this statement is applicable to adults; but young children are sometimes narcotized by even one-fourth of that quantity. Nothing is so difficult as to judiciously manage opium. On this fact I cannot lay too much stress; for no remedy is dispensed so improperly, so prodigally, and with so little inquiry into the idiosyncrasy of the patient. Note well, gentlemen, that this observation has a general bearing, and does not only apply to what is done in the treatment of dyspepsia. During my clinical lectures I shall have frequent opportunities of raising my voice against this abuse. In the malady before us to-day—bulimic dyspepsia with constant diarrhœa —opium is, however, a wonderful remedy, provided it be administered in moderate doses. The laudanum of Sydenham is the most convenient preparation to employ, for its doses are the most easily apportioned. It is prescribed at first in doses of *a single drop*, the dose being augmented if necessary. The patient ought to take it before, and not after, eating. To obtain a successful result from the remedy, this precaution is indispensably necessary. The small quantity of opium received into the stomach before digestion has commenced is sufficient to keep duly quiet and regular its muscular excitability, the inordinate extent of which causes the symptoms you have to combat; this, too, it accomplishes without suspending organic sensibility. Opium, on the contrary, administered in large doses, producing effects beyond those intended, causing slumber both of the muscular excitability and organic sensibility, arresting at once the muscular movements and the secretion of the gastric juice, increases, in place of calming the disturbed state of the digestive function, to the performance of which regular muscular movements and secretion of gastric juice are indispensable.

Belladonna is undoubtedly useful in this form of dyspepsia, though its beneficial action is less decidedly beneficial than that of opium. Perhaps you are surprised to hear me praise this medicine in these cases, as its usual effect is the very opposite of that which we wish to obtain in them. You are aware that belladonna, in common with all the poisonous *solaneæ*, produces relaxation of the bowels, while opium causes constipation. So decidedly is this property characteristic of belladonna, that the physician avoids administering it to patients affected with diarrhœa. But while there is a reason for not prescribing it in cases of diarrhœa in which the cause of the flux exists in the intestine itself, it would be wrong not to employ it in the cases to which I am now directing your attention. I have no hesitation in stating in the most positive manner, that cases of this class occur in which belladonna renders services very nearly equal to those derived from opium itself. Here, a word of explanation is necessary. Experience tells us that the poisonous *solaneæ* are very often our most powerful means of conquering constipation. You all know the effects of tobacco: to some individuals, a cigar is the best laxative; and there are persons whose only security for a daily stool is a daily cigar. There are others upon whom tobacco produces no laxative action, but upon whom this is produced by a pill containing a grain or half a grain of the extract of henbane. These substances perhaps owe this singular property to the poisonous principle which is the active base of all the *solaneæ*. Belladonna, the utility of which in some cases of constipation is so well known, particularly since the researches of Bretonneau, acts in virtue of this principle. Here the same remark applies which I made in respect of opium: it can only be administered in very small doses. A centigramme [the seventh part of an English grain] is generally sufficient; though one is sometimes obliged to give a somewhat larger dose, say, for example, a centigramme and a quarter, or two centigrammes and a half, but there is rarely any necessity for exceeding these quantities. It might appear, that what I have just said contradicts my former statement regarding the administration of belladonna as a means of arresting diarrhœa; but this contradiction is only apparent, for, if the diarrhœa depends upon an increased excitability of the stomach, the belladonna will calm the excitability and suspend the diarrhœa, by moderating the abnormal condition upon which it depended.

Though the poisonous *solaneæ*, particularly belladonna, may be of great service in these cases, we must not forget that their abuse, particularly the abuse of tobacco by smokers, is a cause of dyspepsia. This is an important fact. The nicotine absorbed by smokers in greater or less quantity diminishes the physiological excitability of the stomach. In such circumstances, patients experience almost always a sensation of weight in the epigastric region; stomachic digestion proceeds exceedingly slow, and it is in vain that we have recourse to treatment calculated to rouse the inactive stomach, unless we get the patients to discontinue, or at least to moderate, the injurious habit of smoking.

It is important, therefore, in prescribing belladonna, or any other remedy derived from the *solaneæ*, not to exceed certain limits, otherwise a sort of paralysis might be induced, which would have to be treated by aromatic or alcoholic stimulants, or, better still, by the preparations of nux vomica. It is consequently necessary, as I have just said, to begin with small doses, increasing them if necessary.

In the same category as the *solaneæ*, certain antispasmodic remedies, such as valerian, assafœtida, and oxide of zinc are indicated. All of these remedies ought to be given in very small doses, and always at the beginning of meals.

Gentlemen, acid dyspepsia—often associated with flatulent dyspepsia—is a more common affection than that which I have just been discussing. The physician frequently makes serious mistakes in the acid form of the disease, in which sour eructations and copious secretion of gas occur during digestion. We physicians have the misfortune to be very bad chemists. I am not doing an act of injustice to any one, when I say, that of the 300 now present 299—myself included—deserve this reproach. Nevertheless, with an amount of assurance proportionate to our ignorance, we do not hesitate to apply to therapeutics the little knowledge of chemical theories which we possess. Laboratory experiments having taught us that acids neutralize alkalies, we lay hold of the fact: taking it as a starting-point, the treatment of certain cases of dyspepsia seems simplicity itself. The stomach contains a large quantity of acid, which, say we, must be neutralized: we can obtain this result by administering magnesia, bicarbonate of soda, lime-water, or chalk. Notwithstanding our reasoning, the evil increases, the acid secretion becoming more abundant in place of diminishing. We nevertheless still cling to our original opinion: in the increased severity of the symptoms, we only see an additional reason for insisting more strenuously than ever upon our treatment. We immediately double or triple the doses of the alkali, when we find that we have obtained no beneficial results from the doses first prescribed. Soon afterwards, very probably, the patient is seized with diarrhœa: in place of any benefit having resulted from our treatment, matters have become worse. Being thus baffled in our curative efforts, we are obliged to impute to the obstinacy of the disease consequences entirely due to our untoward interference.

In such cases, as well as in many others, a certain amount of physiological knowledge will suffice to prevent our falling into the errors towards which chemical theorizing tends. Physiology teaches us that the gastric juice is naturally acid—that this acidity is its constant condition both in man and the lower animals, irrespective of species, sex, age, or food—that it is due to the presence of phosphoric, hydrochloric, and lactic acids, but particularly the latter, which alone is found in a free state. These acids are secreted in greatest abundance during digestion; and their secretion is indispensably requisite to the due performance of the functions of the stomach. When digestion is not going on, the gastric secretion is less abundant, and feebly acid; or sometimes, it is neutral or even alkaline. As I have already mentioned, the normal secretion of gastric juice is sometimes partially suspended: but there are other cases in which it is secreted in too great quantity, and this is the point which I have in view. Irritation of the mucous membrane of the stomach, provided it neither proceed to the extent of inflammation, nor too far, causes increased secretion: excessive irritation or inflammation arrests the secretion. This has been exclusively established by the experiments of Beaumont on his Canadian,* and by the often-repeated experiments of Claude Bernard.† Mental emotion and protracted occupation at the desk occasion increased secretion; and are, as you know, very common causes of indigestion accompanied by eructations and vomiting.

In cases of this description, you cannot counteract the acidity by bicarbonate of soda, nor indeed by any other alkalies acting as chemical agents. Farther—and from the practical position upon which I take my stand, the fact is of paramount importance—the experiments of Claude Bernard upon animals prove that the secretion of gastric juice, and consequently the acid fluids of the stomach, increase when bicarbonate of soda, magnesia, or other alkalies are administered; while the secretion is delayed or diminished by giving acids. These positive facts entirely set aside the trivialities of chemical theory, which are of no use as guides in the treatment of disease, and can still less lay down therapeutic laws to us, as some consider ought to be the case. When chemists tell us that alkalies are useful in a considerable number of cases of acid dyspepsia, they only repeat what we had previously learned from clinical experience. But when they state that the benefit is produced by the alkalies neutralizing the acids, we reply that no neutralization has taken place, or if there has, it has been only to a very limited extent. On the other hand, we maintain that these remedies act as powerful modifiers, which not only place their stamp upon the organ, but also impart a peculiar modality to the whole economy, in virtue of which the functions are regulated, and the abnormal acidity of the secretions is corrected.

Let me give another illustration of my view of this matter; and one which, I think, will enable you better to understand it. A person affected with gravel, who has passed some gravel during or soon after his treatment, will remain for six months, eight months, or a year without passing any, when under the influence of a season passed at Pougues or Contrexéville using the waters. Now, will any one say, that these feebly alkaline waters have maintained an alkaline action during all that time? Certainly not. The proper answer is, that these waters, by restoring the economy to a healthful condition, or (if we wish more to localize the effect), by modifying the urinary apparatus in a salutary manner, have restored the kidneys to the natural performance of their secretory function, and have so prevented uric acid from being formed in excess. If they have exerted any chemical influence upon the products of excretion, it has been very transient. In fact they have an action—a vital action—which is much more powerful than any chemical action, and which, when once set in motion, continues for a much longer period.

The same is seen in dyspepsia. If the waters of Vichy, of Pougues, or of Vals have no other action than that which takes place in virtue of the chemical reaction caused by the alkalies which they contain, to be logical, it would be necessary to insist upon the patients continuously using these waters to maintain their supposed neutralizing effect. Their beneficial operation is no more chemical in dyspepsia than in gravel: it depends upon their impressing on the economy a certain modality, in virtue of which the gastric secretions are so regulated as not to contain more than a normal quantity of acids.

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* BEAUMONT: Experiments and Observations on the Gastric Juice and the Physiology of Digestion. Plattsburgh, 1833.

† BERNARD (Claude): Cours de Médecine du Collège de France: Liquides de l'Organisme. Paris, 1859.

The remarks now made in relation to the action of alkalies in gravel and dyspepsia, are applicable to many other articles of our materia medica. Therapeutical action does not admit of chemical explanation: it is essentially *vital*, or if you prefer the expression, essentially *physiological*.

A healthy woman, for example, takes iron in large doses. Menstruation is disturbed, and the catamenia are suppressed, in a great many such cases. What has taken place? The iron, given inopportunely, has deteriorated the health of the individual, the result of which deterioration has been suppression of the menses. But supposing that we give to a chlorotic woman the same remedy in even larger doses, the result will be entirely different—menstruation, which was before imperfectly performed, will become normal. Chemists will have no difficulty in explaining the last fact; but I should like you to tell me how they can explain the first.

If other proofs were wanting to support this medical view of therapeutic action, or (to return more directly to our subject), to explain the action of alkalies in dyspepsia, we should find the required evidence in the fact, that in many, perhaps in most cases, we easily cure dyspepsia connected with an excess of acid secretion, by the employment of other means, which I am now going to mention, and which can hardly be explained by any chemical hypothesis.

Graves stated that abnormal gastric secretion was powerfully and favorably modified by medicines acting specially on the nervous system. At the head of this class of remedies he placed *opium*, given in very small doses. He, it is true, combined it with the subnitrate of bismuth. The particular medication from which he derived marvellous effects, consisted in administering a mixture of two milligrammes and a half [2½ seventieths of a grain] of sulphate of morphia, or five milligrammes [5 seventieths of a grain] of thebaic extract with from fifty to seventy-five centigrammes [7¾ to 12 grains] of subnitrate of bismuth and an equal quantity of magnesia. This is administered twice or thrice daily a short time before meals.

The choice of mineral waters in the treatment of acid dyspepsia is regulated by the causes which produce the disorders of digestion, a fact which supports the thesis I sustain, to the effect that chemical explanations of therapeutic action are worthless. The indications for the use of this, or the other mineral water, do not depend upon the acidity, more or less decided, of the stomachic secretion, but upon the general state of the economy, with which the perverted gastric function is associated. Thus, when acid dyspepsia is associated with chlorosis, ferruginous mineral waters ought to be prescribed in preference to all others. Of this class, are the waters of Spa, in Belgium, of Schwalbach, in the Duchy of Nassau, and of Pougues, Bussang, Forges, Passy, and others in France.

Dyspepsia in hysterical women, in hypochondriacal men, in all very nervous persons, in great eaters, and in old people, is chiefly *flatulent;* that is to say, characterized by the formation of a large quantity of gas, and accompanied sometimes by acid eructations supervening immediately after meals. In this form of dyspepsia, alkaline preparations are also of some use, if given only for a few consecutive days, and immediately followed by the administration of bitters.

Thus, for five or six days, the patient ought to take at the beginning of his two principal meals, and on going to bed at night, a powder composed of magnesia, chalk, bicarbonate of soda—from thirty to forty centigrammes [4½ to 6 grains] of each. These powders ought to be mixed immediately before they are taken in about a fourth part of a tumbler of water.

This treatment is to be followed up by the employment of bitters, among which I think quassia ought to occupy the chief place. In the morning fasting and at midday, at an equal interval between the two principal meals, the patient ought to drink a cup of the infusion of this bitter wood, prepared by leaving a teacupful of cold water for fifteen or twenty minutes, in a goblet made of quassia; or (which is still better) by macerating two grammes of quassia shavings in cold water, for from four to six hours. I have seen this form of dyspepsia yield much more rapidly to this simple treatment, than to the long-continued use of alkalies. In these cases, wine of cinchona is also indicated. It ought to be given either immediately after meals, or immediately after the patient has taken a small quantity of food. By proceeding upon this plan, we prevent pain in the stomach, which is apt to be excited when wine of cinchona is taken fasting.

In flatulent dyspepsia, also, decided advantage is obtained, by the use of certain *liqueurs* administered after meals. Those which I prefer are *anisette fine de Hollande*, and the yellow liqueur of the Grande-Chartreuse, which is simply an alcoholic tincture of various aromatic plants. I need hardly add, that these liqueurs must be taken in very small quantities. Other aromatic preparations may be substituted for them. For example, we may give the infusion of illicium anisatum (or star anise), one of the ingredients of the *anisette de Hollande*, or we may give an infusion of a mixture of star anise, common anise, ginger, and cascarilla bark. These substances, when reduced to coarse powder, are weighed out in packets containing fifty centigrammes of each ingredient. Their infusion is taken immediately after meals.

Mineral waters are of undoubted utility in these dyspeptic affections: but we must not send the patients to Vichy, Carlsbad, or Pougues; for the waters of these places are contraindicated. We must recommend them to go to Niederbronn or Forbach, where the predominating mineral ingredients are the same as those of sea-water. We may also recommend Nauheim, Soden, and Kissengen, which also contain chloride of sodium. The waters of Homburg are likewise in the same category; but unfortunately, the too celebrated gaming-tables of the town damage the reputation of the springs.

Without leaving Germany, we may mention the water of Selters, in the Duchy of Nassau, better known in France by the name of *eau de Seltz*. Each litre of this water contains about two grammes of chloride of sodium, one gramme of carbonate of soda, nearly half a gramme of carbonate of lime, and carbonate of magnesia, a small quantity of sulphate of soda, a minute proportion of carbonate of iron, and an indeterminate quantity of carbonic acid. Its temperature varies between 15° and 20° C. Its agreeable taste has so vulgarized its use, that it is served at the tables of the inns and eating-houses as commonly as artificial *eau de Seltz* is similarly made use of in Paris. I may remark in passing, that artificial does not in any respect resemble the natural *eau de Seltz*.

Some French mineral waters, such as those of Plombières in the Vosges, and of Bagnères-de-Bigorre in the Hautes-Pyrénées though containing only a minute quantity of mineral ingredients, are also very useful in flatulent dyspepsia.

Hydrotherapy is a method of treatment in this kind of dyspepsia which is not less efficacious than those I have now reviewed. Its use in other forms of dyspepsia is not great. Let it be understood that the hydrotherapic treatment which I now speak of is hydrotherapy methodically applied, and carried out in a regular manner.

Sea-bathing, I place in the same category as hydrotherapy. The patient ought to remain a very short time in the water, if he bathe on the coasts of the Manche, or on our northern ocean-coasts. On the sea-shores of the southwest of France—in the Mediterranean—the duration of the bath may be longer, as the climate is warmer. In these regions, in addition to bathing in the sea, the patients may use baths of sand naturally heated by the sun. Patients ought to remain in these baths of sand for from fifteen minutes to an hour—in fact till a decided reaction has been established in the skin.

Unfortunately, sea-bathing, travelling to mineral springs, and hydrotherapy in a hydrotherapic establishment are means of treatment which are not accessible to all. Business necessities and expense—matters which we must always take into account—often place these remedial measures beyond the reach of our patients. In such cases, the hydrotherapic treatment may be pursued according to a plan which I am now going to describe; and which though no doubt less efficacious than the methodical system of a hydrotherapic establishment under medical direction, is nevertheless really beneficial. Home-hydrotherapy consists in enveloping oneself on getting out of bed in the morning in a wet sheet slightly wrung out of cold water. After remaining for one or two minutes wrapped up in the wet sheet, you rub yourself or get yourself rubbed with it, you are then rubbed with linen which is quite dry, but not warmed; after this, you dress, and as soon as possible start on a walk which you continue for three-quarters of an hour. The hydrotherapic operation may be repeated at night before going to bed. Great advantage may also be derived from immersions (not exceeding three minutes' duration) in cold salt water. Hydrotherapy pursued after this fashion will suffice in many cases so to modify the action of the whole economy, as to cure the gastric disturbance and restore to the stomach its lost tonicity.

Gentlemen, I have hitherto spoken of forms of dyspepsia having their causes primarily and directly in the stomach. Before completing what I have to say to you on the subject of dyspepsia, I must speak of the treatment of those cases of dyspepsia which are to a certain extent independent of the gastric apparatus—independent in this sense, that the apparatus is only indirectly involved, that the disorders of which it is the seat are the result of sympathy between affections of the stomach and different parts of the digestive tube; and likewise between the digestive and other organs of the economy. The forms of dyspepsia of which I am now going to speak are those which so frequently accompany chronic affections of the liver and uterus, diathesic diseases such as scrofula and tubercle (particularly pulmonary tuberculization), and marsh and other cachexiæ. I must go over this ground rapidly, otherwise, as you can easily understand, I should run the risk of exceeding my limits, and ranging too widely over the domains of pathology; for there are few maladies in which dyspeptic symptoms do not play a more or less conspicuous part. I shall, therefore, make no attempt to exhaust the subject: on the contrary, I shall limit myself to giving you some practical indications, having specially in view the cases which we have observed together.

In respect of diseases of the liver, of which we have had a certain number of cases under observation, let me say, that in the dyspeptic symptoms which arise in connection with them, alkaline mineral waters are marvellously efficacious. Among them, such waters as those of Carlsbad, Vichy, and Vals, which no doubt owe much of their usefulness to the bicarbonate of soda, their predominating mineral ingredient, are very preferable to the waters of Pougues and the like, in which the bicarbonates of lime and magnesia predominate.

However, whilst I proclaim the efficacy of the alkaline waters, there are cases in which I prescribe acids. You have seen me order acids to be taken by many patients who were unable to digest their food unless they took a small quantity of hydrochloric acid after each meal.

The equality of success which attends the use of alkalies in some, and of acids in other patients, might seem to imply a contradiction; but this is a notion against which I must guard you. The contradiction is only apparent: it is in fact an additional confirmation of the remarks I have just been making as to the worthlessness of chemical explanations of vital phenomena, which belong to the domains of physiology and clinical medicine.

We must bear in mind the fact derived from clinical observation that both alkalies and acids have a general action, not only on the whole intestinal canal, but also, and still more, upon the entire economy. So much is this the case, that it is not a matter of indifference which particular acid or alkali we select. Mineral waters which derive their alkaline properties from bicarbonate of lime or from magnesia, are, as I have already pointed out, much less efficacious than waters containing bicarbonate of soda, in the dyspepsia now under consideration.

The case of one of our patients in St. Bernard Ward afforded a remarkable example of the difficulty which occasionally exists in instituting a regular plan of treatment, and of the necessity which sometimes arises of combining the use of means apparently the most diverse. The patient to whom I refer was a young woman who occupied bed No. 9. She came into hospital on account of severe colitis, characterized by glairy, sanguinolent stools. She was between the fourth and fifth month of pregnancy; and her malady brought on abortion. "If," said Hippocrates, "a pregnant woman is attacked with profuse abdominal flux, there is reason to fear that she will abort." I detected great hypertrophy of the liver with effusion into the peritoneum. For a long time, the patient remained in a condition of considerable danger: nevertheless, convalescence was established, although the liver continued greatly hypertrophied and very painful on pressure, and although digestion was still very badly performed. I tried alkaline remedies, without any good result: the symptoms continued, and there was a speedy return of the diarrhœa. It then occurred to me to try hydrochloric acid. The patient began by taking, after each meal, one drop in a quarter of a tumbler of sugared water: this was found to promote digestion. I then increased the quantity of the acid: first, the increase was to three drops daily, one drop being taken after the morning, and two after the evening repast: subsequently, two drops were taken after each of these meals. From that time, there was a complete cessation of the feeling of weight in the stomach, and of the sensation of fulness after eating: and it is a remarkable fact, that along with this improvement in the digestion, the bulk of the liver diminished: there was nevertheless increased diarrhœa. Under the circumstances, I deemed it advisable to suspend the use of the acid, and give in place of it prepared chalk, which I have always found useful in intestinal flux. The result was arrest of the diarrhœa, and reappearance of the dyspepsia. I then again suspended the alkali, and reverted to the use of the acid mixture, whereupon the dyspepsia again yielded, and the diarrhœa returned. I was greatly perplexed how to act: and at last resolved to combine the use of both medicines, prescribing the chalk at the beginning and the acid at the conclusion of the meal. This combination proved successful: the patient was relieved from all her morbid symptoms.

This history possesses great practical interest: it shows the physician that, in respect of such cases, he in reality knows next to nothing, or absolutely nothing. We search for explanations, and for so doing we cannot be blamed, as in no other way can we systematize our knowledge, and establish for our guidance certain laws, which no doubt may be more or less defective, but which nevertheless prevent us from acting as mere empirics. Unfortunately, our explanations are generally incorrect.

Here, you see is a case of dyspepsia associated with severe disease of the liver in which there could be no doubt as to the utility of acids. They are also useful in numerous cases of dyspepsia connected with chronic maladies.

How was I led to adopt this mode of treatment? Long ago I had read in the English medical journals accounts of cases treated and cured by mixtures having hydrochloric acid as their principal ingredient: I knew that Cullen had said: "All the acids seem to have the power of stimulating the stomach, and consequently of increasing the appetite: the acids particularly used with success are vitriolic acid and marine acid [hydrochloric acid], and that acid which is formed by the distillation of vegetables, and that derived from tar-water." I had also observed in different works published in France, particularly in the work of Dr. Caron, that acids were advantageously prescribed in certain disorders of the digestive system. I had never found, however, the special indications of this treatment formulated with sufficient exactness. I consequently was somewhat incredulous, and inclined to believe that the patients had recovered, not through taking hydrochloric acid, but in spite of having done so; but some years later, when one day sitting at dinner, next to one of those indefatigable tourists who seem to personify perpetual motion, I was informed by him, that he (being compelled by his constant peregrinations to adopt a great diversity of regimen, and to take his meals very irregularly) was indebted to hydrochloric acid for the recovery of his digestive powers lost through the irregularities described. He never travelled without his precious remedy. He always carried with him a little bottle of dilute hydrochloric acid, of which he took from four to eight drops at the conclusion of each meal. I was very much struck by this statement; and after a long conversation with my tourist, I became quite satisfied that his custom was not the result of a mere fancy, but was a positive necessity. I then set myself to study the English authors: the indications which I gleaned from the works of Cullen and other authors, were not more precise than those gathered from conversation with the traveller. Nevertheless, I tried the treatment upon some private patients: at first, I proceeded timidly, but soon found that in certain cases, not however very distinctly characterized, real benefit was derived from the hydrochloric acid; I continued my experiments, some of which you have witnessed in the treatment of our hospital cases.

As I have just stated to you, it is in dyspepsia associated with chronic disease, that the benefit derived from this treatment has appeared to me to be especially marked, although it is likewise seen in cases of another description.

When speaking of the treatment of dyspepsia arising from chronic gastritis, I have spoken to you of the utility of hydrochloric acid, but it has always appeared to me to be more decidedly indicated in cases connected with chronic disease.

In bed 23 of St. Bernard Ward we had a young woman, affected with obstinate chronic diarrhœa, who had fallen into such a state of anæmia and emaciation that I thought she had tubercular phthisis, although, upon the most attentive examination, I was unable to detect any sign of that condition. In addition to the intestinal flux, the patient had that peculiar form of dyspepsia characterized by the state called great fulness of the stomach. I ordered her to take hydrochloric acid at first in doses of one drop, then in doses of two drops, and afterwards in doses of three drops, at the conclusion of each meal: digestion soon became improved, but it was necessary to continue the treatment for a long time, for, whenever it was discontinued, difficulty of digestion immediately returned. It is true that the diarrhœa did not yield. Some of you, no doubt, will recollect this patient, whose curious case has been, for more than one reason, reported in the work of MM. Gros and Lancereaux.* The symptoms with which she was affected, and the nature of which we did not for a long time detect, depended upon constitutional syphilis: they did not disappear till after the patient had been subjected to mercurial treatment.

In the same ward there was, at the same time, a patient suffering from very manifest pulmonary tuberculization. The progress of the tubercular affection seemed for a time to be arrested; lost flesh was regained, and the general condition became improved. The local signs were also modified: moist crackling had succeeded to slightly prolonged expiration, mingled with some disseminated mucous râles: when fresh hæmoptysis took place, the moist crackling reappeared, and to these symptoms dyspepsia was added. Four or five hours after eating, the patient experienced a feeling of weight in the stomach. Hydrochloric acid given at meals remarkably aided digestion, which was only properly performed so long as the use of the remedy was continued.

In bed 27 we had a similar case. This woman, who was also the subject of tubercle in a state of softening, was becoming weaker day by day. During the night she had burning fever, followed by profuse sweating. There was hypertrophy of the liver, as is very often the case in phthisis. She suffered from indigestion and diarrhœa. Hydrochloric acid promptly cured the gastric symptoms, but did not, of course, arrest the progress of the tubercular disease. I could give you histories of a number of similar cases. Indeed, it is chiefly in dyspepsia supervening in phthisis that I have found the acids of great use.

I too have tried to found a little theory of my own upon the results of my experience. I have reasoned thus: During digestion the stomach contains a certain quantity of lactic, phosphoric, and hydrochloric acids. Does, said I to myself, my medication prove successful because it supplies the gastric juice with a certain amount of acid in which it is deficient? I tried lactic acid in doses larger than those I had given of the hydrochloric acid: I began with ten and went on increasing the dose to twenty drops, but, still finding the dose insufficient, I gave as much as two and even three grammes. The results were very variable: the lactic acid, however, I found did less good than the hydrochloric, so for the future I preferred the hydrochloric.

To sum up these remarks, gentlemen: Without giving any account of the action which takes place in the digestive canal under the influence of acid or alkaline remedies, let us always remember that alkalies are not the only therapeutic agents available in the treatment of dyspepsia connected with chronic diseases: that acids are also indicated, but that the indications cannot be formulated in advance with exactitude, and that they can only be discovered by attentive observation in each case.

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* Gros et Lancereaux: Des Affections Nerveuses Syphilitiques. 8vo. Paris, 1861.

I have now come to a very important part of my subject—the treatment of dyspepsia coincident with more or less anæmia, and more or less hepatic and splenic engorgement. Such complications, observed in persons who have long suffered from marsh fevers, or in persons who, though they may not have suffered from marsh fevers, have lived for a long period in marshy countries and been subjected to their miasmatic influences, must be carefully distinguished from the similar complications which characterize leucocythæmia. This is very important, because leucocythæmia is a disease against which medicine is powerless; whereas under the other conditions described there is generally rapid recovery.

Whether dyspepsia and its accompanying visceral engorgement depend on anæmia or be its cause, I cannot say; but in either case the gastric symptoms, the hypertrophy of the spleen and liver, are very often successfully treated by means which one certainly never would expect to be useful. At the military hospital of Vichy, for instance, which contains a large number of patients suffering from paludal cachexia, characterized by hepatic and splenic engorgement, and dyspeptic symptoms of more or less severity, we see recoveries or at least very rapid ameliorations under the use of the Vichy thermal alkaline water, which is specially efficacious in that class of cases. So general is the fame of the efficacy of the waters of Vichy and Pougues, in affections consecutive upon paludal poisoning, that it is a constant practice of patients to resort thither from the Nivernais, the Berri, the Bourbonnais, and Auvergne for their cure. This is a case in which popular opinion and medical observation entirely agree. The medical practitioners of Vichy unanimously proclaim the virtues of their thermal springs in dyspepsia and other functional and organic disorders depending on paludal cachexia. My lamented colleague, Dr. L. de Crozant, late medical inspector at Pougues, published interesting works with a view to make known the usefulness of the waters which he administered with so much science and intelligence.* These remarkable properties of the waters of Vichy and Pougues have been long known to, and admitted by, physicians.

I ask you whether there is, at first view, anything more anomalous, more opposed to chemical theory, than to administer to patients whose blood is in so dissolved a state that dropsies and passive hemorrhages are of frequent occurrence, alkalies, which are looked upon as peculiarly possessing the properties of blood-solvents? Whether the predominating ingredient be bicarbonate of soda, as in the waters of Vichy, or bicarbonate of lime, as in the waters of Pougues, the waters administered are alkaline, and their good effects emphatically contradict the statement of the chemists in relation to the action of alkalies upon the blood. I know very well, that it is customary at Vichy to prescribe the Lardy spring to persons suffering from paludal cachexia, and that this Lardy spring contains a certain very small proportion of the bicarbonate of the protoxide of iron—about twenty-eight thousandths of a gramme. I also know very well that the waters of Pougues contain bicarbonate of iron nearly in the same proportion as the Lardy spring of Vichy; and that both contain carbonic acid gas, and that their beneficial effects may be attributed to the iron and the carbonic acid gas. However, at Vichy the same class of patients recover by using the Grande-Grille spring or the Hôpital spring, as completely as, though less rapidly than when they drink from the wells of Lardy; and still less rapidly than patients who go to Pougues. It appears certain, therefore, that the honor of the cure ought to be attributed to the mineral alkali.

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* L. DE CROZANT: De l'Emploi des Eaux Minérales de Pougues dans le Traitement de quelques Affections Chroniques de l'Estomac. Paris, 1851.

I have been desirous to point out these facts to you, that I might put you thoroughly on your guard against chimiatria, which, particularly in its applications to therapeutics, leads to deplorable mistakes. I am not at all afraid of recurring too often to this topic, so strong is my conviction of the correctness of my views, founded as they are upon long practical experience, and on an attentive observation of cases. Distrust the theories of the laboratory! Remember the remark of my honorable scientific friend Dr. Lasègue, to the effect that, though chemistry is capable of rendering to medicine the most important services, the chemist goes beyond his legitimate sphere when he draws clinical inferences from the experiments of the laboratory; and that chemistry does not approach any nearer to medicine, when teaching the art of preparing and analyzing medicines, than it approaches painting, when it furnishes fixed and durable colors. This proposition, true in respect of the general articles of the materia medica, is specially true of mineral waters, although for them chemistry is endeavoring more than ever to monopolize the right of explanation and to constitute herself the decisive judge. Whatever may be said to the contrary, mineral waters are not simple medicines: whatever may be the mineral element which analysis shows to be predominant, that element does not act alone: by associating with it quantities more or less notable of very various principles which the chemist can isolate, as well as others which have not yet been discovered, nature has given to the mineral element a something which we seek every day to imitate in our prescriptions, when we endeavor to increase or diminish the effects of a particular medicine, by associating it with other medicines. In taking into account, however, the particular effects of this or that ingredient of mineral waters, we cannot attribute them to a single principle, however dominant chemical analysis may show it to be; it is by clinical experience alone that we can arrive at a correct judgment on this point. So true is this, that the forms of dyspepsia associated with a formidable paludal or other cachexia are beneficially modified by waters very different from those of Vichy or Pougues, by waters, the mineral ingredients of which elude, so to speak, chemical analysis, such as the waters of Plombières and of Bagnères-de-Bigorre. Although the first are placed in the class of sulphurous soda waters, and the second are considered as sulphurous lime waters, they have so small a proportion of mineral ingredients that the predominance of one or other mineral ingredient either destroys the classification or renders it purely artificial. If we compare them in respect of their composition with the waters of the Seine, taken at different points of the river as it passes through Paris, with the waters of Arcueil, or with those of the artesian well of Grenelle, the superiority will rest with the latter, at least so far as the waters of Plombières are concerned. But, nevertheless, medical experience tells us that the waters of the Seine have no other peculiar property than that of occasioning diarrhœa (generally slight) in persons not accustomed to use them, which cannot be attributed to the small quantities which they contain of the salts of soda and chlorides. So far as I know, these waters have never yet been inserted in any of the voluminous lists of mineral waters which have been published. In thus instituting a parallel between the waters of the Seine, Arcueil, and Grenelle, and those of Plombières and Bagnères-de-Bigorre (to which I would add the waters of Néris or Mont Dore, which are scarcely more mineralized), I am far from wishing to deny the efficacy of these justly celebrated thermal springs. Plombières and Bagnères-de-Bigorre in the particular class of cases now before us, triumph over rebellious dyspepsia, in virtue of a therapeutic action the nature of which eludes us, and which I do not even attempt to explain. Under their salutary influence, the appetite is restored, and the constitution renovated. Patients affected with dropsy and visceral engorgements who have arrived at Plombières or Bigorre in a deplorable condition leave these places after a single season in a notably improved state, and often recover in a manner quite unexpected.

Gentlemen, the sympathetic dyspepsia which so often accompanies uterine affections, such as displacement of the womb associated with chronic catarrhal inflammation, is often cured simultaneously with the spontaneous cure of the uterine affection. In these cases, local treatment, cauterizations of the neck, for example, which will modify the catarrh when dependent upon ulceration, properly applied bandages, hypogastric bands, more rarely the use of pessaries—local treatment in fact—will prove very useful, not only for the uterine lesion, but also for the gastric symptoms which depend upon it. These means, however, are not in general sufficient in themselves; it is necessary to have recourse to general treatment, in which an important place must be assigned to sea-bathing and hydrotherapy. You will sometimes see women restored, as it were from death to life, after not more than eight or ten days of sea-bathing. But then it is essential that the sea-baths be taken in a proper manner: by a proper manner, I mean, that they be of short duration, at the utmost not exceeding five minutes. The best way of proceeding is to administer the sea-bath *à la lame*. You all know what this means: an attendant taking the patient in his arms, presents her five or six times in succession to the wave, which passes over her. Powerful reaction succeeds this rapid immersion; the temperature of the skin rises. Sometimes after the fourth or fifth bath, the skin becomes the seat of a peculiar eruption, to which the name of maritime urticaria (*urticaire maritime*) has been given. This reaction produces a wholesome derivative action upon the internal organs, as well as a salutary influence upon the digestive apparatus: the gastric functions become normal, the appetite improves, and the dyspeptic symptoms disappear. Simultaneously with these beneficial changes, the uterine lesions likewise improve, the catarrhal affection ceases, and the uterus loses its morbid susceptibility. The general health becomes better, the patient acquires tone, and is able to bear those variations of temperature which formerly occasioned uterine catarrh, just as they might occasion in others pulmonary catarrh, coryza, or sore throat.

Similar beneficial results may be obtained from a course of hydrotherapy conducted at a hydrotherapic establishment; or, if that cannot be obtained, by hydrotherapy carried out at home in accordance with the plan which I have already described to you.

Before concluding this long series, I have still some words to say upon that form of dyspepsia associated with sluggishness of the large intestine and obstinate constipation.

A remedy lauded by Bretonneau, I mean belladonna, is marvellously efficacious in cases of this description. We must begin by prescribing it in very small doses: a centigramme of the extract incorporated with the same quantity of the powder of the leaves, may be administered, in pill or powder, morning or evening. If the constipation does not yield after one or two days, the dose of belladonna may be gradually increased, according to circumstances, to one, three, four, or five centigrammes; but five centigrammes in one day, must never be exceeded. Thus administered, belladonna is perhaps the most active remedy with which I am acquainted in this kind of dyspepsia. It is generally sufficient to produce regular stools, and, at the same time, to re-establish the digestive functions so thoroughly, that individuals who had fallen into a state of deplorable debility and emaciation, rapidly regain strength and plumpness. The remedy, however, acts in these cases only in an indirect manner, that is to say, by restoring to the large intestine its lost activity: but this activity is communicated synergetically to the other parts of the digestive tube, and thus it is, that the stomach regains its original energy.

When the belladonna proves insufficient, its operation may be assisted by giving the patient every evening a teaspoonful of castor oil, simultaneously with the belladonna: the castor oil may be administered in a capsule of gelatin. When the bowels are regularly open, these means may be discontinued. This treatment, I repeat, is sovereign in the cases now under consideration: but it is in an especial manner sovereign, as a means of restoring regularity to the disordered functions. To secure a continuance of these beneficial effects, the co-operation of the patient is required. In the acts of animal life, habit plays an important part. Upon this subject might be written a long and interesting chapter of general medicine. You know, that according to country and social condition, persons become habituated to eat at regular hours, and except at these hours do not feel the want of food. In the same way, the large intestine may become accustomed periodically to contract itself, and the bladder to discharge its contents at regular times—which times may be at pleasure approximated or made more distant. This is a fact which may be profitably borne in mind in the treatment of such cases as we are now considering. Patients affected with obstinate constipation dependent upon sluggishness of the intestine, ought to go regularly to the closet every day at the same hour; at first, their efforts may be unavailing, but they must nevertheless persevere, and if they do so, the results will ultimately prove satisfactory.

Should these means—should the belladonna treatment—prove inadequate, injections are permissible. But, if used, it is essential that the injections should consist of cold water, and be administered in very small quantity: injections of tepid water ought to be expressly prohibited, for their use ultimately leads to an increase of that atony of the intestine which we are endeavoring to combat.

Let us suppose that the constipation has resisted the use of all these means; it is then necessary to have recourse to purgatives, particularly to aloetic preparations, such as dinner pills, *grains de santé*, and similar remedies. Immediately before eating, from one to four of these or such like pills may be taken. Rhubarb in a dose of from fifty centigrammes to a gramme may be advantageously substituted for the pills, without causing diarrhœa, and with the effect of producing only one stool regularly in the twenty-four hours.

In these cases, certain mineral waters are likewise indicated. I refer to the waters of Seidschütz and Sedlitz in Bohemia, which contain sulphate of magnesia, as well as to Forbach, in the department of the Moselle, whither patients are sent for one or two seasons.

I intend afterwards to treat more fully the important question of constipation, which to-day I cannot do more than touch upon in a sketchy manner.

Gentlemen, I beg your attention for a few minutes whilst I speak of a new treatment which you have seen me employ with decided benefit in a very severe case of dyspepsia in St. Bernard Ward.

The anatomical integrity of the blood, if I may use such an expression, is a condition essential to the normal performance of the functions. Whenever the blood is seriously altered, either in the proportion of its constituents, or by the addition of some septic or toxic principle, it necessarily follows that each organ is modified in intimate texture, or at least in respect of nutrition; it also happens that each organic molecule in contact with vitiated blood no longer bears a normal relation to the nutrient fluid, and that the due performance of the functions is in consequence seriously impaired. Hence arise the malnutrition of tissues, and the disturbance of the organic functions.

To speak only of anæmia: we understand perfectly well that the blood deprived of one essential constituent is no longer sufficient for the formation of tissues, and that the nervous centres of animal and organic life are destitute of their natural excitement, and can no longer exert upon the organs which they supply an influence which they have lost. It is evident then that the digestive functions are disturbed, because the tissues are no longer in a normal state, because the ganglionic nervous system no longer regularly supplies the required influence of nervous power, and because the organs themselves, even if in a state of perfect anatomical soundness, cannot extract from the blood all the materials which ought to enter into the secretions.

When anæmia is associated with chlorosis properly so called, the preparations of iron prove rapidly beneficial: even in cases in which iron is badly borne for a few days, it generally at last triumphs over the disease. But the anæmia which follows excessive uterine hemorrhage, particularly that which slowly supervenes as a consequence of great physical fatigue, protracted moral suffering, excess in venereal pleasures, bad feeding, too protracted lactation, a continuance in unfavorable conditions, that anæmia so common in hospitals, particularly in very young girls who have become mothers, and attempt to perform their maternal duties, though ill-fed and hard worked—anæmia of this kind is not in general improved by ferruginous remedies; and, as it is accompanied by excessive debility, and insurmountable dislike of food, we cannot always restore the aptitudes of the stomach, the organ to which, in the first instance, we make our appeal, knowing that good nutrition is the primary condition essential to recovery. Whatever we do, the patients die oppressed by an insurmountable loathing of food, burning fever, and ardent thirst. On anatomical examination of the bodies, nothing morbid can be detected, except universal paleness of the tissues, and a colorless condition of the blood.

Gentlemen, let me relate to you the history of an illustrative case.

On the 5th January, 1864, a young woman, 25 years of age, was admitted to St. Bernard Ward. She had been confined three months previously, under the very deplorable moral and hygienic conditions commonly met with in girl-mothers [*filles mères*]. Poor and isolated, she was compelled to engage in a toil as ceaseless and fatiguing as it was ill-paid: besides, she suckled her child, and was without sufficient food: she was thus the victim of twofold exhaustion. She gradually fell into a state of extreme anæmia and debility, which it is not easy to depict.

Her emaciation was excessive; her debility was extreme. The skin presented as pale and cachectic an appearance as it is possible to conceive. She coughed: she had constant fever, which redoubled its severity at night; and her appearance, as well as the general phenomena of her case, indicated the existence of pulmonary phthisis. It was, therefore, with extreme astonishment that I could not discover any abnormal sounds in the chest, and that I heard the natural vesicular murmur in every part of the lungs from base to apex.

The most minute investigation, moreover, revealed nothing morbid in any other part of the organism. It was therefore necessary to admit that the case was one of *febris alba virginum.*

From the 5th to the 10th of January, the hectic fever continued, and, in spite of all our efforts to prevent it, the strength progressively declined. The following is a description of the patient's condition on the 10th January: the pulse in the morning was 120, and in the evening 130; the fever was ardent; the skin was dry and burning: diarrhœa alternated with constipation. Nothing abnormal was met with on auscultation. On the 11th, the patient had the initiatory symptoms of small-pox—rachialgia and bilious vomiting. The pulse rose to 140. On the 12th, thirty-six hours after these prodromata, a few scattered papules appeared, as anæmic as the skin itself on which they were developed, and more sensible to touch than to sight. On the 13th the papules continued in the same state, and were not surrounded by any areola. There was great prostration. On the 14th, the patient died. During life, the blood was examined, and found to contain very few white corpuscles.

At the autopsy, the organs were found generally in a colorless condition. The heart was small, and exceedingly anæmic. There was no trace of tubercles in the lungs, which, as in severe cases of fever, were congested thoughout their two inferior and posterior thirds. The spleen was voluminous, tense, hard, and of a hepatized appearance: the Malpighian bodies were evidently of increased size. The liver was bulky, and colorless.

I have no doubt that this woman sank from anæmic cachexia, and that the attack of small-pox was only the immediate cause of death. The organism was in so exhausted a state, from the daily loss to which it was subjected, and the want of reparative aliment, that, as in the animals experimented upon by Chossat,* a degree of inanition had been attained which rendered death inevitable.

I have already, gentlemen, often had occasion to deplore my inability to be of any use in cases of this description, and I long sought vainly for a weapon to serve me in the circumstances. I am indebted to my friend and hospital colleague, Dr. Demarquay, for having enabled me in some cases to restore women to life, whom I had been looking upon as virtually dead; and who were in precisely the same position as the young patient whose sad history I have just related to you.

The curative agency to which I refer is the respiration of pure oxygen gas.† You have seen in our clinical wards the successful results of this treatment, results which have not astonished you less than they have astonished me.

The results were as remarkable when looked at from a therapeutic point of view as they were physiologically unexpected and paradoxical.

The woman whose case I am now going to describe was admitted on the 1st April, 1864; and is now a patient occupying bed 7 of St. Bernard Ward. She is 22 years of age. She had been, like the other woman whose case I have just described, recently confined: like her also, she was anæmic, and exhausted by lactation. Her face was exactly like that of a dead person. The first step in the treatment was to separate the child from the mother. There was, however, no improvement between the 1st and 14th April, that is to say, during the first fourteen days in which she did not give suck. On the contrary, there was a continuance of the fever: the pulse ranged between 120 and 130: the skin was dry and hot: and the debility went on increasing. So great was this debility, that the patient could not sit up in bed without fainting, and on this account, auscultation was almost impossible. Nevertheless, it was ascertained with certainty that the lungs were healthy. As there was no tubercular disease, as tonics and ferruginous remedies had failed, and as there was complete anorexia, I resolved to try the effects of inhaling oxygen, with a view to restore appetite and promote digestion. The patient commenced this new treatment upon the 14th, but was so weak, that after the second inspiration of this gas she became insensible from the effort made in inhalation. However, I recommended her to persist, and to inhale during the day, at intervals, a quantity amounting to five or six litres. For three days, she inhaled much less than that quantity, and during that period, the amelioration was not very perceptible.

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* CHOSSAT: Recherches Expérimentales sur l'Inanition. Paris, 1843.

† DEMARQUAY: Essai de Pneumatologie Médicale; Recherches Physiologiques, Cliniques, et Thèrapeutiques sur les Gaz. Paris, 1866.

But, from the 19th, the patient could sit up in bed with impunity; and could eat a little. The pulse was not more than 104 in the minute. On the 21st, she was able to leave her bed for an hour: she asked for food, particularly for vegetables. The pulse was not more than 92. The skin was cool. Upon the 24th, the pulse was 80. On that day, the patient went down into the garden and ate voraciously. To-day, 30th April, and for the four preceding days, the pulse has ranged between 72 and 80. The young woman feels herself so well that she wishes to leave the hospital. I have however asked her to remain, telling her that her cure is not yet complete. In fact, she still continues pale, and the fibre has evidently regained its tonicity to a greater extent than the blood has regained its normal constitution.

One strange and unexpected phenomenon which accompanies the inspiration of oxygen is the production within the chest of an agreeable sensation of coolness by each inspiration of the gas. The pulse, being 84 on the 30th April, when the patient began to inhale the ten litres of gas, had fallen to 76 by the time the inhalation was completed, and remained at that point during the remainder of the hospital visit. The pulse becomes thready after three inspirations of oxygen, and so continues for the two or three minutes of the duration of the operation. These facts prove—were proof required—that hæmatosis is not accomplished in the lungs, but in the general capillaries—that during the inspiratory act, there is a simple exchange of gas in the organs called the organs of hæmatosis—and that, finally, the oxygen acts almost immediately upon the vaso-motory nervous system, producing contraction of the vessels.

I have now terminated my remarks upon dyspepsia. Let me again repeat, that I have avoided attempting to give more than one short chapter of a long history. In pointing out to you in a very summary manner the diverse forms, and the still more varied treatment of dyspepsia, my only object has been to show you, and smooth for you the right road, so that you may be able to follow it in your practice. However incomplete the notions may be which I have attempted to present, they will at least cause you to think. Never forget that dyspepsia will present itself to you, under aspects and under forms the most varied. According to diversity in symptoms and individuals, it demands remedies, for employing which, general indications can hardly be formulated in a didactic manner, as their application is subordinate to a host of circumstances, impossible to foresee, impossible to point out in advance, and the appreciation of which depends entirely upon the tact of the practitioner.

LECTURE LXIX.

CHRONIC GASTRITIS.

Existence of Chronic Gastritis improperly denied in the present day.—Pituitous Vomiting attributable to it.

Gentlemen: In the present day, to pronounce the word *gastritis* is considered equivalent to the intimation of a desire to renew useless controversies raised by a defunct school. Even during the lifetime of Broussais, the very existence of his famous "gastritis," which he attempted to instal as the originator of all other diseases, was denied by the majority of physicians. It has not yet recovered from that negation. Frequent the different services of our hospitals, and I question whether, in a long space of time, you will even hear gastritis named!

Gastritis is nevertheless a real disease. I am speaking, you understand, of idiopathic gastritis; for no one denies, that gastritis is produced by the ingestion of certain poisons. There is also such a thing as gastritis spontaneously developing itself. I admit that it is a rare, a very rare affection. It is easy to understand that it should be rare. The stomach, from the nature of the functions with which it is intrusted, ought to be so organized as to resist energetically the causes of inflammation, which it may have daily to encounter from irritating alimentary substances. Moreover, a considerable degree of excitation is often indispensable to bring into play the functions of the organ. However enduring the stomach may be, its tolerance has limits. To pass these limits, it is necessary that (according to individual tendencies) the irritating causes should be somewhat violent, more protracted, and more recurrent than when they act upon organs of greater susceptibility.

Consequently, that irritating causes produce any serious result on the stomach, they must be very violent and deepseated.

Pathological anatomy has made us acquainted with the lesions which characterize acute and chronic gastritis.

After saying a few words upon chronic inflammation of the stomach, I shall proceed to consider the cases now particularly under our observation.

I shall not discuss at length the different morbid colors presented by the mucous membrane of the stomach: I shall only state that gray, slate-color, or brown, seem chiefly to belong to chronic inflammation: that the morbid color appears in the form of spots, sometimes round, sometimes irregularly shaped, and sometimes uniformly spread over a greater or less surface. The morbid tint is in some cases black, as when the inflammation is produced by certain poisons, but the shade of black is never so deep as in cases of poisoning. Let me caution you against confounding the appearance of which I am speaking with the dark hue so frequently the result of cadaveric imbibition.

But the most essentially characteristic lesion of chronic gastritis is the alteration and hypertrophy of the coats of the stomach. Sometimes the mucous membrane alone, and at other times all the coats, mucous, cellular, and muscular, are thickened: this thickening may be partial, or it may be more decided in particular parts. The stomach then assumes an aspect similar to that presented by the bladder, when it has been the seat of chronic inflammation of that description which is called columnar bladder [*vessie à colonnes*]. This is rarely seen: nevertheless, here is an example derived from our own clinical service.

At the beginning of the year 1856, a man, aged 50, was admitted to our wards, who told us, that for some time, he had vomited all his food. He stated, that he had lost more than 40 pounds in weight during three months. He likewise complained of obstinate constipation. The disorders of digestion and nutrition had never been accompanied by fever. From my very first visit, I was struck by a fact, which appeared to negative the idea of cancer of the stomach, which was, I confess, my first impression: the complexion of the patient was remarkably fresh.

Nevertheless he had incessant vomiting. Irrespectively of meals, irrespectively of the ingestion of alimentary matters, this man vomited a large quantity of a glairy matter, similar to that contained in the urine of persons affected with catarrh of the bladder. Moreover, the glairy matter was sometimes mixed with blackish matter resembling suspended soot, like the melanotic vomiting which generally characterizes cancer of the stomach.

Upon examining with greater attention, and upon several different occasions, almost every day, the epigastric region, I was unable to detect the presence of any circumscribed tumor. However, when, with my hand upon the pit of the stomach, I told the patient to take a deep inspiration, I felt under my fingers a sort of rubbing, which appeared to me to be produced by a stomach which was indurated. Notwithstanding the absence of any appreciable tumor, my diagnosis was—carcinoma of the stomach. This induration, as to the existence of which I had no doubt, the incessant vomiting, the presence of black melanotic matter in the fluid ejected by the mouth, both after meals and during the intervals between meals, and the notable emaciation of the patient, justified my conclusion, although the pink color of the integument was essentially different from the cachectic pale yellow hue of persons affected with cancer. The disease made rapid progress, the emaciation increased rapidly, as the patient could not be nourished; and ere long death occurred.

On opening the body, my attention was immediately directed to the stomach. It was diminished in volume, and its interior presented an appearance exactly similar to that of a urinary bladder which had long been the seat of chronic catarrh. I found no trace of a tumor, but the mucous membrane was exceedingly hypertrophied, and was so blended with the cellular coat as to adhere intimately to it by fibro-plastic tissue. The mucous membrane seemed to be destroyed. At some points, the walls of the organ were two and a half centimetres in thickness.

I begged my distinguished colleague, Professor Charles Robin, to examine the stomach carefully, and to find out whether it presented any elements of cancerous disease. In giving me an account of his examination, he informed me that he had several times seen stomachs in a similar condition, that is to say, presenting only hypertrophy of the fibrous structure, associated with nearly total destruction of the mucous coat, and presenting no trace of heteromorphous products.

Here, then, gentlemen, is a case in which the autopsy placed beyond doubt the existence of chronic gastritis. Our patient who leaves the hospital to-day, and whose case has originated this lecture, evidently had similar lesions, although no doubt they were less decided and less advanced.

This man was admitted about five months ago. His very emaciated condition, and the pale yellow tinge of his skin, showed that he was suffering from extreme cachexia. On admission, he stated that the beginning of his symptoms dated back six months or more. He had in the first instance loss of appetite, and soon afterwards vomiting, which for the last three months had habitually occurred after ingestion of the smallest quantity of food. Diarrhœa was then added to the other symptoms; and it alternated with invincible constipation. The general debility, and the great emaciation, were the results of disordered digestion.

In endeavoring to trace the malady to its source, we could only ascertain that the patient had lived under the most unfavorable hygienic conditions. He pursued the occupation of an itinerant seller of neckties, by which, for a long time, he had been scarcely able to provide a bare existence. He was ill fed, and probably worse lodged. He assured us that he never committed any drinking excesses.

The matter vomited was chiefly of a glairy, stringy, and glutinous character; it was sometimes ejected so copiously that the spittoon was filled within the twenty-four hours.

Upon examining the epigastric region with the greatest care, I could not detect a tumor either in the situation of the great or the small curvature of the stomach; nevertheless, the excessive emaciation of the patient, his cachectic hue, his loss of appetite, and the constancy with which vomiting was occasioned by the ingestion of food, led me to write upon the sheet, "Cancer of the stomach" as my diagnosis. At the same time, I had not then formed an absolute opinion, and was waiting for the result of treatment, to see whether or not the view which I took of the case was right or wrong. Gentlemen, you have very often found me, under similarly embarrassing circumstances, postponing my diagnosis, and refraining from announcing it absolutely, till I had tried several different plans of treatment. It is not, as some might suppose, from any desire to shirk trouble, by avoiding an exact diagnosis at the first examination, that I act in this manner; on the contrary, I use from the first my utmost endeavors to make my diagnosis as rigorously accurate as possible. There are cases, however, in which hesitation is allowable; and there are others in which, although an almost absolute certainty has been obtained, there is still room for hoping that we may be deceived, cases, therefore, in which we look out for new elements to guide our judgment and correct previously formed opinions. The facts of the case now under observation will enable you to understand clearly the explanations I wish to make to you on this point.

From the first, our patient seemed to labor under cancer of the stomach. This diagnosis necessitated my avowal of inability to do any good, and condemned me to absolute forbearance from active treatment; because experience had told me, that cancer of the stomach is an incurable disease, in which it is not only useless but even injurious to employ energetic measures. My only remaining hope was, that I had been deceived. Without, therefore, ceasing to believe that the case might be cancer of the stomach, I endeavored to discover whether there was anything in the symptoms or progress of the disease, upon which I could lay hold, as possible signs of a curable malady.

The phenomena which I observed seemed to present a certain analogy to those presented by the patient whose case I have just laid before you; and I asked myself the question, whether this case was not also chronic gastritis. I clung to the possibility of its being so, and directed my treatment as if it were that disease.

To combat the vomiting of glairy matter, which was the predominating symptom, I had recourse to lime-water, while at the same time I administered, according to the method of Graves, very small doses of opium.

The first trial of this plan proved successful. The vomiting became less profuse; and the patient began to take a little food. Diarrhœa, however, having supervened, I substituted for the lime-water and opium, nitrate of silver, giving from eight to ten centigrammes during the day, in the form of pill, each pill containing one centigramme of the nitrate.

I pursued this plan of treatment, exactly as if I had been endeavoring to modify a catarrhal affection of the mucous membrane of the bladder or pharynx by injections or caustic applications.

During six weeks or two months, I thus combined my therapeutic measures, alternating opium and alkalies with nitrate of silver. Under the influence of this treatment I have had the satisfication to see so great an amelioration in the symptoms, that the patient is able to-day to leave the hospital with restored appetite and good digestion, and notably increased plumpness—in a word, in a state of health relatively most satisfactory.

Although this case, which I consider to be an example of chronic gastritis, leaves some uncertainty as to its nature, it is not the less valuable to you as a source of instruction. Granting that we have only arrived at an imperfect result, that result, so far as it goes, is good. When I come to speak of diarrhœa, you will see how often that affection is the consequence of an intestinal inflammation similar to that which attacks the bronchial tubes, and supervenes under the influence of the same causes. Very recently I saw, in my consulting-room, a young lady, presenting all the symptoms of chronic gastritis, with, however, very frequent attacks of a more acute character. Whenever she was exposed to cold, she had these acute attacks, and then there immediately supervened vomiting of glairy matter. It is easy to understand why it should be so; nay, gentlemen, let me add, that I do not see how it could be otherwise. A thousand times we see exposure to cold produce or aggravate cystitis, uterine catarrh, pulmonary catarrh, catarrh of the mucous membrane of the nasal fossæ, and why should we expect that the internal membrane of the stomach should alone be exempt from affections to which almost all other mucous membranes are subject?

It is in this form of chronic gastritis, that sulphurous baths, hydrotherapy, and sea-bathing, are pre-eminently useful: it is in such cases, too, that patients, after annually making useless journeys to Carlsbad, Vichy, and Plombières, are speedily cured by the Luchon waters, sea-bathing, or hydrotherapy.

I am unwilling to leave this subject, gentlemen, without saying a word upon the affection called vomiting of phlegm [*la pituite*]. Under this name is generally understood, a disease characterized by vomiting of glairy matter, the vomiting occurring chiefly in the morning before eating. The quantity of mucus ejected is sometimes very considerable. I am not speaking, gentlemen, of that kind of glairy salivation, which is so often the precursor of vomiting, and which is evidently the product of the salivary glands and the muciparous glands of the pharynx and mouth, but is not vomiting: what I refer to is glairy vomiting. This is most frequently observed in persons addicted to excess in alcoholic drinks,* and is usually coincident with great want of appetite: it is not, however, at all incompatible with obesity. I have always considered this kind of vomiting as a sign of chronic gastritis, and I place it in the same category with the vesical catarrh which follows acute cystitis, and the glairy stools resembling the spawn of frogs, so frequently observed after acute colitis and dysentery.

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* See the Lecture upon Alcoholism, *supra*, p. 33.

I know quite well that certain persons may have this pituitous vomiting without any apparent deterioration of health; but do you not know many men with chronic cystitis, whose general health is unexceptionable, and many patients with bronchial catarrh, and profuse glairy expectoration, who are invalids only in a small degree? Can we on that account deny that there exists an inflammatory affection of the vesical or the bronchial mucous membrane?

The treatment of pituitous vomiting in no respect differs from the treatment of chronic gastritis.

LECTURE LXX.

SIMPLE CHRONIC ULCER OF THE STOMACH.

Gastralgia with Stitch in the Ensiform and Rachidian Regions is not exclusively a Symptom of Simple Ulcer of the Stomach.—It may be absent in this affection, and it may also be met with in Diseases of the Stomach of very different Characters.—The same is true in respect of Hemorrhage from the Stomach and Intestines independent of Organic Change (in supplementary Hæmatemesis, for example), and in Chronic Gastritis.—Hemorrhage, a character common to Simple and Cancerous Ulceration, may be absent.—In Cancer, Hemorrhage is sometimes as profuse as in Simple Ulceration, although generally the Hæmatemesis of Cancer is less than the Hæmatemesis of Simple Ulceration.—The positive Diagnosis of Simple Ulceration is enveloped in much obscurity.—Treatment.

GENTLEMEN: In bed 8 of St. Bernard Ward, there lies a woman, aged 34, whose history is exceedingly interesting, but which I can only recapitulate in a summary manner. According to her statement, her disease is of old standing, and the result of a blow on the stomach. I do not attach as much importance to this blow as she does. There are two other facts, however, which seem to me to be of special value. In early life, this woman was periodically subject to bleeding piles. This is in itself a somewhat curious symptom, remarkable to this extent at least, that it is seldom met with in young women, at least in our country. She afterwards had periodical headaches, which came on every week, with the usual nausea and uncomfortable feelings; they continued to recur till she became pregnant a year ago. At that time, her circumstances became so bad that she scarcely had the means of procuring a bare sustenance; she was moreover illtreated by the man with whom she lived; and had a severe fall when at the third month of her pregnancy. She did not at the time of its occurrence experience any very remarkable effect from this accident; but on the following day, was seized with bleeding from the nose, and, some hours later, with rather profuse vomiting of blood, preceded by a peculiar feeling of distress in the region of the stomach. The epistaxis complicated the diagnosis, for it became a question whether the blood which she vomited came directly from the stomach, or whether it was not derived primarily from the nasal fossæ—being first swallowed and then vomited. There were other symptoms, however, which elucidated this point. The patient complained of very violent boring pain in the stomach, resembling the sensation which she fancied might be produced by a stake pushed through the ensiform cartilage into the stomach. This pain radiated through the corresponding dorsal region, and presented all the characteristics of the pain which Dr. Cruveilhier attributes to simple ulcer. She miscarried, had profuse diarrhœa for three months, and upon one occasion passed blood by stool. The diarrhœa ceased, but the pain in the stomach became more acute. Every four or five days, sometimes more and sometimes less frequently, she vomited profusely a liquid resembling coffee-grounds, or, to use a more exact comparison, soot dissolved in water. Probably the stools were of a black color; but upon this point we obtained no information.

When I first saw this patient, I was struck with her extreme emaciation, sunken eyes, and deep yellow tinge of skin. There was nothing, however, distinctive in this color of the skin. She told us that her skin was naturally brown; so that it was difficult to appreciate how much of the cutaneous appearance was respectively dependent upon anæmia and cancerous cachexia.

She complained of a total want of appetite, and her disgust for food was increased by the dread of arousing pain by ingestion of the smallest quantity of aliment. Pressure over the pit of the stomach occasioned pain.

The question was, whether these symptoms depended upon cancer of the stomach, upon simple ulcer of the stomach, or merely upon supplementary hemorrhage. In relation to the last point, remember that I told you that this woman in her early youth had been subject to bleeding piles. Give special attention to that fact.

It is not uncommon for men at the period of puberty and adolescence to have periodical epistaxis, and at a more mature age periodical bleeding from piles. Now, the fact that a flux of this nature occurred in our patient, may indicate an unusual tendency to hemorrhage. This individual passed from infancy to puberty, when the menstrual function was established; the bleeding from the piles then ceased, and headaches supervened, which continued to torment her for twenty years, but did not recur from the time that she first became pregnant. It was then that the gastro-intestinal hemorrhages supervened. Is there not ground for supposing that these hemorrhages were analogous to the former hæmorrhoidal fluxes which had been replaced by the menstrual uterine flux? May it not be asked whether the menstrual flux being suppressed consequent upon pregnancy, the hemorrhagic tendency was manifested in the stomach?

Admitting that this hemorrhagic tendency did not constitute the whole disease, it certainly played a great part in it. The frequent return of the symptoms without that periodicity usual in supplementary hemorrhages, the boring pains by which they were accompanied, their predominating seat being the ensiform cartilage and corresponding dorsal region, disposed me to adopt the idea that there was a serious lesion of the stomach; and in what I saw I had no difficulty in recognizing the symptoms attributed to simple ulcer.

The age of the patient, the total absence of any tumor or induration in the epigastric region (which the excessive emaciation and the great flaccidity of the abdominal parietes enabled me thoroughly to explore), led me to conclude that cancer did not exist. I adopted the more willingly my diagnosis of *simple ulcer of the stomach* that I felt of how little use I could be in the event of this woman's malady being carcinomatous. My powerlessness for good would in such a case have been all the greater that the hemorrhages recurred twice or thrice a week, a frequency of recurrence which did not allow me to entertain the hope of being able to prevent them.

Adopting, therefore, the view that the patient was suffering from simple ulcer, I at once had recourse to the use of preparations of nitrate of silver. I began by giving five centigrammes during the day, divided into five pills; at the same time, with a view to assuage the pain, I prescribed the extract of opium in pills containing one centigramme, of which she never had occasion to take more than four in the twenty-four hours. My object was to act upon the visceral lesion by the nitrate of silver, just as I should have endeavored to act upon an ulceration of the pharynx or skin by the solid nitrate.

The symptoms moderated after some days of this treatment; there was no return of the hemorrhage; the pain abated; and digestion, till then painful, and even impossible, became normal. I gradually diminished the dose, but did not discontinue the opium and nitrate of silver. Even when there was good reason to suppose that the cure was complete, the patient continued to take every day a pill of opium and a pill of nitrate of silver. As she had diarrhœa I likewise gave her a gramme daily of the trisnitrate of bismuth.

This treatment, upon the supposition that the hæmatemesis did not depend on the existence of a simple ulcer, and was supplementary to the menstrual flux, could do no harm, and might prove beneficial. It would tend by topical action to modify the congestion and abnormal condition of the surface of the stomach.

Whether it arose from the treatment, or simply from the improved regimen and better hygienic conditions of the hospital as compared with her home, it is certain that the black vomiting and pains ceased, that the appetite, digestion, and menstruation became normal, and, in fact, that the recovery was complete. When she left the hospital, she had regained flesh to a great extent: she left at her own request, feeling that her health was sufficiently restored to enable her to resume her usual work.

In connection with this case, which possesses more than one interesting feature, I shall now mention the case of a man who occupied bed 17 of St. Agnes Ward. It is more interesting than the former case, in this sense, that the autopsy disclosed a lesion of the stomach, the diagnosis of which had proved embarrassing during life.

The patient was thirty-seven years of age, in vigorous health, and by occupation engaged as a laborer in forming earthworks. He stated that three or four months before admission to the Hôtel-Dieu, he had vomited an enormous quantity of blood, and that for two or three consecutive days his stools had resembled tar in color and consistence. From that time, he lost strength: digestion was badly performed; but he affirmed—and this is a point to which I specially direct your attention—that he had never had pains in the stomach. Digestion became more laborious than before: after a certain time, though he continued to eat as usual, he felt, to use his own expression, that the food did not go through him so well. After vomiting the blood, he often suffered from a general feeling of discomfort: he found that the resuming of his work was too much for him. He was pale, short-breathed, and panted upon the least exertion.

Three or four months had passed without his experiencing anything noteworthy in his condition in addition to the symptoms now described; when, some days before his admission to hospital, he had a new attack of hemorrhage from the mouth, followed by black stools. During the two succeeding days, the hemorrhage recurred at short intervals; and he died on the second day, from an attack in which he lost several litres of blood. These vomitings of blood were accompanied by melanotic stools, and every day the vessel (which I ordered to be kept for examination) contained from a quarter to half a litre, and sometimes even a litre, of matter resembling tar. It is unnecessary to add, that an extreme degree of anæmia was the result of these great losses of blood. The skin of the patient had that peculiar tint of old white wax, which is to be seen in women exhausted by profuse hemorrhage.

In exploring with very great care the region of the stomach, I was unable to find the smallest trace of a tumor. The patient, when interrogated, stated that he swallowed food with perfect facility; and this statement was of itself sufficient to exclude the idea of his having carcinoma of the cardiac extremity of the stomach, which on account of the deep situation of the parts would have escaped discovery by manual examination. There might, however, be a tumor near the smaller curvature, or at the bottom of the large pouch, and which, consequently, we should also be unable to feel.

Nevertheless, the attacks of hæmatemesis, the great embarrassment experienced during the first stage of digestion, led me to conclude that there was cancer. There was one element wanting in this diagnosis, viz., pain in the epigastric region; for the patient had never complained of more than discomfort. Let me add, however, that pain is a diagnostic sign which is often absent in cancer of the stomach.

At the autopsy, the stomach was found full of blood. At about from two to three centimetres from the pyloric orifice, there was a depressed velvety surface bounded by an elevated edge, about the size of a two-franc piece. In the centre were seen two open mouths of arteries, sufficiently large to admit a small probe; and one of them was plugged by a clot. These vessels had evidently been the source of the hemorrhage; and I may remark in passing, that this is one of the most unusual ways in which hæmatemesis is produced: I shall afterwards describe to you its ordinary manner of production. It was, however, the microscopic examination which enabled us to detect the error in diagnosis: there was no trace of cancer—the lesion was simple chronic ulcer of the stomach.

At a time when the researches of my scientific colleague, Dr. Cruveilhier,* imparts fresh interest to this important question, I cannot neglect the opportunity afforded by these cases of speaking to you about an affection, the reality of which was for a long time disputed.

To Dr. Cruveilhier unquestionably belongs the merit of having first described simple chronic ulcer of the stomach, *as a disease, special in its nature, and quite distinct from cancer of the stomach, with which it had till then been confounded.* In 1830, that is to say, more than thirty years ago, this distinguished professor devoted a special chapter to simple ulcer; five years later, in the 20th fasciculus of his Atlas of Pathological Anatomy, he added new facts, and new drawings; and in 1838, he published a memoir upon the subject.† In 1839, Professor Rokitansky, of Vienna, published his work;‡ the subject was taken up afresh in 1856 by M. Cruveilhier; and we find in the Archives Générales de Médecine for February and March of that year, the memoir which a short time previously he had communicated to the Institute of France.

While I acknowledge the great service rendered to medical science and art by my honorable colleague by his having established decisively the existence of a disease previously unknown, I cannot help thinking that he has somewhat exaggerated the frequency of the cases in exaggerating the significancy of the symptoms. What are the *symptoms?* At the beginning of the disease, the patients complain of discomfort, of dull pains in the region of the stomach, of a feeling of weight during digestion, which is difficult and painful. The appetite diminishes and is gradually lost: at a certain period of the disease, the repugnance for food is increased by knowing that eating occasions pain. The sensation of weight and fulness arouses and intensifies the acute pains of which I have been speaking, and at last the patients are never at ease, except when they have the stomach empty.

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* CRUVEILHIER: Anatomie Pathologique du Corps Humain. Folio.

† Revue Médicale.

‡ ROKITANSKY: De l'Ulcère Perforant de l'Estomac. Œster. Med. Jahrb., 1839: quoted in Archives Générales de Médecine, 1840.

Though there is observed in some cases, instead of anorexia, an excessive, craving, capricious appetite, and though in some individuals relief from pain is experienced after eating, such cases are exceptional.

Wasting, which rapidly progresses, is the consequence of this defective alimentation. Although the patients continue their usual occupations, they go on day by day losing strength, and visibly become more and more emaciated. At the same time, their mental state is affected; they become sad, melancholy, and easily irritated.

Up to this point, you see that there is nothing characteristic in the signs, which are likewise the signs of many forms of dyspepsia, as well as of incipient cancer. But, at a certain time, the symptoms become complicated with others of real importance, viz., pain and hæmatemesis.

The pain has something special in its character. It is generally confined to the region of the ensiform cartilage. It has a boring character. Or, the pain which is occasioned may be compared to that resulting from a burn, a raw wound, or a violent pinch. It comes in paroxysms, and has exacerbations several times during the day. It is increased by pressing the hand upon the pit of the stomach, and is excited by ingestion of food: supervening sometimes, it is true, a little later, it continues during the whole period of stomachal digestion, and is never so severe as at that time. To this stomachal pain, when it has acquired a high degree of intensity, there is added pain of a similar nature occupying the corresponding dorsal region, that is to say, over the first lumbar vertebra or the three last dorsal vertebræ. In some cases, in place of remaining confined to an epigastric, ensiform, or dorsal situation, the pain radiates upwards, behind the sternum, in the direction of the œsophagus, extends into the intercostal spaces and region of the kidneys.

Let us stop for a moment to consider this symptom, and to inquire whether it possesses the importance which some have attributed to it. Whatever may be its importance, its signification as a diagnostic sign of simple ulcer is far from being absolute: in the first place, it is met with in affections which have nothing in common with the disease we are at present studying; and again, notwithstanding the assertion to the contrary of my honorable colleague, it may be wanting in this affection. No doubt this is a rare occurrence, but that it does occur is conclusively established by the case which I have just related to you, as well as by the history of a case reported by Dr. Louis Gubian to which I shall afterwards refer.*

In cancer of the stomach, we often observe pain in the stomach, characterized in the same manner as the pain accompanying simple ulcer. I therefore think that it is drawing too subtle a distinction to say that the pain of cancer caused by the spasmodic contractions of the stomach is analogous to that caused by contractions of the bladder in retention of urine or by the contractions of the womb in labor; and that the ensiform and dorsal pains of simple ulcer are of an entirely different kind.

According to the admission of M. Cruveilhier himself, the sole peculiarity by which one can distinguish idiopathic gastralgia from the pain which accompanies simple ulceration, is, that in the latter the symptoms are permanent with alternations of exacerbation and remission; while in the former, the pain is temporary, supervening and ceasing abruptly, and being, moreover, at once relieved by opium. You can understand, gentlemen, how impossible it would be to grasp these delicate distinctions as elements of diagnosis. In simple ulceration also, although the pains may not be so acute and boring, M. Cruveilhier distinctly admits that they may be decidedly sharp, boring, and intermittent. In respect of the characteristic sign derived from absolute relief being afforded by opium when the affection is only neuralgia of the stomach, the remedy being inoperative when there is a simple ulcer, I reply, that the most unequivocally idiopathic gastralgia will often resist opium, which on the other hand will moderate or perhaps completely remove pains depending upon simple ulcer or cancer. This we have seen in our patient of St. Bernard Ward.

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* GUBIAN: Gazette Médicale de Lyon, 1856.

I say, therefore, that the pain, however special its character may appear, is insufficient to enable us to distinguish the disease of which I am now speaking.

A similar remark is applicable to black vomit, hæmatemesis, and the melæna usually accompanying it.

In fact, though gastrorrhagia is observed in the majority of cases of simple ulcer, it is a symptom which is sometimes wanting: moreover, it is a phenomenon also belonging to cancer of the stomach, which sometimes shows itself in non-ulcerous chronic gastritis, and is likewise met with in a considerable number of cases, presenting no apparent lesion of the organ which is the seat of the hemorrhage.

This part of the subject possesses so much clinical importance, that I must devote some minutes to its discussion; but before doing so, allow me to say a word upon the mechanism of these hemorrhages. They often originate in the arteries or veins involved in the ulceration, their walls being ulcerated and destroyed. This was the state of matters in our patient of St. Agnes Ward, whose case I have just described. At the autopsy, we found two arteries with open mouths. You can understand that under such conditions, hemorrhage will be more or less profuse: and that it may suddenly destroy life if an important vessel be involved.

Generally, the hemorrhage takes place from small vessels, seen at the autopsy upon the surface of the ulcer, eroded and jagged, some being obstructed by very tenacious solid clots, and others by soft clots which become detached upon the least handling. It is from these small vessels that the hemorrhage proceeds: it is slight, and takes place almost daily, becoming mingled with the food, thus giving rise to black stools and black vomit. However, matters do not always so proceed, and the hemorrhage is frequently the result of a vascular lesion invisible to the unaided eye, situated around the ulceration: in the same way, there is hypersecretion of the gastric fluids. Here, the same thing occurs as in cancer of the stomach; for, in the first instance, in cancerous tumors not yet ulcerated, the hemorrhage does not take place from the surface of the tumor but from the mucous membrane. This is the case in simple ulcer, hæmatemesis being the first symptom of the affection, and continuing when ulceration has not involved the parietes of the vessels.

I have said, gentlemen, that gastrorrhagia is a usual symptom of simple ulcer of the stomach; but I have also said that this symptom is sometimes wanting.

During the year 1858, I saw, in consultation with my friend Dr. Beylard, a young man, an American, who sunk in a few hours, under formidable abdominal symptoms.

In this case it was difficult to obtain an account of the immediate antecedents of the patient. We only learned that he had arrived from London, where he had given himself up for about a week to daily excesses of the table. When I saw him, he had symptoms of cholera, viz., cyanosis, coldness, cramps, absence of pulse, and suppression of urine. Both Dr. Beylard and I were struck with the remarkable circumstance, that there were neither alvine dejections nor vomiting.

The dead body of this young man was taken to America, where by desire of the family an autopsy was made. The physician intrusted with its performance was good enough to send an account of the details to Dr. Beylard, from whom I learned that there had been found indications of subacute peritonitis, occasioned by a perforation in the centre of a simple ulcer of the stomach. Now I was well acquainted with this young man, being in the habit of seeing him daily at the house of his mother, whom I was attending for an affection of the uterus; and he then appeared in the enjoyment of perfect health, digestion being in a perfectly normal condition.

From the absence of characteristic symptoms, and in particular from the absence of hæmatemesis, this case presents a great analogy to the other case which I have just detailed.

Allow me to read to you from the Gazette Médicale de Lyon for 1856 Dr. Louis Gubian's case:

"In number one of the medical clinical ward, there lay, on the 24th August, 1856, Clément Favorain, forty-seven years of age, a stone-hewer. This man, of very so-so constitution, having in a marked manner the lymphatic temperament, and a small amount of intelligence, had always led a morose and pitiable existence. He was first an excavator of earthworks, then a hewer of stones, either in the quarries or on the roads, exposed to all the vicissitudes of the seasons, often without work, ill-fed, drinking only water, either plain or mixed with doctored beverages, taking alcohol very rarely and in very small quantity, and not having aliment sufficient in quantity or adequatively reparative. The appearance of the patient, on his admission to the hospital, denoted a condition of misery and suffering. His face presented a colorless appearance, which he said was usual: and his emaciation dated back for several years.

"He only complained of some pains in the epigastric region, which he had not felt for more than a few days. They were not intense: they were wandering, slightly increased by pressure; and their maximum intensity was not at the ensiform cartilage. For about three months his appetite had diminished; and during that time digestion was slow, difficult, accompanied by bitter, nidorous eructations, tension of the epigastric region, and some flatulent distension of the abdomen. He had not had any kind of vomiting.

"Palpation of the stomach indicated nothing abnormal, nor did it reveal the existence of a tumor.

"The tongue was thick, and a little whitish at the base. The patient had neither appetite, nor disgust for particular kinds of aliment. He had no repugnance to butcher meat. He usually suffered from obstinate constipation.

"Notwithstanding the discomfort which he felt, he had no fever; and had continued his severe and toilsome labor as a stone-breaker.

"From the symptoms of which he complained, there was reason to believe that his malady was simple dyspepsia, and as such it was treated till the 15th September: during that period, no new symptoms supervened. On the 15th September, however, he complained of pain in the abdomen, particularly on the left side, of incontinence of urine, and of œdema of the scrotum. Next day, the face presented a puffy, œdematous appearance. These symptoms alarmed Professor Teissier (of Lyons) who had on that very day resumed charge of the clinical wards. Upon an attentive examination of the organs, there was only discovered dropsical infiltration, which was greatest in the sides, accompanied by acute pain in these regions, particularly in the left side posteriorly, and in the neighborhood of the kidney. The urine, when examined by nitric acid, showed no trace of albumen.

"In the evening, the patient had oppressed breathing; and ere long the mucous râles of the last agony were heard. At this time, the pulse became quick, small, and irregular: afterwards, the circulation became slow, the extremities grew cold; and death occurred during the night.

"At the autopsy, a litre and a half of fluid was found in the peritoneal cavity. There was great thickening of the coats of the stomach in its two inferior thirds: its internal surface had a shrivelled, plaited appearance, presenting elongated elevations formed by very flexuous folds separated by deep depressions, resembling those sometimes seen on the muscular coat of the bladder, and which are known by the name of columnar bladder. Near the cul-de-sac of the stomach, about the middle third of the great curvature, there was a depressed, velvety [*tomenteuse*] ulceration, slightly twisted by the folds of mucous membrane which marked out its circumference by an elevated margin perfectly circular in form. The diameter of the ulcer was about that of a two-franc piece: in depth, it reached the muscular coat, which was fully five or six lines in thickness. This ulcer seemed to be in progress of cicatrization: it was not surrounded by any vascularity. The mucous membrane presented neither marked villosities nor pultaceous nor gelatiniform softening: it was only injected, and slightly hypertrophied, in which latter condition the submucous tissue participated."

I have now laid before you a case in which the symptoms characteristic of ulceration were presented in the first instance, and in which it might erroneously have been concluded that there was cancer of the stomach.

On 10th December, 1863, a woman, aged forty-nine, came into St. Bernard Ward suffering from an abdominal affection. She had fever, greatly altered countenance, and suffered from severe pain in a zone comprised between the two hypochondria and the epigastrium. She had a constant, dry cough, and great oppression of the breathing. On palpation of the abdomen, there was felt a hard, resisting mass, which extended towards the right side, moved with the diaphragm, and was evidently the enlarged liver. The epigastrium was manifestly protuberant: on percussion, a tympanitic sound was elucidated. In the line between the epigastrium and left hypochondrium, there was discovered a hard, oval, pretty regularly shaped mass, which was painful to the touch. Except in this situation, the abdominal walls were tolerably supple. On percussion of the chest, there was perceived a notable dulness about the inferior third on each side. At the base of both lungs, fine subcrepitant râles were heard on auscultation. In these situations, there was an almost bronchial reverberation of the voice.

The following is the history which this woman gave of herself: About nine or ten years ago, she had had vomiting of blood for the first time: it came up in enormous quantities. At this period, she suffered from very acute pains at the pit of the stomach, and digestion was very much out of order. After this, her health was restored; but again, one or two years later, became deranged: she had vomiting of blood, and black matter was ejected. Subsequently to this period, she from time to time vomited her food, having acute pain at the pit of the stomach and in the back: after these attacks all became calm again for an interval, and then the same symptoms reappeared. At last, they ceased to recur; and there seemed to be a return to health and strength. The plumpness also, which had not, however, been much diminished, was restored. The patient thus enjoyed passable health for two or three years, that was till about three months before her admission to the hospital, when she was again seized with vomiting: on this occasion, the matters ejected were not sanguinolent: in the region of the stomach, the pains were violent; and they darted through the diaphragmatic region. Henceforth, she had fever, dyspnœa, and cough. This conjunction of symptoms brought her to our wards.

The difficulties of diagnosis were almost insurmountable in this case. To me it was evident from the woman's story, that she had had ulcer of the stomach. Vomiting of blood, black vomit, acute pain in the epigastrium, the cessation of the symptoms, and the long interval between the times of their recurrence, were all facts in favor of that diagnosis. On the other hand, I found at the pit of the stomach, a soft, indolent, tympanitic tumor, which certainly seemed to be the stomach distended by gas. But then, in the left hypochondrium, there was a hard painful tumor, which might be formed by a cancerous alteration of the stomach. It seemed, moreover, to be independent of the liver, because the soft epigastric tumor was interposed between that organ and the tumor in the left hypochondrium.

There was, however, an element of doubt in the case: I refer to the known antagonism, so to speak, between round ulcer and cancer of the stomach. Be that as it may, the physical signs were of such a character that it was more rational to conclude from them that the disease was cancer. This woman had likewise the signs of inflammation of the diaphragm: respiration was accomplished chiefly by the abdominal muscles, and the morbid physical signs were those of double diaphragmatic pleurisy. There was ground for believing—and in point of fact, I did believe—that the cancer had produced inflammation in its neighborhood which had extended to the diaphragmatic peritoneum, diaphragm, and base of both pleuræ. I was not mistaken as to the existence of inflammation: the parts now named were the seat of inflammation: but its cause was not that which I had supposed.

I shall not recapitulate the different stages of the disease, which, however, I may remark, were very short. You know that the difficulty of breathing went on continually increasing; that palpation of the abdomen became more and more painful; that the patient frequently vomited the fluid as well as the small quantities of solid food which she took; and that at last she died, having had for twenty-four hours preceding death the signs of general peritonitis.

At the autopsy there was found purulent peritonitis, with numerous adhesions matting together the intestines. The liver was greatly enlarged, and presented a marbled appearance: it was throughout the whole of its convex surface adherent to the diaphragm, and by a part of its convex surface it was intimately united to the anterior wall of the stomach. In the situation of the left lobe of the liver there was a purulent pouch circumscribed by thick false membranes, evidently of old date: through a slight fissure in one of them some pus had entered the peritoneum, and had there developed recent inflammation. There was, at the bottom of the purulent pouch, a circular perforation of the anterior wall of the stomach. When this organ was incised in the course of the small curvature, it was at once seen not to be in a cancerous condition; and in a circular line around the pyloric ring was observed a series of ulcerations, three of which were cicatrized, while a fourth, still in ulcerative activity, showed a perforation.

One of the cicatrized ulcers was very regularly circular, another was oval: both were remarkable for the induration and callous thickening of their surroundings: the tissue of the cicatrix which formed this elevated surface was fibrous, and resisted, but did not creak under the scalpel like scirrhus, of which it had, moreover, neither the appearance nor the structure. The size of the perforating ulcer was about that of a two-franc piece. It had the form of a crater; the walls were somewhat thin, and, at the point of perforation, there was destruction of all the coats of the stomach. The perforation resembled a lentil in form and diameter. It was situated in the neighborhood of the sharp anterior border of the liver, so that the abdominal wall was glued to it by false membrane, which for a certain time had prevented the contents of the stomach from entering the peritoneal cavity. On the other hand, the adhesive inflammation extended round the transverse colon: and it was the arch of the colon enormously dilated, placed in the epigastric region, in front of the stomach, which formed the soft and tympanitic projecting tumor, mistaken during life for the stomach itself. The colon was, moreover, projecting between the two lobes of the liver in such a way that at the right side one could feel, through the abdominal parietes, the right lobe of the liver—at the epigastrium, the arch of the colon—and at the left side, the left lobe of the liver, which might have been considered as an abnormal tumor, although there was a sonorous tympanitic space between the solid mass on the right (which was evidently the liver), and the indeterminate mass on the left. Now, from the nature of the gastric symptoms, it was very natural to infer that there was a cancerous tumor of the stomach. To conclude the description of the autopsy: There was a diaphragmatic pleurisy, and adhesions closely uniting the base of the lungs to the pleura: there was no effusion: the inferior third of both lungs were congested and solidified.

I specially call your attention, gentlemen, to the details of this case. First of all, it is very remarkable that there should have been a succession of ulcers, and that the numerous ulcers should have been grouped exclusively at the pylorus by a sort of elective affinity. Then you see that this woman, who had had several years previously, and upon several different occasions, the signs of simple ulceration, presented numerous ulcerations. There had been periods of remission, and even of apparent cure; in fact, some of the ulcers were cicatrized. It was the most recent ulcer which determined the perforation, the cause of the peritonitis and pleurisy. Observe, that this case is an additional proof of what may be called the law of antagonism between the simultaneous existence in the stomach of round ulcer and cancer, like the law of antagonism between the successive presence in the uterus of fibrous and carcinomatous growths.

In this case, moreover, you find an example of *perforation* of the stomach, a symptom rather frequent, and often very formidable, in conjunction with simple ulceration. The perforation may give rise to a series of symptoms of which it is necessary that I should speak. First of all, peritonitis is a necessary result: it may be either partial or general, according to the rapidity with which the perforation takes place. When the destruction of all the coats of the stomach is accomplished slowly, adhesions have had time to form, between the perforated stomach and the neighboring organs: usually the pancreas, the left lobe of the liver, or the mesentery, compensates for the loss of substance, so that the matter contained in the stomach is prevented from flowing into the peritoneum. At other times, as in the case of our patient, the neighboring organ is so disposed as not to supply completely the loss of substance, and consequently peritonitis is produced, which in its turn originates a new series of complications, such as diaphragmatic pleurisy. Finally, the perforation may have taken place with such rapidity as to allow no time for the formation of adhesions, so that through the perforation of the stomach a part of the contents of that organ will pass into the abdominal cavity; then, as you can easily understand, the necessary consequence is a very acute, and speedily fatal peritonitis. On the other hand, when solid adhesions attach the perforated stomach to the neighboring organs (the liver or pancreas), one of two things may occur: there may be no alteration in the organ which furnishes to the stomach an adventitious wall, or at least a new fibro-cellular formation, which imparts thickness to it; or again (and the fact is much more curious) the ulcerative process continues and attacks the annexed organ, so that the ulcerative disease, primarily localized in the stomach, extends by a mechanism, of which I cannot give a very good account, to perfectly different tissues, such as those of the liver and pancreas. The adhesive inflammation in the neighborhood has nothing in it to call forth our astonishment: the occurrence is very common. But that the inflammation, at first simply adhesive, should become ulcerative, in respect of the liver and the pancreas, as it had been in respect of the stomach, is, I confess, a very remarkable fact, affording an additional proof of the specificity of morbid actions, a specificity which is more related to the essence of the disease than to the nature of the tissue which is attacked. This is the fact to which I have been desirous in a special manner to direct your attention. In contradistinction to certain cases in which the existence of simple ulcer of the stomach found at the autopsy, but not discovered during life by any special symptom, and which has not notably given rise to hemorrhages, even of a very slight kind, there are others in which black vomit and melæna of considerable extent are met with, independently of any appreciable lesion of the stomach.

Six years ago, I was called in by my honorable friend Dr. Riembault, to a lady aged 65, living on the Quai des Célestins, and who, I was told, had vomited, and passed by stool, large quantities of blood. My first impression, produced by the pale yellow, cachectic complexion of the patient, was that she had cancer of the stomach: this was also the opinion of the professional brother by whom I was called in, and the look which we exchanged, when examining this lady, only told too eloquently, that we held in common the same opinion of her case, and had not a favorable impression as to her situation. The patient told us that she had been four days in Paris, where she had arrived in perfect health, never having experienced, in relation to her digestion, anything to attract attention. Her appetite was regular; she had never had pains, nausea, nor eructations, and she had been surprised by the vomiting of blood, which had occurred, without appreciable cause, on the day after her arrival in Paris. The previous history of the case scarcely agreed with the idea that there was a cancerous lesion; although there are cancers of the stomach which are completely indolent, and do not reveal any serious disturbance of the economy.

Completely reserving our diagnosis, my colleague and I placed ourselves in the position of persons who had to treat an essential hemorrhage: we prescribed preparations of iron and rhatany. Three days after my visit, the stools had ceased to be black, and there had been no more gastrorrhagia. Next month, the lady returned to the country. Her health became good; and five years afterwards, I learned that it continued excellent.

At the beginning of August, 1861, a man, 33 years of age, was admitted to our wards. At the very time of admission, he was seized with an attack of vomiting, and died suddenly. He had arrived in a state of profound prostration, unable to speak, and unable in any way to give information of his case. All that we knew, we derived from the persons who had carried him to the hospital on a stretcher, was that he had been long ill. The hospital attendants were struck with the yellow color of his skin, and the great bulk of his belly.

The matters vomited, which were carefully preserved, were formed of blood altered by its admixture with the gastric juice: it was a liquid of sepia color, with a deposit of solid matter finely granulated and resembling soot, of which a portion was suspended in the liquid. At the autopsy, the abdomen was found to contain peritoneal effusion, as if encysted. The stomach having been removed with the greatest possible care, was examined and found to present no morbid change. Its cardiac and pelvic orifices were free, and there was no trace of cancerous tumor; upon the mucous surface, the color was like the lees of wine. We did not discover the smallest ulceration. In the duodenum, there was no appreciable lesion. The lungs, heart, and brain were healthy.

The appearance and quality of the matters vomited in these cases, can leave no doubt in the mind as to their nature; and it is very evident also, that the hemorrhages had the stomach as a starting-point. The progress of the symptoms, their happy termination, in the first case, the results of necroscopic examination in the second, clearly showed that there was neither cancerous lesion nor ulceration of the stomach. The profuse sanguineous exudations came from the mucous membrance of the stomach, just as they come from the surface of other mucous membrances: this we sometimes see in the intestine, as in the following case, which I have many times had occasion to narrate to you.

A former functionary of our faculty was seized, about seven years ago, with serious symptoms, all the details of which are worthy of being reported.

Though generally of very good health, he was subject to constipation of such a kind that he never went to stool more than once in ten or fifteen days; and then, he only passed a very small quantity of hard black matter like goat's dung.

One evening, without having previously experienced the slightest derangement in his habitually excellent health, without having committed the slightest excess at table, he suddenly felt an indescribable sensation of discomfort, and immediately afterwards fell down in a state of unconsciousness. For nearly twenty minutes he remained in this state. He was taken home in a carriage; and after a good night, during which his sleep was tranquil, he, on the following day, resumed his usual duties. The occurrence now described took place on a Thursday: on the following Monday, when sitting in his office, he was again suddenly seized with symptoms precisely similar to those which characterized the attack he had had four days previously. On the following day, he twice experienced a recurrence of the same symptoms; but matters assumed a more serious appearance, for through extreme feebleness he was obliged to remain in bed. I saw him upon the Wednesday, during the afternoon. His complexion, generally good, was of such a decided cadaveric paleness, as at once to arrest my attention. Experience made me at once suspect the existence of intestinal hemorrhage. I requested that his stools might be shown to me. I found, however, that he had neither had any alvine evacuation for eight days, nor had he had any vomiting. I immediately prescribed a purgative, the salts of Seignette, so far as I can recollect. The result was, the evacuation of an enormous quantity, estimated at five or six pounds, of black pitchy matter, resembling the tar used for ships. My diagnosis was thus confirmed: I had to do with a case of melæna.

The antecedents of this individual, the progress of the symptoms, and the careful examination of the abdominal viscera by palpation, caused me to reject the idea of hemorrhage depending upon a lesion of the stomach or intestines; and I comforted the family by assuring them that the malady was simple melæna. My prognosis was completely verified. For three months, it is true, the patient retained his anæmic color; but, under the influence of rhatany, cinchona, and iron, he regained his usual color, and his former good health: he has not had, up to this date, any recurrence of the symptoms.

Examples analogous to the case which I have now related are more common than is generally believed. Persons, when in good health, are suddenly seized with an undefined feeling of discomfort; they are observed to become pale and to fall down in a faint. One or two hours later, when they go to stool, their motions are as black as pitch, and this color of the motions is retained for one or two days, after which it ceases. For some time afterwards, however, the patients suffer from debility, loss of appetite, slight gastralgia, buzzing in the ears, and paleness of the skin. The appetite and the strength return: convalescence is complete. The symptoms may nevertheless again occur at a period more or less distant; they recur in the same form, and are often not observed by patient or physician till a more profuse hemorrhage suddenly produces prostration. This is a rare occurrence: usually, they are not observed till a complete cure has taken place.

When, in the course of your practice, you meet with patients who complain of passing blood by stool, or rather when they tell you that their stools are as black as tar, that is to say, presenting the characters of melæna, carefully interrogate them as to their antecedents—ask them, if they have never become suddenly pale, and continued so for a week or a fortnight; and ask them, also, whether these symptoms have not recurred several times. These phenomena will enable you to clear up your diagnosis, and to state that the patient has had intestinal hemorrhage in his former attacks as well as when he has noticed stools of a red, black, or bistre color.

Let me now return to the consideration of simple ulcer of the stomach. The gastrorrhagia which usually accompanies it is, therefore, not a symptom of sufficient diagnostic value to enable the physician to pronounce definitively; for not only is it sometimes absent, but it may be present independently of any appreciable lesion, as in the cases I have just mentioned; as also, in the cases in which hæmatemesis is supplementary to habitual hemorrhage, as, for example, in some women suffering from disordered menstruation; in some patients affected with hæmorrhoids, in whom the menstrual flux is suppressed; and finally, also, it is a common phenomenon both in cancer and in simple ulcer.

It has been said, I admit, and the fact is one which clinical observation will enable you to verify, that vomiting of blood and black motions are to a certain extent more characteristic of simple ulcer, than of cancer of the stomach, inasmuch as they belong to all the stages of simple ulcer, of which, moreover, they frequently constitute the earliest symptoms. On the other hand, we see that in many cases of cancer, there is neither black vomiting nor black motions; and that when they do occur, it is generally at the last stage of the disease. It is also a clinical fact opposed to general opinion, that profuse hæmatemesis and suddenly prostrating melæna belong much more to simple ulcer than to cancer.

However precise this proposition may be when applied to the majority of cases, it is essential to recollect that the exceptions to the rule are sufficiently numerous to prevent its being regarded as an absolute sign that simple ulcer exists.

In cancer, hemorrhage from the stomach or intestines sometimes supervenes, during a condition of apparently perfect health, as the first and only symptom of the disease which will inevitably carry off the patient.

A very near relation of my own, 60 years of age, when in full health and strength, was seized one day at table with syncope accompanied by slight convulsions, such as are commonly observed as a complication of loss of consciousness. I was present, at dinner, with my relation; Bretonneau, who was also present, believed that the seizure was an attack of epileptic vertigo. The state of syncope continued for a long time. The patient was put to bed: for a fortnight, he was very feeble, and deadly pale. I did not, any more than Bretonneau, suspect the nature of the case. A year later, the same person left home to visit one of his estates: all at once, whilst he was giving his orders to his managing servant, he fell down as if struck with a thunderbolt. He was restored by sprinkling some drops of cold water upon his face: when restored to consciousness, he felt an imperative desire to go to stool, and passed a great quantity of blood. Immediately after this hemorrhage, he became deadly pale, as on the occasion of his first seizure. We now clearly saw what had taken place the year previously. Bretonneau and I then understood that upon both occasions there had been an intestinal hemorrhage.

The patient got well; his health appeared to be perfectly re-established, when again, some months afterwards, he was seized for the third time with similar symptoms. He had risen early in the morning to speak to his work-people, when, feeling a desire to go to stool, he hastily returned to his room. Soon, his domestics hearing a great noise in the closet, ran to his assistance, and found him stretched upon the floor, vomiting blood in large quantity: the basin of the water closet was filled with bloody matter, and his clothes were also soiled with similar discharge. After this event, he remained for a fortnight in bed, being unable to put his foot to the ground, so extreme was his debility. He, however, again regained his health; but from that time, he complained of lancinating pains in the epigastric region, where we discovered the existence of a tumor, of which we were able to follow the rapid progress. Soon, all the symptoms of cancer of the stomach were evident; and three years from the date of the first seizure, by which the beginning of the disease had been announced, my unfortunate relative died.

Six years ago, a man living in the environs of Paris frequently consulted me in reference to a frightful vomiting of blood, which he had had a few days previously. He told me that his appetite then was, and always had been, good: he never had had the slightest pain, nor the slightest uneasiness in his stomach. The hemorrhage had supervened when he was in the most perfect health. He estimated at about a litre the quantity of black matter which he had vomited. This great loss of blood fully explained the anæmic paleness of his skin. Upon examining, I discovered in the region of his stomach an enormous tumor, occupying the great curvature, and quite painless on pressure. Notwithstanding this great lesion, the man had preserved perfect regularity in his digestive functions. I prescribed preparations of iron and rhatany, not certainly because I expected to cure his cancer, but to satisfy the inclination of restoring the economy, deeply disordered by the hemorrhage of which he had informed me. Four months afterwards, he returned to consult me. He had regained flesh and a good color. Nevertheless, the tumor was greatly augmented in volume. Six months later, another attack of hæmatemesis supervened, when I was again consulted; I again prescribed rhatany and iron, which once more produced a good effect. Soon afterwards, however, the usual symptoms of cancer of the stomach were developed, colliquative diarrhœa set in, and the patient died.

The vice-president of the Courts of Law in one of our most important towns, was seized during the year 1849 with vomiting of blood and great intestinal hemorrhage, which brought him to within an inch of death. He speedily recovered, and was able to return from quarters in the country to which he had retired, to resume his magisterial duties. He came to Paris for medical advice: it was then ascertained that there existed an abdominal tumor occupying the anterior wall of the stomach. From this date, similar attacks occurred nearly every six months; and on each occasion there was a great loss of blood both by the mouth and the anus. There was, nevertheless, no disturbance in the digestive functions. The appetite was good: in fact, the patient was a hearty eater: he sometimes experienced acute gnawing pains in the stomach. He constantly complained of a state of great debility, which prevented him from taking much walking exercise, or going upstairs without being winded. I cannot say that he was fat, but he had preserved a certain amount of plumpness: his integuments generally were exceedingly pale, and his skin presented a slight straw-yellow color.

I found that his mother had died from cancer of the breast. He himself was perfectly aware of his situation, and spoke constantly of his approaching end, fulfilling, however, at the same time, all his duties with exactitude.

In September, 1856, consequently seven years after the first attack, he went to spend the vacation at his estate in the country: some months previously, he had had hæmatemesis which, like all his previous attacks, was accompained by melæna, and lasted for several days. All at once, without any appreciable exciting cause, or premonitory symptoms, he was seized one Sunday with enormous hemorrhage from the mouth. The bleeding recurred on the following Tuesday, Thursday, and Saturday. On each occasion, the quantity of blood vomited was sufficient to fill a large basin; and also, on each occasion, there was an evacuation by stool of black matter resembling tar. The patient, exhausted by loss of blood, fell into a state of profound debility. He died during the daytime of Sunday, 16th October, eight days after the frightful symptoms now described.

Although in the case now described, the evidence to be derived from post-mortem examination was wanting, there could be no doubt as to the diagnosis. The tumor was perfectly appreciable to palpation; and its existence was ascertained by Dr. Gendrin, and my former *chef de clinique*, Dr. Blondeau.

You see, gentlemen, from these examples, that stomachal or intestinal hemorrhage, however profuse and however frequent, cannot, any more than gastric pain, be given as a positive sign of simple ulcer of the stomach. I may say the same of the vomiting of glairy matter, which is sometimes very profuse in persons affected with this disease.

This kind of vomiting is the result of irritation of the gastric mucous membrane in the vicinity of the ulcer, which irritation causes an increased secretion from the stomach. This symptom has still less diagnostic value in this affection than the other symptoms of which I have just spoken. In a very large number of cases of perfectly simple gastrodynia, increased gastric secretion is an ordinary symptom; it is likewise met with in some forms of chronic gastritis and dyspepsia, and is very frequent in hemicrania.

Neuralgia is often sufficient to excite the secretion of the stomach in excessive quantity; and then, the occurrence is analogous to that which takes place in other parts of the body under the influence of somewhat violent local pain. The statement of a case will be the best means of enabling you fully to grasp my views on this subject.

An individual takes masked intermittent fever, which declares itself in the form of suborbital neuralgia. At the beginning of the attack, the eye is perfectly free from injection and lachrymation. Pain begins: as it increases, the mucous membrane of the eye becomes injected, and sometimes the injection proceeds to such an extent as to be a real chemosis. For five or six hours, matters remain in this state: there is redness and swelling of the eye, with a profuse secretion of tears. The attack then passes off, the neuralgic pain abates, and the epiphenomena disappear, not returning till recalled by a new attack of the neuralgia.

In the same way, violent neuralgia of the stomach will suffice to excite profuse secretion of fluid by the stomach, which fluid will be ejected by vomiting. This copious secretion will also take place under the influence of any irritation of the stomach, be its nature what it may—in gastritis just as in gastralgia, in cancer as in simple ulcer; and consequently, glairy vomiting cannot be considered as diagnostic of the latter.

Though the presence of a tumor in the epigastric region excludes the idea of a simple ulcer, and declares that there is a cancer, it does not follow that the absence of a tumor is a positive proof of the presence of an ulcer; for it is by no means rare that a cancer completely escapes detection from the peculiar position which it occupies.

The most important element in the differential diagnosis between the two affections, as Dr. Cruveilhier has shown, must be deduced from the progress of the disease. In simple ulceration, the alternations of better and worse are thus marked: improvement attends spare diet, and there is always an aggravation of the symptoms when this regimen is departed from. In cancer, on the other hand, the disease advances steadily towards a fatal termination, irrespective of regimen and of treatment.

My honorable colleague, Dr. Cruveilhier, in enunciating this proposition, implicitly admits that so long as the disease lasts the diagnosis is impossible—that is to say, so long as neither death nor recovery takes place; in the latter case he concludes, of course, that the disease is simple ulcer. Here, however, the diagnosis may still be at fault; for, from the cases which I have brought under your notice, you have seen that there are sometimes, in cancer of the stomach, long intervals during which the disease shows no symptom, and during which, consequently, we may suppose that a cure has taken place.

Recall to your minds the magistrate whose case I related. For three years the only morbid phenomena which presented themselves were hæmatemesis and melæna, recurring at pretty distant intervals; and if examination of the epigastric region had not enabled us to detect the evident existence of a tumor, we might have more than once supposed that recovery was about to take place.

The final cessation of symptoms, and the complete restoration to perfect health, the less necessarily implies that the case is one of simple ulcer, inasmuch as I have seen non-ulcerous chronic gastritis sometimes accompanied by stomachic hemorrhage and pain in the ensiform and dorsal regions. Such occurrences are rare, I admit; but as they do present themselves, they are quite sufficient to make us form our opinions with some reserve.

To sum up: profuse and repeated stomachal hemorrhage, with or without accompanying melæna, violent gastralgic pains, apparently localized for the most part in the ensiform region and in the corresponding region of the back, when coinciding with an entire absence of any appreciable tumor in the epigastrium, justify us in supposing that there exists chronic simple ulcer of the stomach, particularly when the symptoms terminate in recovery. This is a general rule, but bear in mind that it is a rule which has numerous exceptions, and that, in the present state of science, the diagnosis of simple ulcer of the stomach is surrounded by much obscurity.

No doubt this obscurity is dispelled when, independently of the symptoms which I have just indicated, there appear other symptoms which essentially belong to cancer, symptoms which vary according to the seat of the disease.

When the cardiac end of the stomach is affected, you meet with phenomena similar to those which presented themselves in one of our patients in St. Agnes Ward, whom we were obliged to feed by the aid of an œsophageal tube. There is a form of dysphagia, characterized by regurgitation of the food, which at first seems to be easily swallowed, but which, in point of fact, only accumulates in the lower portion of the œsophagus. At the beginning of the disease, this regurgitation takes place immediately after deglutition, because the œsophagus then reacts much more powerfully upon its contents, as it has not yet become accustomed to distension. In proportion as it acquires this tolerance, the food is only rejected from the mouth at an interval more or less protracted after ingestion; but at first, individuals affected with cancer of the cardiac end of the stomach can only take liquid or semiliquid aliment, and that only by swallowing it rapidly.

There is, however, a fact with which I must make you acquainted. You will see patients who have complained of having been subject for a longer or a shorter time to regurgitation of aliment, to such an extent as to be unable even to retain liquids, and who eat without difficulty, swallowing even bulky mouthfuls. You must not allow this apparent amelioration to impose upon you; it depends in some cases upon the tumor which obstructed the cardiac end of the stomach having become softened, thus allowing the passage into the stomach to be temporarily free from obstruction. In some days, however, this obstruction may be reproduced by new cancerous growths forming around the opening.

If the lesion occupy the pylorus, there will generally be frequent but not profuse vomiting; it will occur less and less frequently, but will become more abundant.

You can understand, gentlemen, the reason of these differences. At first the stomach rebels against the presence of alimentary matter, which, after having undergone chymification, ought to be propelled into the duodenum, the entrance of which is shut. At a later stage, the stomach becomes more tolerant, and accustoming itself to the contact of its contents, allows itself to become distended till the quantity of ingesta is greater than it can retain.

In cancer of the pylorus, there likewise exists great constipation, provided the cancer be not deeply ulcerated; in which case, while the vomiting becomes less frequent, there sets in a diarrhœa which soon becomes lienteric: the alimentary matter escapes through the permanently open pyloric orifice, before having been subjected to sufficient elaboration in the stomach.

In general, we can detect a cancerous tumor by palpation, a circumstance which facilitates the diagnosis of cancer of the pylorus. In exploring the region corresponding to the inferior orifice of the stomach, we find a more or less bulky tumor fixed in the situation which it occupies; while a cancerous tumor of the great curvature of the stomach will change its position as the stomach may or may not be distended. Mobility is also a sign of great value in detecting the existence of tumors of the liver, of which organ they follow the movements, rising and falling with the diaphragm in respiration.

Apart from the local characteristic phenomena of cancer of the stomach, there are others of equal importance.

Under the influence of great disturbance of digestion, a patient visibly loses flesh; his skin assumes a straw-yellow tint, which, I know, may also show itself in persons who have had profuse hemorrhage, but which, in cancer of the stomach, will occur where there has been no loss of blood at all.

Cancer diffused over the mucous surface—that form which has been called *cancer en nappe*—much more frequently eludes our means of direct exploration. You recollect a woman, aged fifty-five, who was admitted to the Hôtel-Dieu in the beginning of September, 1861, and who presented extreme cachexia and emaciation, loss of appetite, pain in the abdomen, particularly in the right side, great flatulent distension of the stomach, ejection of gas and acid water by the mouth, vomiting of several months' duration, and diarrhœa. At a subsequent period, it was observed that the matter vomited was of a blackish color, and deposited a sediment resembling soot. There was well-marked tympanitis, particularly upon the left side, occupying the hypochondrium and iliac fossa: there was also tympanitic distension of the hypogastric region; and on the right side, there was a sensible depression in the hypochondrium. It was supposed that this tympanitis arose from distension of the transverse and descending colon, and also of the sigmoid flexure.

The cachectic state of this woman, and the black matter which she vomited, scarcely left any room for doubt as to the diagnosis. Still, to make it certain, we were anxious to find a tumor in the region of the stomach; and, not having found any such tumor after having several times carefully examined the abdomen by palpation, I thought that meteorism of the large intestine caused an obstacle to investigation by palpation and percussion. By the end of September, the vomiting had become more frequent; the patient always had in her mouth a blackish acid fluid; she could no longer take food; and at last died without a struggle on the 29th of September.

At the autopsy, I found upon the face streaks of blackish matter issuing from the mouth; tympanitic distension of the belly in the same region in which it had been observed during life; and a sensible depression of the right hypochondrium, imparting to the abdominal parietes an anomalous appearance which arrested the attention of those present. The abdomen was opened carefully, when it was perceived, to the great amazement of everybody, that the special form of the abdomen depended upon a great gaseous distension of the stomach, which began in the cardiac region, occupied the right hypochondriac and the hypogastric regions, and terminated at the margin of the right iliac fossa. This distension, therefore, was vermicular, and had for its superior and inferior extremities the cardiac and cæcal regions. The whole of the small intestine had fallen into the pelvis: the large intestine retained its normal relations, excepting that the transverse portion of the colon had been a little dragged downwards by the great curvature of the stomach. There was no gas in the intestinal tube. The pyloric extremity of the stomach was in juxtaposition with the cæcum, and, in subsiding, had dragged down the upper portion of the duodenum. The liver, which was atrophied, had descended in front of the kidney. When the stomach was opened, it was found that its parietes had their natural consistence and thickness; it contained a large quantity of black liquid;

and in the pyloric portion, the mucous membrane of the stomach was the seat of diffused cancer, which extended over a surface of four or five centimetres beyond the pylorus. The edges of the cancer were denticulated, pale, like the remainder of the cancerous surface. The duodenum was intact: it contained a small quantity of blackish fluid.

The seat of the cancer, and its diffused character, accounted for the extreme distension of the stomach, and also for the difficulty—I may say the impossibility—of determining during life whether or not a tumor existed. In fact, it was necessary to take the pyloric end of the stomach between the fingers to ascertain that it was increased in bulk. There was no cancer in any other part of the body. The lungs showed cicatrices: and also tuberculous deposits in both summits. On the right side of the chest, there were cellular adhesions, the remains of an old attack of pleurisy.

When the cancer is beyond the reach of our ordinary means of investigation, as in the case I have just detailed, there is a valuable diagnostic sign which I shall now point out to you. The sign to which I refer is obliterative phlebitis [*phlébite oblitérante*], to which I directed the attention of pathologists fifteen years ago, being then, I believe, the first who had noticed it.

Should you, when in doubt as to the nature of an affection of the stomach; should you, when hesitating between chronic gastritis, simple ulcer, and cancer, observe a vein become inflamed in the arm or leg, you may dispel your doubt, and pronounce in a positive manner that there is cancer.

One day, my lamented colleague and excellent friend, Dr. Legroux, showed me in his wards a very anæmic man of sixty years of age. He never had had hemorrhage nor vomiting: he only complained of gastric symptoms, and his chief complaint was of loss of appetite. The patient presented exactly the appearance of a person with leucocythemia. Although neither the spleen nor liver was of abnormal size, I was disposed to adopt that diagnosis, when, upon uncovering the inferior extremities, I found that one of the legs was very œdematous, and that the posterior part of the calf of the same leg was the seat of acute pain. There was, in fact, well-marked *phlegmasia alba dolens*. That was sufficient to inform me that the gastric symptoms depended upon cancer of the stomach, a view of the case which was confirmed some weeks later at the autopsy of the patient.

I have several times, in the wards, called your attention to similar facts; and have at the same time pointed out to you that obliterative phlebitis is not a symptom which belongs peculiarly to cancer of the stomach, but that it is equally symptomatic of cancer of any other internal organ. I propose on some future day to return to this important point, when I shall have an opportunity of entering fully into the subject of *phlegmasia alba dolens*.

Before concluding this lecture, allow me to add a few words on the *treatment of simple ulcer of the stomach*. Regarding the treatment of cancer, of which I have incidentally spoken, I need scarcely tell you that we cannot arrest the disease, and can do no more than administer palliatives.

In simple cancer of the stomach, I usually institute the following plan of treatment. I order two or three grammes of trisnitrate of bismuth to be taken an hour, at the least, before meals, three times a day. It ought to be suspended in mucilaginous water, so as to be well spread over the surface of the stomach; but should the patient have a strong dislike to take the medicine in this form, it may be administered in an envelope of moist wafer-paper. My object in confiding the medicine to the empty stomach is to render its action more immediate, and consequently more efficacious. In this affection, bismuth, as well as other therapeutic agents of which I shall have to speak immediately, acts in the same way as if it were applied directly to a wound, or to the mucous membrane of the vagina, nose, mouth, or eyes, in the chronic inflammation of which it is a topical agent of great value. Bismuth, in point of fact, constitutes the basis of the treatment of simple ulcer. After having administered it for ten consecutive days, in the manner which I have described, I substitute for it pills of nitrate of silver, each pill containing one centigramme of that salt: the patient takes, for five consecutive days, three or four of these pills during the day, each pill being taken at least an hour before eating: I then, for ten days, resume the bismuth: after that, for four or five days, I give to the patient, before breakfast in the morning and at midday, a powder composed of one centigramme of calomel and fifty centigrammes of sugar. After this course of medication, I resume the bismuth and proceed with the other medicines in the order already detailed. Again and again, this routine is repeated for three or four months.

When cessation of pain, and a return of strength and appetite, lead me to conclude that the cure is complete, I suspend the treatment for a month. I then resume the same treatment for two consecutive months; then I suspend it for two months, and recommence it, continuing it during a month. I proceed in this way for at least two years. It is by pursuing this patient plan that simple ulcer of the stomach is cured, and its recurrence prevented.

I need not tell you that ferruginous preparations must not be omitted when there is great anæmia caused by profuse hemorrhage or imperfect nutrition.

To combat the violent pain I have recourse to opium, which I am always careful to administer in small doses at mealtimes.

Hemorrhages are treated by rhatany, sulphuric acid, and ice; when they have been arrested, and when the pain has been subdued, I prescribe bitters such as the decoction of cinchona, infusion of quassia or of colomba root; sometimes also certain medicines which combine bitter with slightly purgative properties, such as rhubarb. Finally, I administer the preparations of iron.

But the grand point in the treatment is the regulation of the diet, which ought not to be of an exclusive character, but specially adapted to the peculiarities of the patient's digestion.

Professor Cruveilhier says, while insisting upon the advantage of milk diet, that "the great problem to solve in the treatment of simple ulcer of the stomach is, to find an aliment which shall be borne by the stomach without producing pain, and in relation to this point, the instinct of the patient is a surer guide than all the rules of art."

When the stomach becomes a little more tolerant, other kinds of food must be tried; for diversity of food is perhaps the most useful medication in the dyspepsia which accompanies ulcerous gastritis, as indeed it is in all other kinds of dyspepsia. I cannot too often repeat that the stomach likes variety; and, in opposition to the plan which I see followed by most of my professional brethren, I require that my patients make a meal of several dishes. I do not say that this can be carried out by sheer force, or at one bound; but it is a point which must be attained, and, moreover, it can be attained much more rapidly than is generally supposed.

LECTURE LXXI.

DIARRHŒA.

Classification according to Proximate Causes, that is to say, according to the Mechanism by which the Diarrhœa is produced.—Catarrhal Diarrhœa: this may be a Specific Affection.—Sudoral Diarrhœa [Diarrhée Sudorale].—Nervous Diarrhœa.—Catarrhal Diarrhœa, in which the Affection is consecutive upon increased Secretion from the Digestive Canal or its Appendages.—Diarrhœa resulting from Increased Tonicity.—Diarrhœa resulting from Indigestion.—Diarrhœa associated with Organic Disease.—This Classification is Artificial: the different kinds are blended with one another.

GENTLEMEN: When the alvine excretions are abnormally fluid, frequent, and profuse—when they consist of an undigested or imperfectly digested alimentary residuum—when they consist of the products of secretion from the intestinal mucous membrane, from the pancreas, or from the liver—when they contain, or do not contain, blood or débris of mucous membrane—we say that there is diarrhœa. Of all the diseases which the physician meets with in practice, diarrhœa is undoubtedly the most common; it is also that which requires to be combated by the most varied measures. This diversity of remedies being necessitated by the multiplicity of causes, it is essential to know what these causes are before we can institute a rational mode of treatment.

With a view to facilitate the study of the subject which I have to bring before you to-day, I distinguish several kinds of diarrhœa. The division which I adopt is quite different from any of those which you will find in classic authors; but, without attempting to discuss the merit and the advantages of one or the other, I propose to lay before you my own views, because I thus understand the subject, and because, before everything else, I practice medicine upon principles derived from my own experience, submitting them to your appreciation, and entirely delivering them over to the control of your judgment.

I consider that there are seven kinds of diarrhœa: one is catarrhal, or inflammatory diarrhœa; the second is sudoral diarrhœa; the third is caused by increased intestinal secretion from disturbance of innervation: the fourth is also a catarrhal form of diarrhœa, but it is a catarrh supervening consecutively on an excessive intestinal flux; the fifth is diarrhœa from excess of tonicity in the intestine; the sixth depends upon unsuitable aliment, or aliment bad in quality either absolutely or relatively; and, finally, the seventh is associated with different organic diseases.

The *catarrhal diarrhœa* is the most frequently observed form of the affection. All mucous membranes—the mucous membrane of the eye, the nose, the ear, the mouth, the pharynx, the larynx, the bronchial tubes, the uterus, the urethra, the bladder, the kidneys—are liable to inflammation. From the nature of the tissue attacked, the inflammation generally assumes a peculiar character, which constitutes catarrhal phlegmasia. The mucous membrane of the digestive canal is not any more protected from attacks of this character than the other mucous membranes, and perhaps it is even more subject to such attacks than they are.

Like every phlegmasia, catarrhal phlegmasia may be simple and genuine; but likewise, whatever may be its seat, it may be specific, and so form a certain number of species, each of which, bearing a certain relation to its origin, will run a special course, manifest symptoms peculiar to itself, and related to its specific cause.

These different kinds of diarrhœa do not resemble one another, but they do resemble themselves when they occur in different individuals. They differ essentially as to their symptoms, their duration, their degree of severity, and also as to the therapeutic measures required for their cure: this latter point is one of which you must never lose sight.

In catarrhal phlegmasia of the ocular mucous membrane, for example, along with those simple inflammatory affections occasioned by exposure to cold, or the introduction of a foreign body under the eyelid, you will have that epidemic catarrhal phlegmasia vulgarly called *cocotte*. You will have also purulent ophthalmia, blennorrhagic ophthalmia, and the like, which are very different in their symptoms and in their modes of termination.

In catarrhal inflammation of the mucous membrane of the nasal fossæ, besides simple coryza, you will have the coryza of measles, scarlatina, small-pox, glanders, scrofula, syphilis, &c.: no one will mistake these affections; for their characteristics are differential and distinctive.

We likewise have both simple and specific catarrhal inflammations of the intestinal canal: the intestinal inflammation, for example, is specific which accompanies measles, scarlatina, and the onset of confluent small-pox: it is also specific when related to the herpetic or other diathesis. I have already at some length directed your attention to these facts when lecturing on other subjects, particularly when discussing dyspepsia.

These inflammations, whether specific or non-specific, have characters in common, in addition to the characters which distinguish the one from the other. Some of these common characters belong inherently to the anatomical structure of the mucous tissue: I refer to flux, and increased discharge from the mucous membrane, the secretions from which are modified both as to quantity and quality. The others are subordinate to the seat of the inflammation, that is to say, to the organs affected: they are functional derangements, for in that way alone is an organ diseased—the function allotted to it is more or less disordered, and perhaps is entirely in abeyance. It is unnecessary to add, that the functional disturbance necessarily varies according to the particular organs implicated.

If it be the nasal mucous membrane which is inflamed, the sense of smell is enfeebled, perverted, or lost. If it be the bronchial mucous membrane, the disturbane is much more serious. The digestion of oxygen, if I may use that expression, being badly accomplished, hæmatosis takes place imperfectly, and according to the degree in which the catarrhal inflammation is more or less extensive, more or less deepseated, or more or less persistent, the disorder of the function of hæmatosis may attain such a height as to induce cachexia. Should it be the mucous membrane of the intestinal canal which is implicated, digestion will be disturbed, and the nature of the disturbance will depend upon the portion of that passage which may be peculiarly affected. When inflammation attacks the stomach, its secretory apparatus immediately performs its functional office in an abnormal manner, and the gastric juice being no longer appropriate in quantity and quality, chymification is imperfectly accomplished. When the stomach remaining healthy, the intestines are affected, chylification will either be performed badly or not at all, in proportion to the degree in which the inflammation has disturbed the intestinal secretions.

But, whilst this increased secretion of gastric and intestinal fluids leads to a vitiated elaboration of the food, this badly elaborated food in its turn acts as a foreign body upon the mucous membrane of the digestive tube, augmenting the secretion and the profusion of the flux. It also irritates the muscular coat of the intestines, exciting its contractions in such a way as to render the peristaltic movements both more frequent and more rapid. This increased frequency and rapidity in the peristaltic movements, which is excited also by the presence of the excrementitious principles of the bile, which, as I have just said, is poured in large quantity into the duodenum —this increased frequency and rapidity in the peristaltic movements explains the increased frequency of the stools.

In diarrhœal catarrh, then, the flux consists of the residue of badly elaborated elementary matter, of humors secreted by the intestinal surface, and of secretions produced by sympathetic influences upon the great glandular organs of the digestive apparatus, the pancreas and liver.

As you know, gentlemen, when the extremities of the different canals of a gland open upon an irritated mucous membrane, the irritation is propagated by sympathy to the gland, and its secretory functions are thereby often augmented. Simple irritation of the conjunctiva, passing by sympathy to the lachrymal gland, produces an abnormal flow of tears, the affection called epiphora. Excitation of the mucous membrane of the mouth, caused by chewing the root of Anthemis pyrethrum, or any other sialagogue, will occasion profuse salivation, the result of sympathetic irritation of the salivary glands. So, in like manner, an inflammation or irritation of the mucous membrane of the duodenum will react upon the pancreas and liver, causing an increase in the pancreatic and hepatic secretions. In proportion to the degree of this sympathetic excitement of the liver, will be the greater or less amount of biliary matter in the diarrhœal discharges.

The cause, then, of the first kind of diarrhœa is an irritation or inflammation of the gastro-intestinal apparatus, a *gastro-enteritis*, or an *enteritis*, to use expressions which some physicians of the present day seem to have erased from their vocabulary.

Let me here repeat what I have said elsewhere. It is quite right to impeach gastritis and gastro-enteritis, as understood by Broussais; but to deny the very existence of such affections is proceeding too far, is indeed proceeding to the opposite extreme of his error. I certainly do not believe that inflammations of the stomach and intestines occur so frequently as Broussais supposed, and still less do I believe that all the general symptoms which he attributed to them can be charged to their account. I do not see —as Broussais saw—gastro-enteritis in every disease; but neither do I see why the mucous membranes of the stomach and intestines should alone be exempt from attacks of inflammation. From the very nature of the functions which it has to perform, it is, I admit, more enduring, less sensitive, than other mucous membranes; nevertheless, that form of inflammation, that catarrhal inflammation, which, so to speak, only strikes the surface of the organ (whatever may be its cause) is in it not the less common. In fact, it is a much more common affection than is generally supposed.

I have now to speak of the second or *sudoral* form of *diarrhœa*. The details into which I entered regarding it, when lecturing upon the sudoral exanthemata, were so full, that I might on the present occasion pass over the subject lightly, were I not anxious that you should clearly understand what I mean by the term sudoral diarrhœa, so as to grasp more thoroughly the different therapeutic indications which correspond to the different causes of the intestinal flux.

Such of you as have already had some practical experience in our art, particularly if your practice has been among children, must have observed the kind of diarrhœa to which I wish to call your attention. You must have seen persons in whom the influence of a slight increase of external temperature, arising, for example, from an excess of bedclothes, invariably produces more or less diarrhœal discharge. This observation has been made in respect of the lower animals as well as in respect of the human species: some horses, ere they have run half a league or a quarter of a league, have their skin covered with sweat, and, at the same time, have liquid alvine discharges. The diarrhœa and sweating are both phenomena of the same class, and arise from an abnormal secretion, the one from the internal and the other from the external integument, the result respectively of a fluxion to the secreting organs of the intestines and the skin.

There are other cases in which it seems as if all the emunctories were scarcely adequate to disembarrass the blood of the excrementitious matter produced in it in excessive quantity: then there occurs as a physiological, that which we have seen as a pathological, phenomenon, in measles and other eruptive fevers; or, as I have said, the exanthemic fluxion has taken place simultaneously from the skin, intestines, and bronchial tubes, manifesting itself by the characteristic eruption, diarrhœa, and bronchial catarrh which accompany the earliest of the pyrexial symptoms. The concurrence of profuse sweating and intestinal flux is likewise met with in the fever accompanying suppuration: in this case, the diarrhœa is explained by the irritation of the tegumentary membranes caused by the serous part of the pus being absorbed, and trying as it were to become eliminated by its natural emunctories: this is explained by the establishment of a sort of sympathy between the adventitious membranes of the suppuration and the mucous membranes.

If excessive sweating and intestinal flux show themselves simultaneously, the latter is in general only supplementary to the cutaneous secretion. Let me explain. You are acquainted with that sort of compensation which exists between the functions of the skin and mucous membranes—particularly the intestinal, bronchial, and urinary mucous membranes. You know that their secretions are destined, besides accomplishing other uses, to modify the composition of the blood by removing from it effete matters useless for the maintenance of life: there can be no change in either without a disturbance of their equilibrium: hence is it, that an increase or diminution in the action of one or other of these secreting organs will occasion a diminution or an increase in the action of the other. Nowhere is this antagonism of secretions so conspicuously manifested as between the skin and intestinal surface. You will now be able to understand why such a disturbance of the functions of the skin as prevents the secretion of sweat will often induce too profuse a secretion from the intestine. This is the explanation of attacks of diarrhœa supervening upon chills and suppressed perspiration.

Intestinal fluxes are sometimes so excessively copious as to lead to serious consequences. These are the fluxes which the older writers called *colliquative*, and of which a typical example has been furnished by the well-known circumstances which occurred to Morgagni. When travelling post on a fatiguing journey, he had a sudden attack of diarrhœa, and in twelve hours discharged from the bowels "at least sixteen pounds of nearly limpid water." This discharge, which was accompanied by only slight pain, ceased after the vomiting of a greenish matter resembling a small leaf of cooked grass. Morgagni adds: "On the following day, I realized the danger I had been in, when I looked at my body, but particularly when I saw my face and hands as flaccid as if I had emerged from a very severe long illness: I felt great dryness in the mouth and throat, disgust for food, and a sense of lassitude. The symptoms lasted only for two or three days, with the exception of the anorexia, which continued for a longer period."*

There is another form of sudoral diarrhœa, one which is rather frequently met with in women at the change of life. As you are aware, in the majority of women at that critical period, the approach of the final suppression of the catamenia is indicated by flushings of the head and of the skin of the whole body, accompanied by a profusion of hot sweat, before there is any irregulariy in the flux. These inconvenient flushings and sweatings sometimes recur from twenty to forty times a day. By and by, the menstrual periodicity becomes modified, and at last the flux entirely ceases: the hot flushings, or what women call the "*bouffées de chaleur*," then begin gradually to diminish, although they may continue for some months longer, and even sometimes for two or three years.

Now, in these women, it not unfrequently happens that the hot flushings disappear for a time, being then replaced by a serous intestinal flux, accompanied by borborygmi occurring in a strangely sudden manner, either in connection with, or independently of, mental emotion or errors in diet.

I have thought it well, for reasons which you can appreciate, to place this form of diarrhœa in the *sudoral* class, although, in reality, it ought rather to be classed with the forms of diarrhœa which I have called *nervous;* and of which I am now going to speak.

The influence of the nervous system on the secretions is a physiological fact so exceedingly well known, that I hardly require to recall it to your recollection.

The beautiful experiments of Claude Bernard upon the functions of the liver have shown us, that by pricking the floor of the fourth ventricle in a particular place glucosuria is produced, polyuria by pricking it in another situation, and albuminuria by pricking it in a third place.

These facts, so clearly elucidated by experiments on living animals, have been conclusively demonstrated by pathological observations: as you all know, neuralgic pain excites secretion in the glands near the affected parts, and toothache is often accompanied by excessive salivation, just as neuralgia of the fifth pair occasions lachrymation.

Similar effects are produced upon the secreting organs by the passions, by even moderate excitement, and by intellectual engrossment.

Pain, joy, a tender affecting sight, draws tears from the eyes. The mere idea or the recollection of a delicious dish, excites the salivary secretion; and, to use the popular expression, makes the mouth water. Mental disturbance, if somewhat intense, will cause a frequent desire to make water.

Similar mental influences are observed in the lower animals. Of the correctness of this statement, I require no better proof than that which is afforded by that wonderful phenomenon, the rush of milk to the mammary gland. It has been alleged, says Müller, that the mere sight of her colt will excite the lacteal secretion in a mare.† It is certain, that the manner in which cows are milked marvellously modifies the result of the operation;

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* MORGAGNI: Recherches Anatomiques sur le Siège et les Causes des Maladies. Lettre XXXI.

† J. MULLER: Manuel de Physiologie, traduit de l'Allemand par Jourdan. 2d ed., Paris, 1854.

that a cow, milked by a gentle person, one who knows how to proceed, will give more milk than when operated upon by an individual who milks roughly. It must, however, be remembered, that, although the cow retains her milk when she dislikes the clumsy or coarse manipulation of the milker, there is also a special action, an excitation by the hand of the teats, which excites the secretion of milk, just as the soft agreeable sucking by the lips and tongue of the infant determines a rapid rush of milk to the nurse's breasts.

I have told you, when speaking of the convulsions of children, how much mental emotion, a fit of anger, fear, or cynic spasm, may modify the lacteal secretion in women. Let me here add, that, to constitute a good nurse, it is not merely necessary that the breasts should be well formed, with the skin marbled by numerous veins, indicating a copious circulation in the organs, but that the flow of milk, generally made known to the woman by a peculiar sensation, should take place with ease and rapidity: this rapid rush of milk is generally coincident with an easy erection of the nipple, an erection which is often of a voluptuous character.

The secreting apparatus of the mucous membrane of the digestive canal, and its afferent glands, the pancreas and liver, are no exceptions to this general law. And again, in relation to the influence of mental emotion, the effects of the first cannon-shot upon the raw soldier are universally known; and further, we see children seized with diarrhœa upon being threatened with chastisement, or when anything has frightened them.

Neuralgia in the region of the eye causes an increased flow of tears; and in the same way, a local pain propagates abnormal excitement to the secreting apparatus of the intestines: hepatalgia will induce an excessive flow of bile. Along with the increased secretion there will be, as in the example which I have just cited in relation to the influence of disturbed innervation upon the lacteal secretion, a morbid change in the composition of the product secreted.

Here, then, is an abnormal flux, having for its cause a peculiar modality impressed upon the nervous system, in fact, a nervous diarrhœa. The diarrhœa, thus originally excited, will be proportionately the more abundant that the afflux of fluids into the intestinal cavity produces indigestion, from a change of relation between the food which ought to be elaborated in its passage through the canal, and the juices which ought to accomplish this elaboration.

It was necessary, gentlemen, that I should enter into these details, because nervous diarrhœa is one of the most frequent forms of the affection, and is at the same time one of those in which the physician can be most useful, when he knows how to recognize it.

In the *fourth species*, the diarrhœa is also catarrhal as in the first; but there is this essential distinction, that, while here the increase in the intestinal secretion is dependent upon irritation primarily developed in the intestinal mucous membrane, in the species of which we are now speaking, there is, on the contrary, a secretion which is at once excessive in quantity and vitiated in quality, which produces irritation, catarrhal inflammation of the intestines.

The morbid changes which occur in other organs under analogous conditions will enable you the better to understand my views. A coryza which lasts only for some hours produces in the upper lip, however little susceptible and delicate the skin of the individual may be, an irritation which, if the coryza last, will cause excoriation of the parts. Observe, gentlemen, that it is not only the flow of mucus more or less thick to which we must attribute the phenomena which I am pointing out; for you will see nothing similar supervene in children who are badly attended to and snotty (*morveux*), provided they are otherwise healthy. The mucus must have some peculiar properties; it must be the product of a morbid secretion, like that which accompanies the simplest catarrhal inflammation of the Schneiderian mucous membrane. The consecutive irritation developed in the skin may develop itself under the same influence in the pharynx: and I am convinced that many catarrhal sore throats have no other cause than the contact of irritating mucus which proceeds from the posterior orifice of the nasal fossæ affected by coryza.

In this case the patients complain of feeling the mucus fall from the nose into the throat; and, in point of fact, if you look into the throat, you see that the posterior and upper wall of the pharynx is covered with stringy purulent mucus, which, after a certain time, by contact, produces catarrhal sore throat.

Does not a profuse flow of tears cause a somewhat similar effect upon the cheeks? Although this flux be in no degree inflammatory, there will be produced redness of the eyelids, occasioned much less by the contact of the tears, than by the individual constantly rubbing his eyes. But, should the lachrymation be dependent upon an ophthalmia, of however simple a character, the epiphora will ere long be accompanied by an irritation of the parts which are bathed in tears; you will see the skin become the seat of erythema or eczema, and a greater or less extent of excoriation will follow.

Uterine catarrh, which under certain circumstances may be compared to catarrhal inflammation of the nasal mucous membrane, will often be the starting-point of ulceration of the neck of the womb. Nay, let me say, that in four-fifths, or perhaps even in nine-tenths of the cases, the excoriations have no other cause, and it is as superfluous to treat them as it is unnecessary to treat eczematous affections of the upper lip consecutive upon coryza. Both undergo spontaneous cure, when the catarrh in which they originate has ceased. Ulcerations of the cervix uteri are, moreover, not the only consequences of catarrhal inflammation of the womb. It is by no means unusual for irritation occasioned by leucorrhœal discharge to extend to the mucous membrane of the vagina, to the vulva, and even to a greater or less extent of the skin in the neighborhood of the genital organs.

Let us apply these facts to the occurrences which take place in the digestive organs. Let us first of all recollect, irrespective of its cause, that which we see daily in young children suffering from diarrhœa. Do we not then see the skin of the nates and legs covered with an erythematous redness or eczematous eruption? Do we not frequently see, in such cases, more or less deep excoriations?

You certainly cannot have forgotten a fine child of nine months old, who was admitted to bed 16 of our nursery ward in November, 1861. There was visible, round her arms, an elevated ridge of mucous crusts, resembling syphilitic mucous crusts. Now there was nothing in this child to indicate constitutional syphilis: the mother was perfectly healthy, and the child herself had had nothing the matter with her till then, excepting a rather violent diarrhœa which had continued for twelve days. There had been, first of all, a little redness round the anus; then, the diarrhœa continuing, the skin became more inflamed, when she had local symptoms apparently of a very serious character. In two days, the diarrhœa was modified: then, by applications of a liniment containing glycerin and trisnitrate of bismuth, these manifestations, apparently so threatening in character but really so little serious, disappeared.

Although we cannot positively assert what supervenes within the intestinal cavity in such a case, we may conclude that there is a similarity between the condition of the digestive mucous membrane and the condition of the parts which are visible. There is ground for believing, that a secretion, profuse in quantity and vicious in quality, whether proceeding from the stomach, duodenum, or upper part of the small intestine, or from the annexed glands, the liver and pancreas, will induce irritation of the mucous membrane of the ileum, cæcum, or large intestine, precisely in the same way that diarrhœal matter will produce irritation and excoriation of the skin in the neighborhood of the anus and of the legs. This irritation, this consecutive inflammation of parts in the first instance not affected, will cause an excessive secretion in these same parts, which will show itself as an intestinal flux or diarrhœa.

Gentlemen, I have now to explain to you what I mean by *diarrhœa resulting from augmented tonicity*, that form of diarrhœa which constitutes the *fifth species* which I have named.

When a horse is killed, and the mass of intestines is removed from its still palpitating carcass, we see their contractions continue for eight or ten minutes: these contractions are sufficiently energetic in the colon to cause the excrementitious matter to be propelled from the upper to the lower parts; so, in fact, as to accomplish defecation. In this way, we can witness upon the anatomical table exactly what takes place during life in the abdominal cavity. There occurs a series of movements separated by intervals of rest, movements influencing the whole length and breadth of the intestinal canal, but which, though they present great irregularity and apparent confusion, show a predominance of what is called the *peristaltic* motion alternating with that other motion which is called *anti-peristaltic*. The object and the result of these movements is, to mix more thoroughly, by a sort of churning process, the materials undergoing digestion, so as to enable them to undergo more intimate reactions, and to multiply their points of contact with the absorbing surfaces.

I do not require to say more regarding the phenomena which pertain to physiology: I would only add, that the slowness and the rapidity of the intestinal movements are proportionate, in the different species of animals, to the necessity for alimentation which varies with the species. I would also recall to your recollection that, in a normal state, these movements are performed more rapidly in the upper than in the lower part of the intestines—that they are more rapid in the ileum than in the large intestine—in the jejunum than in the ileum—in the duodenum than in the jejunum. But, however great their rapidity may be, it is proportionate, I repeat, to the necessity for alimentation, in such a way that the alimentary mass may have time to undergo, in each of the parts of the digestive canal, that elaboration with which each part is intrusted. When, for one reason or another, this rapidity is augmented, the elaboration is incomplete, digestion becomes disordered, and is performed badly or not at all; the food intrusted to the stomach ought to remain in it for a certain time before being converted into chyme, and passing into the duodenum, where it will be subjected to a new process. If the stomach, contracting too energetically, propel the imperfectly elaborated aliment into the intestine, the aliment will there act upon the organ as an irritating foreign body, from the organ not being prepared to receive it in the conditions in which it is presented. The organ will rebel against it, and try to get rid of it as quickly as possible. The alimentary mass reaching the large intestine with part of its elements in this state, which, in a normal condition, would have been converted into chyle and absorbed, undergoes a process analogous to that which we see when beef tea or milk enemata are administered. It is, in fact, an error to believe that injections of this kind can be used in place of food. The large intestine is not made for the reception of alimentary substances, until they have been subjected to the previous treatment of digestion in the stomach and small intestines. The large intestine, far from assisting in their absorption, kicks against them (allow me the expression). Their presence excites energetic contractions, excites secretions, and, in a word, acts like a purgative.

Augmented tonicity of the stomach and intestines is a cause, then, of diarrhœa. Diarrhœa thus induced is lienteric; or in other words, the stools contain a certain quantity of the food in the state in which it was eaten.

This augmented tonicity itself is, like the augmentation of secretion of which I have just been speaking, under the influence of the nervous system. When we come to speak of treatment, we shall see that this kind of diarrhœa yields generally in a remarkable manner to narcotic medication. Though the causes which bring into play this increased tonicity generally act directly upon the parts affected—though, to express my idea more exactly, we must seek for the starting-point of that form of diarrhœa now under consideration in the stomach or in the small intestine, the starting-point is also often found in the large intestine and in its lowest part.

In this lecture, I have already explained to you that irritation of the extremity of a canal is sufficient to cause irritation of the entire canal; and I have referred to what takes place in different secreting glands, to illustrate what occurs in respect of the liver in intestinal catarrh. Similar phenomena are observed consequent upon irritation of the lowest part of the large intestine. This irritation is transmitted by sympathy from the rectum to the colon, and from the colon to the small intestine. Does a day pass in which we do not meet with cases proving the truth of this statement? Is not this the explanation of the manner in which a lavement acts? Certainly, two hundred or three hundred grammes of water injected into the rectum, do not pass very far up the large intestine, but they are nevertheless sufficient not merely to cause it to contract, but likewise to induce contractions in the entire intestinal canal. An example of a very limited local irritation, propagated by sympathy to a great extent of surface, is afforded by intestinal contractions and frequent stools being excited by the introduction of a simple suppository into the anus. This is the mode of action of hæmorrhoidal tumors, the presence of which will not only produce tenesmus, but also frequent diarrhœal stools.

You can now understand how such a lesion as ulceration of the rectum, or chronic inflammation, may cause obstinate diarrhœa, which will not yield till the treatment has been made to bear directly upon the local condition giving rise to it.

The *sixth species* of diarrhœa of which I have to speak, is *diarrhœa originating in indigestion.* It is not an unusual affection in adults, but it is more frequent in children, particularly in infants at the breast.

Although—as I have already said over and over again—the stomach is exceedingly tolerant of very course ingesta, its tolerance has limits; and sometimes, it rebels against its contents when they are excessive in quantity or unsuitable in quality.

Under such circumstances, the stomach will always endeavor to get rid of contents which incommode it. Substances which it has failed to elaborate, or which it has only imperfectly elaborated, will be rejected through one or other portal—through the cardiac or pyloric opening: they will be vomited (perhaps the most fortunate alternative), or they will pass downwards into the duodenum. If they pass into the duodenum, they will excite abnormal secretions, and peristaltic movements, in virtue of the mechanism which I described when speaking of diarrhœa from excess of tonicity, the result being diarrhœa.

Excess in the quantity of food may lead to this result. To select one of the simplest possible examples: nothing is more usual than to see diarrhœa supervene in infants nursed by women whose milk is very abundant and rushes too quickly into the breasts. This disorder of the bowels, to use the common expression, neither arises from the food being of bad quality, nor from the stomach being in an unfit state for its reception, but from the food having been taken in too great quantity at one time. I have selected this illustration, because it gives me an opportunity of putting you on your guard against a mistake which is often committed. A child suckled by a woman having every appearance of being a good nurse has diarrhœa: the family, and sometimes the physician, are in haste to change the nurse, when nothing more is required than to order that the infant be not allowed to suck too long at one time.

Though diarrhœa from indigestion is less common in adults, it is also observed in them pretty frequently.

While in adults, as in children, it may depend on excessive alimentation—*diarrhœa ab ingluvie*—it may likewise depend on the bad quality of the food, and this bad quality may be either absolute or relative.

Every one knows what is meant by food absolutely bad in quality; but it is necessary to explain the meaning of the food being relatively bad. It is a fact generally admitted, that certain aliments and drinks which agree perfectly well with some persons are not borne by others; while the same persons who cannot bear these tolerate quite well the dietetic articles not supported by the other individuals. These stomachic antipathies are so essentially special to the individual, that it is impossible to lay down any rules on the subject; and it is only by personal experience we discover what will and what will not agree with the stomach.

In speaking of dyspepsia, I have already stated the practical conclusions to be deduced from these data: I called your attention to the unfortunate tendency which we all have to regulate the diet of our patients in accordance with our own tastes and digestive aptitudes. The importance of the subject justifies my recurring to it to-day.

I knew a man who suffered from diarrhœa for years, notwithstanding the trial of every sort of treatment, and whose general health was seriously impaired by the affection. The symptoms disappeared, as if by enchantment, upon the patient, of his own accord, discontinuing tea to breakfast, which for twelve years he had been in the habit of taking.

I attended the family of a ship-builder of Havre whose children were unable to tolerate milk for the first seven years of life. A succession of nurses was tried for all of them: lactation with the milk of the cow, the goat, and the ass, was also attempted: but all proved futile. A few mouthfuls of any kind of milk at once caused diarrhœa and vomiting. It became necessary to have recourse to farinaceous drinks, such as decoctions of grits and pearl barley: by this regimen, these children were reared as successfully as others fed in the usual way. This was certainly a very rare exceptional case; for, as a rule, diarrhœa supervenes in infants when a premature attempt is made to feed them with farinaceous food in place of milk, their natural aliment. When I come to speak to you on the subject of weaning, I shall have to return to this point, and to treat it with all the fulness which it merits.

Here, I conclude my remarks on the different forms of diarrhœa, or rather upon the different modes in which they are produced. I intentionally omit speaking of diarrhœa caused by organic disease, reserving that subject for a special lecture, materials for which will be furnished by the cases of several of our patients affected with chronic diarrhœa. I shall merely add that all the forms which I have discussed are far from presenting themselves with the simplicity which I have assumed in my descriptions, for the purpose of enabling you the better to appreciate their causes: sometimes, they may present this simplicity, but in general, they have not that distinctiveness of character with which I have invested them. It is for the physician in each case to disentangle the predominating element as the symptoms evolve.

Gentlemen, all artificial divisions of disease are devoid of interest, if they lead to no therapeutic results. In establishing these divisions, my aim has been to simplify the treatment of diarrhœa, an affection the cure of which is too often attempted by one and the same routine of means, irrespective of the diversity of causes in which it originates.

In treating *catarrhal diarrhœa*, we must always bear in mind that the *catarrhal* element is the same in character as in ocular, nasal, bronchial, urethral, or intestinal catarrh; that it is impossible to predict its duration; and that here the *specific* element plays an important part. Simple coryza is an affair of a few days; but syphilitic coryza is essentially chronic. The pulmonary catarrh of measles is a transient affection, while the catarrh of influenza long and obstinately resists all our therapeutic efforts: an ordinary catarrh, though ceasing more quickly than the bronchial affection of influenza, has nevertheless a very uncertain duration.

So is it likewise with intestinal catarrh. Diarrhœa consequent upon a chill is, in ordinary circumstances, a very transient affection. The treatment is simply dietetic. All required is that the patient for a day or two be put on light food in the form of soups, so that the intestines, having little to do, may be allowed to rest; and thus the symptoms will spontaneously cease.

Cases, however, occur in which it is difficult to carry out this regimen. I refer to cases in which the catarrhal affection, apparently localized in a particular part of the intestinal canal—at the end of the ileum or beginning of the large intestine—causes no loss of appetite, the stomach not being disturbed in the performance of its functions. The expectant system and low diet will certainly accomplish a cure, while ingestion of food will keep up, and may even increase the disorder.

Under these circumstances, nevertheless, it is proper to assist nature; and I do not know of any medication so decidedly calculated as the substitutive to accomplish this object: the best remedies, I mean, are *purgatives*. The selection of the purgatives to be employed is not a matter of indifference: those to which we ought to have recourse are the sulphate of soda, the sulphate of magnesia, and the salts of Seignette, which is the double tartrate of potash and soda. Patients affected with this kind of diarrhœa ought to take in the morning (fasting) from 25 to 40 grammes of one or other of these medicines: the result will be a temporary augmentation of the intestinal flux, but generally at the end of twenty-four hours the symptoms will have entirely ceased.

When the catarrhal inflammation has lasted a little longer, when it has acquired (if I may use the expression) a right of domicile, the substitutive medication is still indicated. If the stools show superabundance of bile, if, at the same time, the tongue is saburral and coated with a thick yellow fur, if there be loss of appetite and a feverish condition, emetics are specially indicated; and the emetic assuredly the most efficacious is ipecacuan, given according to the plan which I have already formulated to you.

The patient is allowed to rest the day after the emetic has been given, and then, on the following day, he takes a saline purgative. Emetics and purgatives are topical irritants, and act simply by substituting for the catarrhal inflammation another and a special inflammation which yields spontaneously much more quickly than that which preceded it. What takes place in the morbidly affected mucous membrane of the digestive canal is exactly what takes place when caustic collyria, nitrate of silver, sulphate of copper, sulphate of zinc, or acetate of lead, are employed in the treatment of catarrhal inflammation of the conjunctiva with a view to substitute for it an inflammation excited by the topical agents, and which will spontaneously cease.

Should the diarrhœa have lasted ten or fifteen days, the saline purgatives ought to be administered in another manner. On the first day, for an adult, I prescribe 25 grammes [6½ drachms] of Glauber salts, and on the five, six, or seven following days I give 10 grammes [2½ drachms]: under this treatment, the patient will come to have not more than one or two diarrhœal stools in a day; and sometimes, even constipation supervenes. The treatment must then be stopped. To children, I prescribe the salts of Seignette, giving 5 or 6 grammes on the first day, and only 3 grammes on the following days.

The substitutive method is likewise available when the catarrhal inflammation has assumed a still more chronic form. The medicinal substances to be employed are, however, not those of which I have been speaking. Though, occasionally, saline purgatives are useful, mercurials are much more efficacious. I prescribe from five to ten centigrammes of calomel divided into eight or ten parts, one of which I order to be taken every hour for eight or ten consecutive hours. The same medication may be repeated daily, for not more than three or four days, care being taken to observe its action and avoid giving it up to the point of salivation; for when the gums become sore and swollen from calomel, a special form of diarrhœa sets in, in which the stools are of a greenish color, and the flux much more obstinate than that which it is wished to subdue.

At the end of three or four days, it is time to stop this treatment. Sometimes, the symptoms are definitively subdued: the desired modification in the condition of the mucous membrane of the intestine is produced by the calomel, just as a modification of the state of the ocular and nasal mucous membranes, when catarrhally affected, is produced by mercurial topical applications. Generally, this treatment does not prove sufficient in itself; and to render the cure complete, it is necessary to give a neutral salt.

In place of calomel, you have not unfrequently seen me give *hydrargyrum cum cretâ.* To children—for whom I prefer this preparation—I give, for one, two or three days, twice a day, from five to fifteen centigrammes.

To adults, I give from 10 to 25 centigrammes of the English blue pill mass in the evening, following up this dose next morning by a saline purgative.

When the diarrhœa has resisted all other measures, you have more than once heard me prescribe a pill in which I combine calomel, opium, and ipecacuan. This is my formula:

Ipecacuan, ...	2 centigrammes.
Calomel, ...	5 milligrammes.
Extract of Opium, . . .	5 milligrammes.

To be made s. a. into one pill.

During the twenty-four hours, the patient takes, in the interval between meals, from one to three of these pills. This medication is continued for from five to ten days. If it be longer continued, the mercury almost invariably acts upon the mouth, a result which ought to be guarded against for reasons I have just stated.

I very frequently employ the crystallized nitrate of silver. It was long ago recommended by Boerhaave as a drastic purgative in dropsy; but I use it as a substitutive remedy in rebellious catarrhal diarrhœa. I make a pill containing in solution one centigramme of the lunar salt: this solution is dropped on a quantity of crumb of bread, tragacanth, or starch, sufficient to make a pill. From four to ten such pills may be given daily, during eight or ten days, as much as possible in the intervals between meals. Neither nausea nor any other disagreeable consequence results from the use of this remedy. In some cases, like saline purgatives, it temporarily increases the diarrhœal flux; but, as a rule, it promptly arrests the diarrhœa.

It is principally, however, for the treatment of chronic, tuberculous diarrhœa (regarding which I shall have to address you in a separate lecture), and for other forms of intestinal inflammation that the nitrate of silver administered both by the mouth and in lavement ought to be reserved.

Of all the remedies employed to cure somewhat obstinate catarrhal diarrhœa, the subnitrate of bismuth is that which I most frequently have recourse to: I generally give it along with prepared chalk, which is the precipitated carbonate of lime. This prescription proves useful, and never produces any bad effects. The powder which I usually order consists of equal parts of chalk and bismuth, from 4 to 10 grammes of each; but much larger doses may be given. One of my colleagues, Professor Monneret, administers it *larga manu*—in tablespoonfuls—without ever seeing the very slightest inconvenience result.

The English chalk mixture produces the same effects: it is composed of 30 grammes of prepared chalk, and 60 grammes of a weak infusion of mint leaves, aromatized by the addition of 30 grammes of orange flower syrup.

These preparations are often in themselves sufficient to cure catarrhal diarrhœa; but it likewise often happens that they do not act beneficially, unless the intestinal inflammation has been previously modified by a purgative.

Some persons are seized with diarrhœa whenever they are exposed to the slightest chill. Patients of this description derive a marvellous amount of benefit from *hydrotherapy*, and, when it can be had, from *maritime hydrotherapy:* this treatment tonifies their whole system, and enables them to resist variations of temperature, without contracting the intestinal catarrh from which, under similar circumstances, they were previously in the habit of suffering.

I was speaking to you about specific catarrhal affections: and at the beginning of this lecture, I told you, that the specific element showed itself quite as much in intestinal as in other catarrhs. I referred to the diarrhœa dependent upon the herpetic diathesis, as well as to certain forms of bronchitis and coryza. These diarrhœal affections occur, at longer or shorter intervals, in persons subject to cutaneous eruptions. Here, *sulphurous remedies* are exceedingly useful; and of them the best are the natural mineral waters, such as those of Luchon, and Aix in Savoy, but particularly the former.

Arsenical treatment will also prove of great service in these cases: to prove effective, however, it must be long continued. In employing this treatment, gentlemen, I cannot too strongly urge you to formulate your own prescriptions, so as to be quite sure of what you are about. The arsenical solutions of Pearson and Fowler require to be administered with the utmost caution, for the slightest error may produce most serious symptoms. I prefer the following solution:

Arseniate of Soda, . . .	5 centigrammes.
Water, ...	125 grammes.

Each teaspoonful of this solution represents about 2 milligrammes of the arseniate. The dose may be increased up to one centigramme.

The patient takes this solution daily for a month, when it is suspended for 10 days, to be again resumed for another month; and I continue to repeat it, at similar intervals, for a long period: for do not forget the precept, that for chronic diseases (and as such we must regard all diathesic maladies) chronic treatment is requisite. A combination of this arsenical medication with the sulphurous treatment generally cures the kind of diarrhœa of which I am now speaking.

The treatment of *sudoral diarrhœa* is naturally suggested by what I have said regarding the causes which excite increased intestinal flux. The preventive measures consist in not covering the body too warmly, and in abstaining from violent exercise after eating. When the affection actually exists, cool drinks and light food will generally suffice to accomplish a cure. That, therefore, is a subject into which it is unnecessary to enter at greater length.

The treatment of *nervous diarrhœa* is less simple, and requires that I should go into some details. In this affection, *obtunding and antispasmodic remedies* are peculiarly indicated. *Opium* takes the first place in this class of medicines: but it is a remarkable fact that almost equally high in the list is *belladonna*, which perhaps you will be surprised to hear me laud as a curative agent in diarrhœa, knowing, as you do, that along with the other physiological properties which it has in common with henbane and other medicinal *solaneæ*, it relaxes the bowels, and is consequently prescribed with advantage in some kinds of constipation. The contradiction which seems to exist between the opposite results obtained from the same remedy is only apparent, as you will easily perceive by reflecting upon the mode of action of belladonna.

In virtue of its obtunding powers, it is, according to the nature of the case in which it is given, either purgative or antidiarrhœic. When constipation depends upon a sort of spasm of the intestine, belladonna relieves the spasm, and so acts as a purgative: when diarrhœa of the form now under consideration depends upon exalted irritability and increased nervous sensibility of the intestine, belladonna soothes this irritability and calms this sensibility, in the same way that it arrests the lachrymal flux in supraorbital neuralgia by calming the neuralgia which was its exciting cause.

In nervous, but only in that kind of diarrhœa, belladonna is of unquestionable utility: when the cause is a catarrhal phlegmasia this medicine aggravates the symptoms. It may often be very advantageously substituted for opium; but like it, it requires to be managed with extreme prudence, and only to be given internally in small doses, such as from one to three centigrammes in the twenty-four hours, and distributed in several pills: when the diarrhœa is accompanied by gastralgic and enteralgic pains, its use may be restricted to frictions of the abdomen, particularly to frictions over the pit of the stomach.

For reasons similar to those now stated, antispasmodics are likewise admirable remedies in nervous diarrhœa. Of this class, *ether* is assuredly the most powerful agent; and it can be very conveniently administered in the now commonly used gelatinous capsules.

Nitrate of silver, which I mentioned as a useful remedy in catarrhal diarrhœa, is likewise useful in nervous diarrhœa, its action, however, in the latter, being that of an antispasmodic, and not that of a substitutive. This treatment (devised by Graves) requires to be directed with circumspection: it must not be continued for more than four or five days consecutively; and within the twenty-four hours, there must not be given more than four pills, each pill containing one centigramme of the salt. When combined with belladonna or opium, nitrate of silver has an exceedingly good alterative effect in those attacks of diarrhœa in which borborygmi supervene in nervous women and hypochondriacal men.

Although neuralgias of the abdominal viscera—gastralgia, enteralgia, and hepatalgia—are usually associated with obstinate constipation, it is not a very rare occurrence for them to occasion diarrhœa, the profusion of the flux being proportionate to the intensity of the pain. It is in such cases that we interpose usefully by administering opium and antispasmodic remedies, the *modus operandi* of which we can explain. Benefit is also derived from the essential oil of turpentine, which is a very powerful remedy in a great many forms of neuralgia, although I cannot give you an account of the way in which it acts.

Here, too, opium must be administered with great circumspection; for if the doses are too large, the consequence is—if I may so express myself—an extinction of the aptitudes of the stomach, a stoppage, or at least an impediment to digestion—indigestion.

In administering the essential oil of turpentine, certain precautions likewise require to be observed. Above all, we must avoid the old practice of giving it in the form of emulsion; for so administered, it will irritate the upper and least tolerant parts of the digestive canal, the pharynx and œsophagus. The best, nay, let me say the only proper mode of giving turpentine is in gelatin capsules, each capsule containing fifteen or twenty drops. When thus administered, patients can take from 100 to 150 drops a day, without experiencing any inconvenience except eructations, which, however, are less frequent when the medicine is given in capsules, particularly if taken immediately before a meal. It is very unusual for turpentine to occasion vomiting.

To put a stop to, and also to prevent a recurrence of nervous diarrhœa, *hydrotherapy* and *maritime hydrotherapy* are beneficial, as in catarrhal diarrhœa; but my former remark must be remembered, that sea-baths are useful only when they are of short duration. It has often happened in my experience that patients who have during one season derived the benefit which I looked for from sea-bathing, have in the following year experienced no advantage from it, simply because they thought they might with impunity deviate from my prescription, to the effect, that they were to remain only a very short time in the water.

The treatment of the fourth kind of diarrhœa—that in which the intestinal catarrh is the result of an abnormal secretion from the digestive canal and its annexes—is at once that of original catarrhal diarrhœa and of nervous diarrhœa.

The inflammatory disturbance being more specially localized in the large intestine, it is necessary, independently of saline purgatives, to have recourse to topical treatment, acting directly upon the affected part. This topical medication consists in the administration of lavements.

We generally take up very erroneous notions as to the way in which lavements act. When, at the anatomical table, we measure the capacity of the large intestine, it seems as if it might quite well contain three or four litres of fluid between the anal orifice and the ileo-cæcal valve. This may be perfectly possible in the dead body, because by death the intestine has entirely lost its contractility, but it is otherwise during life, the contractility existing. Dr. Briquet, my honorable colleague of the Hôpital de la Charité, has discovered, upon examining the bodies of persons who have died soon after taking lavements, that the 500 grammes of water of the lavement had gone as high as the cæcum, and had, in some cases in which force had been employed in injecting the liquid, entered the small intestine, having forced the ileo-cæcal valve. Dr. Briquet's observations show, that while lavements do not always ascend so high up in the intestinal canal, they generally do so; and the fact is important, inasmuch as it proves, that we may hope to be also able to introduce as high up in the canal the different topical agents by which we may try to modify the state of the inflamed organ. The topical agents most suitable for the attainment of this object are the neutral salts: when the affection is obstinate, caustics, such as the nitrate of silver and the sulphate of copper, are indicated; and when the catarrhal affection is associated with the herpetic diathesis, lavements containing from five to thirty centigrammes of the sulphide of potassium or sodium will prove of great benefit, by acting upon the mucous membrane of the intestine in the same way that sulphurous lotions act on the skin in herpetic affections.

The treatment of *diarrhœa arising from excess of tonicity* consists almost wholly in administering *opium*. There is no medicine from which good results can be more easily obtained; nor is any medicine more improperly employed. This arises from our impotence being concealed by opium giving temporary relief from pain, when it produces no curative effect on the malady. Herein lies an evil tendency against which we know not how to be on our guard: forgetful of the *quidquid meditetur et faciat, si natura non obtemperat naturæ non imperat*, the physician believes that the disease cannot baffle him: when he is unable to put an end to it, he tries to keep it quiet, though it be only for a very short time. In general, opium is the knout most willingly employed to stifle the manifestations of the disease. But beneath the compulsory quiet induced by the opium, the disease will continue, and will be all the more dangerous, that it is so masked by narcotism that its characteristic symptoms can with great difficulty be recognized. Alarmed at the symptoms which he has been the means of setting up, the practitioner completely abandons the use of a remedy which he had not the ability to use with moderation, and so loses the very great benefit which he might have derived from its judicious employment. Opium is the most powerful remedy we possess for the form of diarrhœa now under consideration; but to obtain success from it, we must know how to administer it. Given in small doses it does much good, and not the least harm. In my lectures on dyspepsia, I insisted at considerable length upon this point. I said that in some cases five centigrammes of the extract were often sufficient to produce the best results in affections of the digestive apparatus.

There is no medication in which it is of more importance to take into account, not only the idiosyncrasies of the patient, but also the exact time for administering the medicine. Recall to your recollection the two women who were patients in St. Bernard Ward in whom a single drop of laudanum produced narcotism, not on one occasion only, but every time we renewed the treatment. In infants at the breast, half a drop taken in the twenty-four hours sometimes induces similar symptoms.

The economy is most tolerant of opium when it is administered immediately after or during meals—in this sense, that it is least apt to induce drowsiness when not received by an empty stomach.

Administer opium in small doses. One drop given to an adult, and one-quarter of a drop to an infant, fifteen or twenty minutes before eating, will calm the state of erethism of the digestive canal and prevent a diarrhœal flux, which, when it comes on two or three hours after taking food, proceeds from a morbid excess in the peristaltic movements.

It is only by opium that this kind of diarrhœa can be calmed and cured. In cases in which opium alone is insufficient, it will at least assist other medicines by allowing their presence to be tolerated so as to remain longer in contact with the intestines—medicines such as the subnitrate of bismuth, chalk, nitrate of silver, and calomel, which prove beneficial by their modifying action upon the mucous membrane.

When diarrhœa has as its starting-point irritation localized in the lower portion of the large intestine, as is the case after a dysenteric attack, the treatment required is essentially topical. Lavements containing nitrate of silver, sulphate of copper, or better still, lavements consisting of a sort of hasty-pudding mixture of subnitrate of bismuth, prove of marvellous efficacy.

When the contractility of the parts renders them intolerant of these remedies, laudanum will intervene most beneficially in doses of from one to fifteen drops, according to the nature of the case; it will calm excessive irritability, and so allow the lavement to be retained.

I need not at present stop to discuss the diarrhœa which arises from indigestion. More interesting topics on which to address you are the diarrhœa of prematurely weaned infants, and the infantile cholera which so often accompanies premature weaning. Several such cases which we have had in our clinical wards I propose to make the subject of a special lecture.

Here, I stop to-day. At our next meeting, I shall address you on chronic diarrhœa, illustrating my remarks by cases at present in our wards.

CHRONIC DIARRHŒA.

Diarrhœa complicated with Fever and Nocturnal Sweats is almost always associated with Tubercle.—Chronic Syphilitic Diarrhœa.—Herpetic Diarrhœa.—Chronic Diarrhœa depending upon Simple Chronic Catarrh of the Intestine.—Chronic Diarrhœa the Result of Insufficiency of Food.—Treatment varies according to the Cause.—The Use of Raw Meat.

GENTLEMEN: In bed 27 of St. Bernard Ward lies a woman who has suffered from diarrhœa for the last eight months. Every kind of treatment has been tried with a view to stop the intestinal flux; but it has never yet been checked for more than two days. When I saw the patient for the first time, she had very evident signs of peritonitis; the abdomen was hard and painful, giving everywhere on percussion a dull, or at least an obscure, sound. Some days before admission to the hospital this woman had had acute bronchitis accompanied by intense fever.

The opinion I formed was that the diarrhœa depended upon chronic enteritis, complicated, as it often is, with chronic peritonitis. Upon inquiring into the previous history of the case, I ascertained that from the very first appearance of the symptoms, she had had night-sweats, evening fever, and considerable wasting. I concluded that she was the subject of a tuberculous abdominal affection.

I was not led to this conclusion by the obstinacy of the diarrhœa, but by the existence of nocturnal sweats and fever. The state of the respiratory organs did not present any significant indication, for although for some time past there had been such an amount of cough as to arouse our fears, repeated and very careful examination by auscultation and percussion did not reveal the slightest pulmonary hepatization. Mucous râles, characteristic of bronchitis, were heard disseminated throughout the whole chest.

It was, then, the coincidence of night-sweats and fever with diarrhœa and peritonitis which led me to the conclusion that the patient was under the influence of the tuberculous diathesis. In forming my diagnosis I was relying, so to speak, upon the long and valuable experience of Chomel. How often has my lamented predecessor repeated to crowded audiences in this place that *chronic diarrhœa, accompanied by fever and night-sweats, is an almost certain sign of tuberculization*, a proposition which I have had many opportunities of verifying in the course of my medical career.

An additional confirmation of the proposition is afforded by the case now before us; the patient has succumbed after languishing about six weeks in hospital. At the autopsy we found tubercular deposit on the surface of the peritoneum, and in the lymphatic glands of the mesentery. Tubercular matter was also found upon the pleuræ and in the bronchial glands; but, strange to say, no trace of tubercle existed in the parenchyma of the lungs. This then is an additional case to add to those exceedingly rare cases which constitute exceptions to the famous law formulated by Louis, to the effect that *whenever tubercle is found in one viscus, it will also be invariably found in the lungs.* Here, however, I must remark that this rule, though generally true in respect of adults, is not applicable to children. In them, it is exceedingly common to meet with tubercular lesions of the encephalon, abdomen, and even of the bronchial glands, without finding any in the lungs.

We have at present, in the same ward, in bed 28, another patient suffering from chronic diarrhœa. In her, the symptoms date back six months. From that time she began to lose strength, and visibly to grow thin. Her breathing was oppressed, and she became winded on the least overexertion; she had nocturnal sweats, and every morning (about seven o'clock) a paroxysm of fever, setting in with rigors. At the time of my visit the fever still existed. I observed in this patient a peculiar formation of the fingers and finger-nails; the nails, particularly of the thumbs, were beginning to grow inwards. You are aware of the value which Hippocrates attached to this sign; in his second book, "De Morbis," he says: "*Quum quis tabescit, ungues contrahuntur.*" I think that this patient is affected with chronic tuberculous diarrhœa. Although auscultation does not enable us to detect any sign of pulmonary disease, I am convinced that this woman is phthisical; and I certainly believe that, although we may not be able to find any thoracic lesions, there exist abdominal lesions similar to those detected in the other case of which I have just given you the history.

However decided my convictions as now stated were in this case, they have not caused me to lose courage; I have striven, and I shall still strive, to subdue the symptoms, hoping to moderate, should I fail to cure them.

My aim, first of all, has been to put an end to the quotidian fever. In giving cinchona, however, I have been well aware that the paroxysms of fever were not those over which that wonderful medicine exercises an influence. I knew very well that the fevers which respond to the cinchona treatment are seldom quotidian and are generally diurnal—that intermittent night fevers being generally symptomatic resist antiperiodic remedies. I nevertheless tried the sulphate of quinine, and I gave it in high doses. This treatment did not modify the fever, which unfailingly returned every night notwithstanding scrupulous exactitude in administering the sulphate of quinine. But to my extreme surprise, the diarrhœa entirely stopped for more than a month.

The patient at the same time regained appetite, strength, and even a certain degree of plumpness. Digestion, nevertheless, remaining somewhat painful, slow, and laborious, accompanied, likewise, by weight in the stomach after eating, you saw me prescribe hydrochloric acid, after having in vain employed alkalies. This woman took daily, at the beginning of her breakfast and dinner, three drops of the acid according to the plan which I described to you when lecturing on dyspepsia. The digestion became easier in consequence of this treatment. Nevertheless, though her state is improved, my unfavorable opinion of her case remains unchanged, because the fever and night-sweats continue. Sooner or later this patient will sink from pulmonary phthisis.

In the bed next to that which she occupies—in bed 22—there died, a short time previously, a poor woman who also was exhausted by an obstinate diarrhœa of two years' duration. In this case, however, the intestinal flux had no relation to any such causes as those which existed in the other two patients. When she came into hospital she was exceedingly emaciated, and so anæmic that the first question I addressed to her was an inquiry as to whether she had profuse uterine discharge. The extreme paleness of the integuments, and the bellows-murmur in the vessels, warranted my suspicions. They were really, however, unfounded.

In this case I set aside the idea of tuberculous diathesis, because fever and sweating were absent, and the state of the respiratory organs was good. The history obtained was sufficiently ample to enable me to explain the symptoms. The patient stated that two years ago she was living in Champagne, when she was obliged, by the pressure of poverty, to seek a home elsewhere. Accompanied by her husband and one child, the sole survivor of six, she came to Paris to seek a subsistence. In this attempt she failed, and in place of obtaining the hoped-for relief, she got involved in still deeper misery. Her husband fell ill; she, he, and the child, had nothing to eat save the ration of bread allowed by public charity. This state of matters has continued for two years; and consequently, for the last two years, this poor creature has been wasting for lack of sufficient food.

The diarrhœa then, in her case, arose in the same way that we see it occur in animals allowed to die from inanition. It might be supposed that restorative diet, substantial alimentation, would have put an end to the symptoms. Unfortunately, the problem which had to be solved was not by any means so simple. A result had occurred such as that which happens in all similar circumstances. Deficient aliment had produced impoverishment of the blood. In its turn the impoverishment of the blood had caused alteration of the gastric, intestinal, hepatic, and pancreatic secretions, so that digestion was imperfect, even when the food was highly nutritive and of faultless quality.

We were evidently shut up within a vicious circle. It was necessary that we should feed the patient, but the food, however good it was, caused a succession of attacks of indigestion. Not only was the diarrhœa persistent, but taking the smallest quantity of food caused vomiting. I tried to assist nature by sometimes giving hydrochloric acid, and at other times opium by itself, or in conjunction with astringents: I likewise administered ferruginous remedies and alkalies: in a word, I put in requisition many modes of treatment. My efforts, however, were unsuccessful: ere long hectic fever was kindled, and the patient died.

At the autopsy no appreciable organic lesions were found, except some small superficial erosions in the large intestine. The spleen, liver, and lungs, presented nothing abnormal except a pale appearance.

Such of you as have been attending my hospital visits for some time past will remember the history of a young woman long resident in our wards, whose case—interesting for several reasons—has been recorded by Drs. L. Gros and Lancereaux.* I refer to the woman who latterly occupied bed 34 of St. Bernard Ward.

I am not going to narrate the case in all its details. I will only remind you that the patient came into hospital for an obstinate diarrhœa which lasted thirteen months, was complicated with lientery, gastralgia, and vomiting; and which, after resisting many medicines, ultimately yielded to mercurial treatment. This woman's diarrhœa, the cause of which I was long in discovering, was the first, I may say the only manifestation of constitutional syphilis; and if we may believe the patient's statements, the venereal taint had not been indicated otherwise than by the appearance, two months previously, of a greenish vaginal discharge and acute pain in making water.

I was led at last to the correct view of the case by the patient suffering from pains in her head, notably aggravated at night, principally osteoscopic and seated in the course of the membranes; by her having tumors over both tibiæ, then over the right radius, and a little later over the left humerus; finally, a gumma [*gomme*], which ulcerated, on the calf of the right leg placed beyond question the accuracy of the diagnosis.

The diarrhœa, I say, yielded to mercurial treatment. Van Swieten's liquor, which I first tried for twelve days, and other mercurials afterwards administered internally, were not supported: sublimate baths were consequently resorted to, and under their influence all the symptoms rapidly yielded. After a residence of twenty-three months, the patient went out completely cured.

I have been desirous, gentlemen, to lay these facts before you in juxtaposition with each other, that I might once more show you how different, according to the nature of the case, is the diagnosis, prognosis, and treatment of a phenomenon the signification of which appears, when looked at by itself, to be identical.

It appears, then, that chronic diarrhœa, whether dependent upon intestinal catarrh, or on a more deeply-seated phlegmasia, may result from very different pathological conditions.

In the first two cases which I cited, the intestinal flux evidently proceeded from the tuberculous diathesis: the recurrence of the fever and nocturnal sweats every twenty-four hours, and their resistance of all treatment, led me to that conclusion. Unfortunately, in respect of the patient of bed 27, the autopsy confirmed the accuracy of my opinion. Though anatomical proof is wanting in respect of the patient of bed 23, the law laid down by Professor Chomel is of too general an application to permit us to hope that we have to do with an exception to it.

This is a point that you must constantly bear in mind. When—particularly in persons about the age of puberty—you have to treat a case of chronic diarrhœa complicated with fever and night sweats, be reserved in announcing your diagnosis. Do not expect to master the malady too easily; and if you succeed in moderating or modifying it, do not count on the amelioration being of very long duration. Beware of exciting in the relatives hopes in which you cannot participate. Generally, indeed I might say always, in such cases, there exists the tuberculous diathesis; sooner or later, it will explode, and the patients will succumb. Foreseeing the danger, you will not be exposed to annoying mistakes. When you have exhausted all the resources of your therapeutic arsenal, you will not be surprised at having failed to subdue a disease which from its nature is incurable.

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* Gros et Lancereaux: Des Affections Nerveuses Syphilitiques. Paris, 1861.

The case of our patient of bed 34 is an example of the rare exceptions to Chomel's law which I have referred to as being occasionally met with. During the first period of this young woman's stay in hospital, I observed that she had an intermittent fever of quotidian type, which led me to think that the diarrhœa might be dependent on the tuberculous diathesis. Although I examined the chest daily with some care, I was unable to detect any sign of pulmonary solidification: and there was neither cough nor expectoration. The fever soon assumed a well-marked tertian type, a circumstance which was in itself sufficient to exclude the supposition that the tuberculous diathesis existed; symptoms which showed themselves at a later date dispelled all my doubts, and inspired the hope that we should soon cure a diarrhœa which was symptomatic of a malady usually very amenable to treatment. Mercurials fulfilled the therapeutic indication, just as sulphurous and arsenical preparations would have fulfilled it had the intestinal flux been dependent upon a herpetic diathesis.

The chronic diarrhœa with which the patient of bed 23 was tormented being independent of diathesis, we should have been justified in hoping for recovery, had not the symptoms dated back two years, and had we not had to do with an utterly broken down constitution, which made no response to any dietetic or medical treatment which was tried. The unfortunate woman, from the long period which her alimentation had been insufficient, was exactly in the position of an animal perishing from inanition. The blood being deprived of the materials required for its renovation, the intestinal secretions were vitiated, and the digestive functions exhausted, so that we could not count on treatment which, under apparently similar circumstances, is unquestionably useful—that is to say, in chronic diarrhœa unconnected with organic lesion, such for instance as occurs in the convalescence from serious and protracted maladies. The treatment to which I refer consists in *nourishing the patients with minced raw meat.* When I come to treat of the diarrhœa of infants at the period of weaning, I shall revert more in detail to this plan of treatment, which may appear strange to such of you as have not seen me employ it.

Fifteen or twenty years ago, I was summoned to a lady of twenty-three or twenty-four years of age who had been suffering for six months from an intractable diarrhœa. I was one of four physicians called to this consultation. After a careful examination of the patient, and a most minute inquiry into all the circumstances, we entered upon our deliberations. I cannot now recall the conditions under which the diarrhœa had supervened: I only recollect that from the long continuance of the diarrhœa, we suspected the existence of the tuberculous diathesis. However, the general aspect of the patient, her antecedents, the absence of fever and of all symptoms characteristic of phthisis, caused us to reject the tuberculous hypothesis, and to consider the diarrhœa as a local affection, the result of chronic irritation of the intestinal mucous membrane. All the methods of treatment usually adopted in similar cases had been tried; and they had all completely failed. When each of my brethren had stated the treatment which he recommended, I gave my opinion as to the plan which ought to be adopted. I stated that the resources of pharmacy had been exhausted, that not only could no good be obtained from the farther trial of drugs, but that I looked on pharmaceutical intervention as mischievous. It would, therefore, I argued, be necessary to rely entirely upon dietetic treatment. My brethren replied that regimen had been tried in every form, without the least impression having been made on the disease, that the patient had a great disgust for every kind of food, and that every kind of aliment was at once rejected by the stomach. I then proposed the use of *raw meat*. My proposal was received with skeptical derision ; but I was not dismayed, and maintaining my opinion, supported by a case in which the raw meat system had proved wonderfully successful, I begged that it might be tried. The ordinary physician of the family, though equally skeptical with the other two as to the attainment of that success which I hoped for, consented to make the experiment. It then became necessary to induce the patient to accept the proposed treatment ; and this, it was thought, might not be easily accomplished.

Are not the individuals few in number who would at once take to such a diet, though in point of fact the repugnance to it is nothing more than the result of habit? Consider the question, and ask wherein lies the difference between cooked meat and raw meat? Be that difference what it may, it is the result of habit ; and we all know that habit is a second nature difficult to change.

I went to our patient and asked her whether she would have any objection to eat the under side of sirloin of beef "*under done*" [*filet de bœuf peu cuit*]. She replied that she would willingly take it. I then gave my instructions to the cook, telling her to place the meat before a very strong fire for a few minutes, just long enough for the outer layer to be acted on by the heat, the interior remaining absolutely raw. Farther, I directed that before serving the meat so treated, its most cooked part should be removed, the remainder only being minced for the patient's use. All this was done in accordance with my directions ; and on the first day, the lady ate and perfectly digested two slices of raw meat. Next day, she ate three slices : she then took four slices, and at last took a tolerably large daily portion. In less than two weeks, the diarrhœa had ceased, and complete re-establishment of health had taken place. The great advantage of this treatment consists in its at once reconstituting the mass of the blood, so fitting it to fulfil all its functions. In this case, there was no relapse. My stratagem succeeded to the utmost of my wishes.

When this stratagem fails me, there is another to which I am in the habit of having recourse. I give the raw meat under the name of "*conserve de Damas*."* Why this name? I confess that I should be greatly puzzled were I to try to answer the question. In fact, I adopted the name which first suggested itself. The *conserve de Damas* is simply raw meat reduced to a pulp, and mixed with currant jelly or conserve of roses. When I prescribe it, I take care to give notice to the apothecary of what I intend him to give. You can quite understand that remedies of this description can hardly be introduced into the Pharmacopœia. An uninitiated person never recognizes raw meat in this disguised form and therefore generally takes it without the lest repugnance.

It seems extraordinary that stomachs and intestines incapable of bearing or digesting even the lightest kinds of food should so soon become accustomed to aliment of so strong a description. Bear in mind what I said in relation to this point when I was lecturing on dyspepsia.

When a patient complains of disordered digestion, our first idea is to put him on a diet easy of digestion. In certain cases, how can this be done? Food easily digested by some patients is not easily digested by others for whom it is prescribed; and the conclusion naturally arrived at is, that a diet still more rigidly plain must be tried. The patient, however, grows weaker, his dyspepsia increases, and his diarrhœa continues: all the consequences of inanition supervene, the blood becomes impoverished, the secretions of the digestive organs become altered; and to avoid one evil, a greater evil is fallen into.

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* Reveil : Formulaire Raisonné des Médicaments Nouveaux, et des Médications Nouvelles. 2d edition, p. 69. Paris, 1865.

Ten years ago, a very busy and very skilful Parisian physician—a great worker—became dyspeptic. In consequence of a chill, he was attacked with gastric symptoms, which, in the first instance, he successfully combated by restricting his diet. Whenever he resumed his usual regimen, he felt pains which made him return to more moderate fare: he was satisfied to live on beef tea and diluted milk. Under this system of feeding, he soon fell into a state of great debility; fever lighted up, and vomiting supervened. He consulted Chomel, who suspected cancer of the stomach, without discovering, however, any material signs of this disease. Chomel advised the patient to continue the regimen which he had adopted. I was next consulted: I concurred in Chomel's view of the case, though as unable as he had been to discover any distinctive signs of cancer. However, upon obtaining additional information regarding the previous history of the case, it appeared to me that there was some ground for attributing the symptoms to inanition; and I therefore recommended more substantial nutriment. I urged my suffering brother to take a basin of meat soup: this he did in obedience to my wishes, but as he felt the pains more severe on the following day, he lost heart, and resigned himself to die. I endeavored to give him hope, and at the same time urged him to pursue the dietetic course which I had recommended. "You have," said I, "the melancholy conviction that you are a hopelessly doomed man: very well, then, having realized the worst, confide your case to me, and let me do what I please. As a favor to me, I ask you to eat, from this day, the wing of a partridge very slightly cooked." He granted my prayer, without however counting on obtaining the benefit which I promised him from his compliance. To his great astonishment, three hours after his repast, digestion was accomplished, and he felt revived; next day he took double the quantity of aliment, eating two wings of partridge. On the following day, he felt a return of strength. He now became hopeful of recovery, rejecting the idea of cancer, and taking the same view of his disease as I took.

He was soon quite well, and he has ever since remained well. My honorable brother is now in the enjoyment of the same excellent health which he used to enjoy, and he pursues his professional avocations with very great talent and admirable devotion.

Gentlemen, this case shows you that it is frequently impossible to know *à priori* the food which will agree best with a patient. Many persons will digest pork and ham whose stomachs cannot bear a light panada. Do not interrogate me as to the wherefore of these singular diversities, for I know nothing about idiosyncrasies and special aptitudes of digestion. The physician cannot lay down for himself fixed rules whereby to regulate his dietetic prescriptions: he must feel his way by experimental trials, which will occasion neither danger nor inconvenience if judiciously directed.

It is important not to forget the necessity of varying the nature of the alimentation. When a patient continues for more than five or six days to live upon the same kind of food, he is apt to loathe it: his stomach is wearied with it, and the symptoms reappear. It is then supposed that the treatment instituted is unsuitable, whereupon the new direction is abandoned, and the patient is allowed to fall into the old rut whence he had emerged.

Again I repeat, that I have obtained real service in certain forms of apyretic chronic diarrhœa from giving minced raw meat. Alone, I have often found it sufficient to accomplish a cure; but frequently, it has also been necessary concurrently to have recourse to agents of the materia medica. Occasionally, under certain circumstances, it is necessary to give very small doses of laudanum before meals: occasionally also, alkalies are useful, and at other times, bitters or tonics are of service. Nux vomica and its substitutes take the first place as tonic remedies in many cases. There are other such cases, again, in which ferruginous medicines are indicated; as, for example, when the diarrhœa is complicated with great anæmia—whether this be produced by the profuse intestinal flux, or by an impoverished state of the blood, and consequent debility, as is frequently the case in young subjects.

In this class of cases, in addition to the characteristic paleness of the integuments and the great emaciation which accompany the diarrhœa, there sometimes occur sanguineous exudations into the subcutaneous cellular tissue, and there are seen, more or less disseminated over the body, ecchymotic spots. Another very common symptom is œdema of the lower extremities, and even a state of pretty general anasarca; but these dropsical symptoms are unaccompanied by albuminuria.

Cinchona, bitters, and in a special manner ferruginous medicines, will powerfully aid a tonic regimen. When iron cannot be borne internally—a not unusual occurrence—prescribe ferruginous baths, each bath containing 500 grammes of the sulphate of iron.

In obstinate chronic diarrhœa, you will also find hydrotherapy and certain natural mineral waters very useful: you will likewise obtain specially beneficial results from sea-bathing and maritime hydrotherapy.

I must not conclude my remarks on chronic diarrhœa without saying two words upon the good effects of saline purgatives. How often, for example, have you seen me persist with a sort of obstinacy in the use of Glauber salts. Gentlemen, this is a very important method of treatment. I begin by giving not more than 10 grammes of the salt dissolved in a very small quantity of water, and I recommend the patients not to drink for some time after taking the medicine. On the following day, I do not administer more than 5 grammes: and on each succeeding day, for a fortnight, I repeat this dose. If (as is usually the case) the diarrhœa cease, I only give the remedy once in two days, always selecting as the time of administration, the morning before taking food. Should the patients feel great repugnance to the medicine dissolved in water, it may be given inclosed in wafer-paper.

Rhubarb in very small doses—say from 10 to 15 centigrammes—administered in the morning before taking food, is sometimes exceedingly useful.

Lastly, let me recommend to you a combination of remedies which you often see me prescribe in the wards. I prescribe pills, in each of which there are two centigrammes of ipecacuan, half a centigramme of calomel, and half a centigramme of extract of opium: one of these pills is taken morning and evening for five days. I then return to the saline purgatives or rhubarb: and afterwards, I again resume the pills of ipecacuan, calomel, and opium. For two or three months I pursue this routine of treatment, interrupting it, however, occasionally, and always paying great attention to the regimen of the patients.

LECTURE LXXII.

INFANTILE CHOLERA: DIARRHŒA OF CHILDREN.

Infantile Cholera is different from Asiatic Cholera Morbus.—Conditions under which it is developed: influence of Season.—Particularly occurs at the period of Weaning.—Symptoms.—Prognosis.—Treatment.—Diarrhœa of Weaning Infants treated by Raw Meat.

GENTLEMEN: Some days ago, when we were getting into the very hot weather, I said that most probably we should not be long without seeing cases of the disease which in France is called "*choléra infantile*," and which American physicians have described under the name of "*summer disease*." My anticipations have been only too completely realized. The day before yesterday, a child who occupied bed 13 of our nursery ward died, after having suffered for a short time from this disease.

I accept the name "*choléra infantile*" because its use has been ratified by long custom, and because I am opposed to the introduction of new names, when the old ones are familiar and well understood. Were it not for these reasons, I should prefer to call the disease by its American name, because I think it is much more appropriate.

Infantile cholera is essentially different from cholera morbus, though the latter does not spare very young children.

The influence of season, which in America has given this affection the name of the "summer disease," would appear to be its principal cause irrespective of that which belongs to the individual. From the earliest ages, this disease has been observed: during the hot season, it appears every year in every country.

Cholera morbus, which did not make its appearance in Europe till less than fifty years ago, only returns at certain epochs, and its advent is irrespective of season: while it ravages numerous localities it does so, not simultaneously but in succession like epidemics, its cause being as yet unknown. In these respects, the two diseases present fundamental distinctions; and they do not the less differ from one another in respect of the symptoms by which they are respectively characterized.

Asiatic cholera morbus, both in children and in adults, has special features, which we all know how to distinguish from those of cholera *nostras*.* The two kinds of cholera, no doubt, have some symptoms in common, which, if considered separately, might lead us into confusion; but there is something specially distinctive in the aspect of the patient, in the appearance of the tissues, in the changes which take place in the temperature of the skin, in the aggregate of the general phenomena, and in the respective course and gravity of the symptoms.

The same sort of comparison may be instituted between these two diseases which may be made between many others. In the same sort of way, we might compare influenza with bronchitis or simple catarrh; and dysentery with acute colitis. Amid the similarities which these affections present, they show dissimilarities still greater, so that it is impossible to confound them with each other. The points of dissimilarity are so well marked, so clearly defined, that they evidently bear the stamp of specificity.

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* See the article on Choléra Asiatique, by Desnos; article on Choléra Nostras, by Gombault; and article on Cholera Infantile, by P. Lorain: in 7th volume of Nouveau Dictionnaire de Medecine et de Chirurgie Pratiques, published at Paris in 1867.

To follow out the same illustrations which I have already used—simple bronchitis is, at least generally, a mild and transient affection, but when this bronchitis, the result of a cause which we cannot detect, prevails as an epidemic, that is to say, when it is influenza, it assumes an entirely different character in respect of severity and inveteracy. Who does not know, that under these circumstances the intensity of the evil, the high fever, the pains in the back and chest, the general feelings of discomfort and prostration, in a word, all the general symptoms consequent upon an attack of influenza, bear only a slight resemblance to those observed in bronchitis, even in an attack of the most violent character?

Similar remarks are applicable to acute sporadic colitis, and epidemic colitis which takes the name of dysentery. In both, the large intestine is the seat of the characteristic lesion: in both, the stools are composed of bloody, glairy secretions. In sporadic colitis, however, the intestinal disturbance is transient, the accompanying pain and tenesmus are slight, and the fever is moderate: notwithstanding the local symptoms, the general state of the economy is good. In dysentery, while the severity of the local inflammation, generally so intense as to produce some amount of mortification, may to a certain extent account for the severity of the general symptoms, it is, on the other hand, not uncommon for the symptoms to assume a character more formidable than those of acute sporadic colitis, even when the local lesions are insufficient to explain the profound disturbance of the whole system.

In a word, if influenza and dysentery are inflammatory diseases, they are inflammations nosologically the same—the one belonging to the genus bronchitis, and the other to the genus colitis; but this epidemic bronchitis differs as much from hooping-cough, and this epidemic colitis differs as much from common catarrh of the large intestine, as the natural history of one animal or vegetable species differs from another animal or vegetable species of the same genus.

When we read the description left us by Sydenham of cholera as observed by him, and as we find it described by authors who wrote at the beginning of this century, bearing in mind at the same time the symptoms presented by epidemic cholera in 1832, we at once perceive the greatness of the difference between the cholera described by Sydenham and the Asiatic cholera morbus which ravaged Paris in the year just named, which same disease has since reappeared here at several subsequent epochs, and has also desolated many departments of France and many foreign countries. Having already sketched, in a few words, the great differential features of these two diseases, I now proceed to consider the subject more immediately before us—infantile cholera.

It is when infants are being weaned that they are most liable to this disease. Not a day passes in which I do not call your attention to this important fact at the bedside of patients in our nursery ward: I am constantly telling you, that it is whilst infants are being weaned that they are most exposed to serious disorders of the alimentary canal. During the period of lactation, so long as they live upon the natural aliment supplied by the nurse, there is seldom much reason to fear such affections; but should the weaning be badly managed—even if the infants are as old as fourteen, fifteen, or sixteen months—should they be suddenly deprived of the maternal milk, without the observance of certain rules (which I shall point out to you), indigestion will be caused and maintained, which will lead to diarrhœa, and this diarrhœa again, under certain circumstances, will become the starting-point of infantile cholera.

The disease usually manifests itself suddenly, and is announced by symptoms of which I shall now give you a rapid sketch.

The physiognomy of the child rapidly changes. On looking at him, you are at once struck with the very sunken appearance of the eyes, and with a bluish line encircling the lower eyelids. You hear the child uttering incessant cries, often as if being suffocated; for (as in Asiatic cholera) the pitch of the voice is altered, although it be true that the degree in which this alteration takes place is not great. The skin is cold. These phenomena having occurred abruptly, have very naturally alarmed the family, who on the evening before they showed themselves, or perhaps immediately before their appearanse, were but little alarmed at the diarrhœa.

Vomiting previously absent, or existing only in a slight degree, has now become an exceedingly urgent symptom. The little patient vomits all fluids which we try to get him to take. He is, however, tormented by burning thirst, as is indicated by his cries, his impatience, and his alternately opening and shutting the lips, as if for the purpose of sucking in cool air. If a spoon or tumbler be placed near his mouth, he will raise his head, however great his debility, and with voracity clutch it, that he may swallow the liquid presented to him.

The matter vomited is bilious and green. The *stools* are no longer lienteric; but consist of a greenish serosity, in which floats a substance resembling chopped spinage or sorrel, and which is found deposited upon the swaddling-clothes; or sometimes the stools (very liquid) have a slightly yellow, yolk-of-egg tint. They are always absolutely serous, but never have that appearance of rice-water, so characteristic of the stools in Asiatic cholera morbus.

The abdomen is usually sunk in; its skin is soft and flaccid, and when pressed between the fingers, it retains for several minutes the fold which has been made. This want of tonicity in the skin is found everywhere, both in the extremities and trunk of the body.

Sometimes the collapsed condition of the body follows tympanites; but tympanites, an unfavorable symptom, is never so great as in the subsequent stage of the disease. The pulse becomes exceedingly rapid, and the temperature goes on falling; the extremities, nose, chin, and tongue become as cold as in Asiatic cholera morbus, with this difference, however, that there is very little cyanosis, and very seldom viscid sweat, in infantile cholera. On the contrary the skin remains dry, and it is only the nails which acquire a bluish color. The countenance has a leaden hue, but not that peculiar color which is presented in cholera morbus; the features are notably drawn and obliterated.

Too frequently death takes place in the first stage, indeed very soon after the invasion of the disease. If the child resist death for a longer period, other phenomena show themselves. Vomiting then seems to recur more frequently, while it very often happens that the diarrhœa stops at the same time. Then, also, the tympanitic distension of the abdomen becomes great.

A notable elevation in the temperature of the skin succeeds the fall of temperature of which I have just spoken: the skin at the same time regains its tonicity to such a degree that the folds made by pinching it do not remain, as was formerly the case. The tongue is red and dry; the eyes are injected.

Infantile cholera now begins to enter into a new stage—the *typhic stage*, which though analogous to the typhic stage of Asiatic cholera morbus, differs from it in several characteristic particulars.

Sometimes, simultaneously with a cessation of the vomiting, the diarrhœal stools reappear. They have a bilious color, more or less decided, and sometimes they bear a strong resemblance to the evacuations in epidemic dysentery from their glairy, sanguinolent, and at times even purulent appearance. Then, the tympanitic distension of the abdomen diminishes a little, but does not disappear.

During the continuance of these symptoms, the child falls into a stupor. This state of stupor, combined with the injected, upturned eyes, give the child the aspect of a patient affected by cerebral fever: appearances are the more calculated to mislead from the patient occasionally uttering that plaintive cry heard in hydrocephalus, and which may occur in the *tache cérébrale* as well as in encephalo-meningitis.

I have stated that infantile cholera sometimes proves rapidly fatal to children. If the cold stage continue for more than twenty-four or twenty-six hours, death is almost invariably the issue.

When there is a sensible diminution in the evacuations, life is prolonged; and the typhic stage may last for three, six, or even eight days.

However serious the *prognosis* may be in this disease, which every year snatches numerous victims, there is always a hope that the child will recover, if placed in favorable hygienical conditions, and *treatment* be steadily carried out such as I am now going to describe.

The most important prescription is rigidly low diet: as suitable drinks, we ought to order the decoction of barley or rice, and the *eau albumineuse*, which is made by diluting the white of four eggs with a litre of water: this albuminous water is sweetened to taste by adding sugar, and aromatized with orange-flower water.

I look upon the *mustard bath* as the most powerful medication in infantile cholera, when the disease is in its first stage. Into a bath containing twenty-five litres of water, we put fifty grammes of flour of mustard formed into a porridgy paste with cold water, and inclosed in a little linen bag, just as is done in preparing a bran bath. By squeezing the bag, a strongly sinapised water is obtained. Observe that the mustard paste is to be made with cold water: the use of hot water, in place of promoting, prevents the extraction of the essential oil, which is the most active principle of mustard. It would be a similar mistake to use vinegar, with the view of producing a stronger sinapism.

The little patient is immersed in this bath for twelve or fifteen minutes, a time required to obtain reaction, which comes slowly, from the state of the skin. The child is then wrapped up in very dry linen: and the same treatment is repeated two, three, or four times during the day. The proper duration of the immersion can be estimated by the person who supports the child in the bath. The nurse ought to be told, that as soon as she feels her immersed arms smarting with heat, the child must be taken out of the bath.

Gentlemen, in connection with the mustard bath, allow me incidentally to direct your attention to the remarkable effects which you have seen it produce on more than one occasion in women in St. Bernard Ward, for whom I prescribed it in many very diverse circumstances, a subject regarding which I shall probably have to speak to you on some future day.

You, no doubt, have been surprised, as I myself have been, to hear patients complain, that some minutes after immersion in the sinapised water, they have experienced an exceedingly painful sensation of cold. It seemed to them, they have said, as if they were in freezing water, the ice on the top of which had been broken; and (to use their own comparison), they felt as if cut in two by the cold. If we are present when such patients get their bath, we will observe that they shiver with cold, that the cutaneous surface becomes intensely red, and assumes the appearance called goose-skin. So severe is the feeling of cold, that some of our female patients beg to be taken out of the water before the lapse of the prescribed time; and they even continue to shiver for some minutes after they have been replaced in bed, well wrapped up in woollen blankets.

Reaction, however, is not long in being established; and the icy coldness is soon succeeded by a notable elevation of temperature.

Having made this statement, I now resume my remarks on the treatment of infantile cholera. In the first period of the disease, the sinapised bath is, I repeat, one of the most powerful medications—perhaps the most powerful medication—with which I am acquainted. Concurrently with it, however, you require to employ other remedial measures.

You will, in the first instance, have recourse to *ipecacuan*. To a child between one and two years of age, administer from 30 to 40 centigrammes in the twenty-four hours, divided into two or three doses. This, gentlemen, you will find is a new application of the substitutive method, the good effects of which in affections of the digestive organs I have already pointed out.

Next come *diffusible stimulants*. *Ether*, in the form of syrup, is the most convenient preparation to employ. It may be given in dessertspoonfuls every hour or every half-hour; and, in fact, as it contains only a small proportion of ether, the patient may take from 100 to 200 grammes of it, without any inconvenience, during the twenty-four hours.

You, at the same time, prescribe the distilled waters of mint and balm-mint; and, as a tisane, you order the "*decoction blanche*" of Sydenham, or, better still, the "*eau albumineuse.*"*

In the cold stage of infantile cholera, *purgatives*, as well as emetics, are indicated. The purgative which I prefer before all others is the *hydrargyrum cum cretâ*, a remedy of which I have formerly spoken, one greatly esteemed on the other side of the Channel, but which is too little used in France. This medicine (which is mercury killed in chalk) administered in doses of from five to ten centigrammes, generally stops the vomiting, while it also modifies the character, and diminishes the quantity of the stools.

Along with these useful remedies, there is another—*opium*—of which I have already spoken as a medicine which there is a great temptation to use imprudently. This is a point on which I have already stated to you my views: I cannot express myself too strongly against this agent: I repeat, that I am not acquainted with one more disastrous in its effects, nor more frequently and more imprudently employed.

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* The "white decoction" of Sydenham is a remedy much used in France for chronic diarrhœa. It is prepared by adding the following ingredients to 1000 grammes of water:

Calcined hartshorn, 8 grammes;
Crumb of white wheaten bread, 24 grammes;
Gum arabic, 8 grammes;
Simple syrup, 60 grammes; and
Distilled cinnamon-water, 8 grammes.

The albuminous water is described in the preceding page.—TRANSLATOR.

I often see it prescribed in doses so large, that if they were not in great part vomited, the patient would inevitably be poisoned. Recollect the statement I made in a previous lecture: I told you that a single drop of laudanum suffices to throw a year-old infant into a stupor which may last for forty-eight hours; and nevertheless we find that five, six, seven, or eight drops are fearlessly given in potion or lavement. Opium is largely given in the form of syrup; and when there exists timidity in giving that preparation, none is experienced in administering the syrup of white poppy, 30 grammes of which contain 30 centigrammes of the extract of poppy, which, though supposed to be a very harmless dose, frequently acts more energetically than five centigrammes of extract of Smyrna or Constantinople opium. Syrup of lactucarium is also prescribed, the action of which is dependent on the quantity of opium it contains.

But perhaps there is no opiated medication so dangerous as a lavement of decoction of poppy-heads. This preparation, generally looked upon as harmless, and constantly being given without medical advice to very young infants, is one of the most treacherous which can be used, in consequence of the variable amount of the quantities of the active narcotic principles contained in the head of a poppy. Not a year passes in which we have not deaths to register from the improper use of this medicine.

The best method—in my opinion the only proper method—of administering opium, particularly if the patient be a child, is to give Sydenham's laudanum, the doses of which it is easy to graduate. We may begin with a quarter of a drop or half a drop, progressively increasing the quantity, according to the observations we make on the susceptibility of the individual patient. Thus acting, we may proceed in all security, because we know what we are doing.

This is an absolute rule which must never be deviated from. I have already formulated it to you many times: nevertheless, I again insist on it to-day, for its importance is so great that it cannot be too earnestly proclaimed.

In the disease, now specially before us—in infantile cholera—opium in every form ought to be rigorously avoided. Though, in some cases, it put a stop to the vomiting, it too rapidly leads to the typhic stage, which is most to be dreaded when it comes on early in the course of the disease, exactly as in Asiatic cholera.

Mustard baths, emetics, diffusible stimulants, and mercurial purgatives, are the most important remedies in the cold stage of infantile cholera. By their use, we may obtain very successful results; and when we cannot arrest the progress of the disease by employing them, we may at least prolong the patient's life, and so enable him slowly to pass without drawback into the second stage, during which there is an increased chance of recovery.

In the second period, the indications are to continue the albuminous and feculent drinks, and to use mild laxatives, such as the neutral salts, but particularly calomel in very small doses.

When vomiting has ceased, and the diarrhœa is quite established, we administer levigated chalk, trisnitrate of bismuth, and lime-water.

The cold bath is a very useful means of subduing nervous symptoms.

Should the diarrhœa persist, recourse must be had to nitrate of silver: in potion, the dose is one centigramme—in lavement, from 5 to 25 centigrammes, dissolved in from 60 to 100 grammes of distilled water.

You must, however, remember, that the cases in which recovery takes place are few in number, death being the usual termination of infantile cholera, particularly when it attacks children prematurely weaned. We augment the chances of recovery in such cases by providing a good wet-nurse. Do not suppose that although a child has been a long time without taking the breast, it will be impossible to induce him to resume it. No doubt, the longer the period which has elapsed since an infant has discontinued the habit of sucking, the more difficult will it be to get him to begin again to take the breast: but with patience and perseverance the object may be attained, even after three, four, or five weeks have passed without sucking. The younger the weaned infant, the more readily will he be induced to resume sucking, because his actions are more the result of instinct than manifestations of volition.

The most serious consequence resulting from premature weaning is infantile cholera. It is something more than an excessive intestinal catarrh, proceeding from enteritis caused by continuous indigestion, in itself sufficient to determine a vicious alimentation, out of harmony with the digestive aptitudes of the individual. The influence of season, by putting its stamp upon it, makes this enteritis a special enteritis of a character so malignant, as too often to baffle our therapeutic efforts.

In these cases of intestinal catarrh, in which the special element of infantile cholera is absent, the treatment ought to be conducted in accordance with the indications which I described in a general manner when lecturing upon diarrhœa.

Here, the raw meat system of which I spoke to you when discussing chronic diarrhœa, plays an important part.

Some months ago, you saw me prescribe this treatment for a child who occupied (along with his mother) bed 19 of St. Bernard Ward. This child came in with an obstinate diarrhœa, which, in the first instance, resisted all our curative measures. I then resorted to the use of raw meat: from the second day of this treatment, the intensity and copiousness of the diarrhœa decreased.

This treatment is not a novelty. I have employed it for many years: and it has also been adopted by others, particularly by Drs. Blache and Henri Roger, my colleagues at the Hôpital des Enfans. Notwithstanding its efficacy, it has hardly yet taken its proper place in practice: many physicians are hostile to it: at least, when I propose it, I find my proposition treated with ironical incredulity by some of my honorable brethren.

It came to us from the north, thirty years ago. A Russian physician, Dr. Weisse, of St. Petersburg, introduced it to the notice of the medical profession. He was led to make use of it from circumstances which I shall now describe.

For some months, he had been treating a year-old infant, exhausted by colliquative diarrhœa and reduced thereby to the condition of a skeleton. One day, the mother asked Dr. Weisse to allow her to give raw meat to the child; and he consented, recollecting that some physicians allege they have obtained good results from its use in disease accompanied by a hectic state. Next day, he was amazed to see the child chewing a bit of raw meat. Having found portions of undigested meat in the stools, he ordered that in future the little patient should not have more than three spoonfuls a day of very finely minced meat. Digestion was performed easily; and in some weeks the patient, formerly supposed to be hopelessly lost, was completely restored to health.

Dr. Weisse's remarks having come to my knowledge, and similar facts having also been reported to me by foreign physicians, I, in my turn, made experiments; and have ever since had occasion to speak favorably of this mode of treatment.

My observations, which were at first confined to children, were afterwards extended to adults; and when I was speaking to you of chronic diarrhœa, I cited the case of a young married lady who was cured by the raw meat system. But there is no condition in which this singular method of treatment is more useful than in the diarrhœa which supervenes at the period of weaning. Whether weaning take place prematurely, or after complete dentition, the digestive canal is unaccustomed to the new description of food.

Under what form ought the raw meat to be given?

Take a piece of lean beef, mutton, or fowl—beef or mutton, however, being preferable—cut it up into very small morsels, so as to constitute a sort of hachis, and then put it into a mortar and with the pestle work it into a thick mass. The pulp so made is forthwith passed through a cullender, so fine as to permit nothing to pass except the juice of the meat, the fibrin of the blood, leaving behind only bloodvessels and cellular tissue. By this means, a real *purée de viande* is obtained, which is collected by scraping the external surface of the cullender.

The preparation of the meat, as you see, demands a certain amount of patience. When so complete a result cannot be obtained, we may substitute for the *purée de viande*, meat chopped as small as possible; for this can be digested easily, though with less facility than the *purée*.

The proposal to use this singular remedy is generally received disapprovingly by mothers, who estimate the probable repugnance of their children by their own actual disgust. You also run the risk of opposition from servants, who greatly dislike to add to their usual routine of duty, a task so troublesome as the preparation of the *purée de viande*.

As for the children, it often, nay it generally, happens that they show none of that repugnance to this kind of food which they have been expected to manifest. At once, they take it without the least grumbling. From the very first day on which it was given, you saw our little patient of bed 19 devour daily his 125 grammes of raw meat. In this, there is nothing which need surprise you, when you see children take, not only without disgust, but even with satisfaction fish oil, which very few of you would like to taste.

There are some, however, who have a profound aversion to raw meat. For them—allow me the phrase—we must gild the pill. There is nothing more easy than to do this. We make the pulpified or minced meat into little balls with salt, sugar, fruit-jelly, or conserve of roses, the mixture and selection of ingredients being regulated entirely by the taste of the patient. Raw meat, when well prepared and disguised in this manner, is easily taken: its taste is masked in such a manner as not to be at all disagreeable. Should the meat not agree with children when mixed up with salt, powdered sugar, jelly, or conserve of roses, it may be put into a clear gravy soup, as if it were tapioca or sago. It may likewise be mixed with chocolate, made with water; and although such a combination is in direct opposition to the ordinary rules of the culinary art, it is one which patients find much to their liking.

By trying these different combinations, which admit of being infinitely varied, we discover the one which is most readily accepted. Children soon become accustomed to the use of raw meat, and ultimately take it, not only with pleasure, but even with so much voracity, that on their little allowance being consumed, they will demand another supply, just as if it were the most dainty dish.

In adopting the raw meat regimen, it is necessary to proceed with a certain amount of caution. Begin by administering small quantities; for if you all at once give the patient large quantities, there is a risk of aggravating in place of curing his indigestion; and on the other hand, by commencing too abruptly, you may create invincible disgust for your aliment.

Nothing is more simple than to measure and weigh the quantities to be given in the twenty-four hours. This requires no special apparatus: our current French coins will serve very well as ordinary weights, and will be more easily remembered. As you know, a silver twenty-centime piece represents exactly one gramme—and a silver five-franc piece weighs twenty-five grammes.

We may begin by prescribing raw meat equal in weight to a five-franc piece, to be taken daily in three doses. If these 25 grammes are well digested, the daily allowance may be at once doubled, and progressively increased from day to day, according to the manner in which the child bears preceding doses, till a daily administration of from 100 to 150 grammes is attained. Having reached this point, the child must be kept at it for some time. If the appetite be keen, and health be evidently returning, the daily quantity of raw meat may be increased at the rate of 25 grammes a day till a daily allowance of 200 grammes or even of 500 grammes has been reached!

During the time that the raw meat regimen is being pursued, it is indispensable to forbid the use of every other kind of aliment, and all drinks except nutritive drinks. The nutritive drink which is specially appropriate is the *eau albumineuse;* it is useful from the modifying influence which it has upon the diarrhœa, and from its being so agreeable that children take it willingly.

For some days after the commencement of this regimen, it is very usual to find the raw meat almost unchanged in the stools of the children; the fecal matter, at the same time, contains a large quantity of decolorized fibrin. That ought not to surprise you; nor ought it to discourage you from persisting in the treatment. Assuredly 75 or 100 grammes of raw meat in passing over the intestinal surface must leave some nutritious matter to be absorbed. In point of fact, it is soon seen that the little patient has regained strength. After the lapse of a certain time, sometimes after four, six, or eight days, the excrementitious matter begins to be moulded in the gut: its smell is exceedingly fetid, recalling the odor of the excrement of purely carnivorous animals. This little inconvenience is of slight consequence, and need not be a cause of anxiety. All that is necessary is to know that such an effect is produced, and to give the relations notice of what may be expected.

It is difficult to fix a period for the duration of this treatment. Sometimes, it does not admit of being suspended, from the children becoming so habituated to the raw meat that they will take no other kind of aliment: and sometimes also, a change of regimen, brings on unfavorable symptoms.

One of my grandsons was an example of this. When sixteen months old, he was attacked by diarrhœa, which resisted bismuth, chalk, nitrate of silver, rhatany, monesia, opium, and in fact every remedy, till I gave him raw meat. From that time, his malady yielded, and health was re-established: I continued the regimen, however, for more than a year. The child at last was taking daily 500 grammes (rather more than an English pound) of pulpified raw meat. Whenever I suspended its use, the diarrhœa returned; and to maintain recovery, it was necessary to continue the treatment.

Gentlemen, I certainly do not announce this medication as an infallible means of cure: I only say that in a great number of cases it has proved remarkably successful in my hands, and in those also of others; and that I have obtained cures through its instrumentality when all hope of recovery seemed to have been lost.

Twin sisters, aged 17 months, daughters of one of the largest manufacturers at Mulhouse, were brought to Paris at the time of the Universal Industrial Exhibition in 1855. They were in a frightful state of emaciation. They each weighed between fifteen and sixteen pounds (French), their skin was covered with petechial patches, some of which were equal in diameter to a five-franc piece. They vomited all food that was administered to them, and even vomited the sugared water which they took. For three months, from the date of their weaning, they had been exhausted by serous diarrhœa. Considering the formidable character of their symptoms, and the radical deterioration of their constitutions, I could only entertain very slight hopes of improvement. We were, at the period to which I refer, in the full tide of summer. The family lived in the Champs Elyseés, where the poor babies were daily dragged about under an ardent sun in a little carriage. Upon one occasion, their nurse returned from the promenade in a state of great excitement, declaring that she did not wish to go out with them again, as she had been taunted by passers-by, who had expressed themselves as indignant at seeing children taken out who appeared more dead than alive. I narrate this little incident to show you the terrible state to which these wretched little creatures were reduced. Though I had not much hope of being useful, I was anxious to try something. I recommended raw meat. The result exceeded my hopes. Digestion was re-established: and when the little girls left Paris, they could not have been recognized as the children who had arrived. They had regained health, and a surprising amount of plumpness. The raw meat regimen was continued for a year. Some time later, their father, in testimony of his gratitude, sent me their photographs.

It is a curious fact, that one of the little girls was affected with tænia solium during the course of her malady. The extract of male fern soon brought away from ten to twelve metres of tapeworm. Six months later, and during the time she was eating the raw meat, the same child had a second tænia solium, which she got rid of by the use of the same means which were employed on the former occasion. Are we to attribute the presence of these worms to the regimen which the child had been following? These entozoa, as you are aware, are frequently met with among the inhabitants of Abyssinia, who habitually make use of raw meat. We may ask, whether we are to attribute the worms in this child to her having passed a portion of her summers at Bâle (in Switzerland), where tænia may be said to be endemic?

I am inclined to believe that the regimen was really the cause of the tænia in this child, because several physicians, including Weisse, Braun, and Von Siebold,* have often observed worms, particularly the tænia solium, in persons whose exclusive aliment was raw meat. Be that as it may, the verminous affection was no obstacle to the cure of the diarrhœa, which was the cause of my having been called in.

To assist the beneficial action of, and facilitate the tolerance for, the raw meat, it is necessary to employ agents of the materia medica in conjunction with it. Opium administered in minute doses, in accordance with the rules which I have given you, is a valuable resource. When it fails, I give chalk, and subnitrate of bismuth, at mealtimes, and in the interval between meals, in doses of from one to four grammes. Along with these medicines,

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* BRAUN and SIEBOLD: Journal für Kinderkrankheiten for January and February, 1858. Erlangen, 1858.

I sometimes give the sesquinitrate of iron, a preparation specially commended by Graves: I prescribe it to be taken in doses of two or three drops during meals.

Finally, when the circumstances call for it, I also recommend tonic treatment. In this class of cases, I find the tincture of nux vomica a very useful tonic. Of this I order to be taken, in the course of the twenty-four hours, only one drop, which is prescribed in a liquid mixture, so that it may be taken in three separate doses. I have also had good results from the use of hydrochloric acid.

LECTURE LXXIII.

LACTATION, FIRST DENTITION, AND WEANING OF INFANTS.

LACTATION: *Natural, Artificial and Mixed.—Lactation in respect of the Woman.—Conditions essential to a Good Nurse.—Influence on the Lacteal Secretion of Menstruation, Conjugal Relations, Pregnancy, and Intercurrent Diseases.—Lactation in relation to the Nursling.—Weighing the Infant is the only means of ascertaining whether it is sufficiently suckled.*—FIRST DENTITION: *Mode of Evolution of the Teeth in Groups.—Order of Succession in which they appear.—Casualties of Dentition.—Febrile Discqmfort.—Convulsions.—Diarrhœa.*—WEANING.

GENTLEMEN: The subject of my last lecture was infantile cholera, which I told you was the most serious affection which resulted from premature weaning. I reserved for to-day's meeting some considerations regarding lactation, the first dentition, and weaning.

Every one knows the meaning of the term *lactation.* It signifies the alimentation of the infant, by its sucking from the breasts of its mother or another woman. In the definition of *natural lactation,* feeding with the milk of animals has been sometimes included; but this more properly is considered as comprised in *artificial lactation,* which for the most part consists in giving the milk of the cow, or the goat, by means of a feeding-bottle or spoon. *Mixed lactation,* the system usually adopted, is a combination of the two others. Natural lactation, as I shall tell you forthwith, is undoubtedly the method which ought to be preferred: but from this general rule, there are exceptions.

Before considering how lactation ought to be conducted, the question arises, What are the conditions required in a good nurse?

First of all, a woman, to be a good nurse, must be in the enjoyment of perfect health: but from this proposition—to which self-evident truth gives a character of commomplace—it is not to be inferred that every perfectly healthy mother is fit to nurse her infant. Some puny-looking women are very good nurses. There are also vigorous robust women whose breasts secrete an insufficient quantity and a bad quality of milk; and this inaptitude to nurse occurs without our being able to assign a reason, or when consulted to predict its occurrence.

However, with the reservation now stated, a healthy woman will in general make a good nurse. We judge of the state of health by the woman's appearance, by examining the state of the different organs, and by such additional information as we can obtain. No very great importance can be attached to the complexion and color of the hair: fair and dark women make equally good nurses. Beautiful teeth, so important in the opinion of some persons, have really no other advantage than increasing good looks.

The manner in which the menstrual function is performed may, to a certain extent, be regarded as an indication of the manner in which milk will be secreted. If menstruation be irregular and scanty, there is a fear that the lacteal secretion will be badly accomplished: and nursing fitness is also improbable when the menses are habitually too abundant, for the chances then are, that after giving suck for two or three months, the menstrual flow will recommence, the mammary fluxion, hitherto energetic, being counterbalanced or annihilated by the uterine. The presumption is in favor of a woman making a good nurse, if she be regular in her menstrual function as regards time and quantity of flow. I use the word *presumption*, because we cannot, I repeat, give in advance an absolute opinion.

The state of the breasts, even, does not give us sufficient evidence by which to decide the question, although it furnishes information of positive value.

We must judge of the breasts less by their size, than by their form, the appearance of the skin covering them, and the shape and development of the nipple.

The largest and roundest breasts are not always those which yield most milk; for it often happens that mammary development is due to a predominance of cellular tissue and fat, while smaller pearshaped breats indicate development of the gland itself, promising therefore a more abundant secretion of milk, particularly if their skin is marbled by veins, testifying richness of circulation.

As to the nipple: it ought to enter easily into a state of erection, and be of size sufficient to enable the infant's mouth to get a good hold of it in sucking.

When there is to be an abundant secretion of milk, it begins to be secreted long before parturition. The breasts, which from the very commencement of pregnancy had been somewhat swollen and tender to the touch, and at the third month were surrounded by the characteristic areola, show, towards the end of the fourth month, an oozing of colostrum, which is sometimes so considerable as to stain the woman's linen. Immediately after delivery, this flow of colostrum becomes more copious; but four or five days elapse before the secretion has all the characteristics of milk.

It is usual to press the breasts, with a view to determine whether the milk is or is not abundant; but this manipulation requires certain precautions, and the taking into account of many details. It is essential to practice this sort of milking with the greatest possible gentleness, otherwise unpleasant mental emotions may be caused in the woman when under examination, which will prevent the secretion of milk, just as it is stopped in the cow and other animals which furnish a less supply of milk when the operation of milking is roughly conducted. For the same reason, it is necessary, in addition to being careful to make gentle pressure, to avoid producing a sudden feeling of change of temperature in the skin, by using a cold hand.

When these precautions are properly taken, the milk spurts out vigorously through several orifices in the nipple, unless indeed these apertures are partially plugged by milk which has remained in them since the woman last gave suck: under such circumstances, the nipple ought to be washed before practicing the manipulation.

It often happens that when the secretion is very abundant, the milk spurts forth spontaneously, not only from one breast while the infant is sucking the other, but even from both breasts when the infant has not been sucking for some time. This spontaneous secretion—this rush of milk [*montée du lait*] as it is called—is announced by a peculiar sensation, a sort of itching, which, though generally agreeable, is sometimes painful.

The quality of the milk generally corresponds with its quantity. It would be wasting your time, to enter here into all the details of this subject. I shall confine myself to recalling to your recollection facts which I have had occasion to bring under your notice when speaking upon other questions. The milk of a woman may be perfectly good for a particular child, and exceedingly bad for another: certain circumstances may modify the good qualities of the milk; and I must also remind you, that there are a few exceptional infants whose digestive organs cannot tolerate any kind of milk, whether it be woman's milk, or the milk of a cow, goat or ass.

Although, as a general rule, women do not menstruate when giving suck, there are some in whom the menses reappear during lactation. If the menstrual discharge is very abundant the secretion of milk not unfrequently ceases; if the menstrual flow be moderate, the lacteal secretion and lactation suffer whilst menstruation is going on. Besides being injured by having less to take, the infant sometimes suffers from intestinal symptoms, such as diarrhœa and colic.

A nursing mother grows fat during the first months of lactation; but towards the end of the first year, this plumpness is lost, showing that the lacteal secretion is too great a pull on the health. In these cases, the infant requires supplementary diet.

I have still a few concluding remarks to make on lactation in its relations to the woman. *Conjugal intercourse* is not injurious to nurse or nursling, provided it be regulated by great moderation. *Pregnancy* supervening during lactation produces no other bad consequences than a diminution, or complete drying up of the lacteal secretion; the milk, however, does not acquire any bad properties from the existence of pregnancy. If the infant begin to ail, it is because its food is no longer sufficient in quantity. Supplementary alimentation then becomes necessary; and this may lead to the bad consequences which I have brought under your notice, as liable to occur when the nursling is not old enough or otherwise not in a suitable condition to be weaned.

It has been alleged that lactation favors recovery from intercurrent diseases, the idea being that convalescence is shortened by the mammary fluxion establishing a sort of derivative action of a very energetic kind. Without giving quite so affirmative an opinion on the subject, I may say, that, according to my own experience, lactation does not complicate acute diseases, and that it even seems as if a woman by discontinuing to give suck during the course of a malady, probably of short duration, does that which was dangerous both to herself and infant. Should, however, the disease continue for a month or six weeks, nursing must be suspended, as it then proves a cause of exhaustion additional to that arising from the intercurrent acute affection. The discontinuance of nursing may be not a matter of choice but of necessity; for the long continuance of a febrile state may diminish or quite dry up the supply of milk.

You know, moreover, that the lacteal secretion, particularly in a good nurse, recommences very easily, becoming as abundant as before, even after having ceased for a fortnight, three weeks, or a month. I have even seen it begin again after having been suspended for three months.

I have already told you how necessary it is that the nipple should be so developed as to give the infant's mouth a good hold in sucking; this suitability of development is also an important matter for the mother, for when the nipple is short and difficult to lay hold of, it is irritated by the infant's difficult attempts at sucking, and so rendered more liable to erosions and fissures, which latter are sometimes very deep and painful. The fissures, too, may become the starting-point of inflammation, which, reaching the mucous membrane of the mammary canals, will cause them to become obliterated, and so lead to the breast being affected with engorgements of milk, constituting "the hair" [*le poil*], an affection which terminates in the formation of numerous abscesses.*

When erosions and fissures begin to form, it is necessary, when they are limited to one breast, to cause the infant to suck, as much as possible, only from the other breast. The fissures ought to be washed with Goulard's water, a decoction of rhatany, or a decoction of oak bark: they may be dressed with pomades of tannin, rhatany, white precipitate: or, red precipitate: or, better still, they may be touched with the solid lunar caustic. If there is a tendency to the formation of new fissures, the woman must adapt an artificial teat to her nipple. It ought to be small, and so constructed as to embrace the nipple. Before it is used, it ought to be washed in warm water and sugared milk; and the nursing woman ought to press her breast in such a way as to facilitate the infant's exertion of sucking.

So much then for lactation in its relations to the woman: let us now see how it ought to be conducted for the benefit of her nursling.

The infant has just been born! As soon as the mother is able to sit up —by that I mean, two or three hours after delivery—the infant ought to be put to her breast, even although the milk may not yet have come into it. This practice is, on the one hand, useful, as the suction fashions the nipple: on the other hand, it frees the ducts from concreted colostrum: thirdly, from the first, it gives the nursling good habits. I am opposed to the custom of giving with a spoon sugared water to infants during the first twenty-four hours after birth, because it is teaching them to drink without sucking: this is bad; for sucking is toilsome work, which an infant will only be too glad to avoid if shown how otherwise to obtain its needed nourishment.

Let, therefore, the infant be put immediately, or at least as soon as possible, to the mother's breast. After the second day, it will there find sufficient nutriment. At first, it ought always to be put to suck on awaking from sleep. But after ten or twelve days, when sucking abundantly at each time of nursing, the feeding ought to be by rule, for the sake both of mother and child.

By sucking abundantly, I mean the infant taking each time that he is put to the breast, 60 or 80 grammes [about 1½ to 2 fluid ounces] of milk. If a strong vigorous infant takes less than this quantity, the nurse is bad. Here, gentlemen, is the one and only test by which you can determine whether a woman is or is not a good nurse: it was devised by my honorable colleague Professor Natalis Guillot.†

Before applying the infant to the nurse's breast, it is weighed in its swaddling-clothes: when it has sucked, it is again placed in the scales without any change of clothes being allowed. The excess of weight at the last over the first weighing gives the exact weight of milk which has been swallowed. The infant, I repeat, ought to take at least from 60 to 80 grammes, during the first period of lactation: when four or five months old he ought to take 250 grammes at one nursing, and about 1500 grammes in the twenty-four hours.

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* "*Le poil*" is the vulgar name in France, for *mastitis*; and is really a translation of the Latin, "*morbus pilaris.*" This absurd term was originally applied by the ancients to mammary engorgements, from the strange notion of Aristotle, that they were caused by accidentally swallowed hairs passing from the stomach into the mamma!—TRANSLATOR.

† NATALIS GUILLOT: De la Nourrice et du Nourrisson. Union Médicale, 1852 pp. 61–65.

Some children, whose nurses have a very copious supply of milk, always regurgitate some of it immediately after sucking. With a view to prevent this occurrence—not, however, one of much consequence—the woman ought to place her finger upon the orifices of her nipple which will prevent a too rapid flow of milk. The infant ought to be so trained as to suck five or six times between six in the morning and nine in the evening. It is very important that it should, if possible, not be nursed during the night, so that the mother may have eight or nine hours of undisturbed sleep, which is necessary for her retaining health, and properly nursing her infant. If she do not get this amount of rest, her strength will become exhausted, and her nursling will suffer. If the infant is a bad sleeper, it must be sent away from its mother to another room, and suckled with the feeding-bottle during the night.

Under these rules, the infant attains the age of four, five, or six months. He may then be allowed to take thin farinaceous soups made of arrowroot, tapioca, cassava, vermicelli, rice-flour, breadcrumb (well boiled and passed through a cullender), or better still, hasty-pudding made of wheaten flour: this latter is the best and cheapest farinaceous food. These farinaceous aliments ought to be prepared with milk diluted with sugared water, or with butter. In certain proportions, meat soups may also be given; but they must not constitute the principal part of the supplementary diet.

Natural lactation, as I have stated, is unquestionably the best, and on principle it is prescribed.

Artificial lactation, or in other words, feeding infants by means of the feeding-bottle or spoon with the milk of animals, is, generally speaking, a deplorable system.

In Paris, in particular, but in all large towns, it is the chief cause of infantile mortality: one dies out of every four subjected to this plan of rearing: the three who resist death are generally damaged in health and constitution. As we shall afterwards see, rickets is a very common consequence of this kind of feeding.

When circumstances, irrespective of wishes, compel families to have recourse to artificial lactation, the bad consequences to which it naturally leads may be moderated by attention to certain rules. First of all, the infant must be made to suck from the feeding-bottle, and not allowed to drink from the cup. Drinking from the cup is very objectionable: the milk traverses the mouth too rapidly to be duly mingled with the saliva, the alkaline nature of which prevents the milk being too quickly coagulated on its reaching the stomach.

Cow's milk is the best: the infant ought if possible to have *an average milk*—that is to say, a mixture of the milk of a great number of cows living in the same byre. This milk ought to be mixed with very thin panada, decoction of barley, or decoction of grits, the proportions of the fluids being one-third of water to two-thirds of milk. This mixture when given to the infant is moderately sweetened, and heated to the temperature of the body.

When this aliment does not agree with the infant, a small quantity of bicarbonate of soda ought to be added: not more than from 30 to 50 centigrammes of it ought to be given in the twenty-four hours, the quantity administered at each feeding being, therefore, about five centigrammes. Should this admixture with bicarbonate of soda not prevent the milk being rejected, one drop of laudanum—but not more than one drop—may be added to the entire quantity of food taken during the day.

Notwithstanding the adoption of all these precautions, some infants will not thrive on artificial feeding. To them, a wet-nurse is a necessity. The lapse of a long period since weaning is no reason why such infants should not be made to resume the habit of sucking. To get them again to take the breast, enticement must be mingled with compulsion: the nurse's nipple must be moistened with sugared milk, and the infant must be deprived of every kind of food and drink, so that hunger may force it to recommence sucking the breast.

I have now come to the great question: At what age ought infants to be weaned?

Gentlemen, you every day hear parents announce with the utmost precision the date at which their infants ought to be weaned—fixing it, as the case may be, for the age of nine months, a year, or fifteen months. The proper time for weaning, cannot, however, be thus determined by consulting the almanac. It is not at nine months, a year, or fifteen months, and far less is it at an earlier age, that we are to place the limit of lactation. Remember this truth, and with it indoctrinate the families who ask you to direct the health of their children. Your true guide in this matter is the more or less advanced state of dentition. The infant ought to be suckled till the time is past during which the formidable complications of teething supervene.

The *first dentition* comprises the evolution of the twenty temporary teeth, usually called the milk-teeth, and which about the age of seven begin to be replaced by the permanent teeth.

They make their first appearance in groups, at times, and in an order remarkably determinate.

The *first group* includes the *two lower middle incisors.*

The *second group* includes the *upper incisors*—the middle coming first, and then the lateral. The infant then has six teeth, four in the upper and two in the lower jaw. In passing, let me observe, that, strange to tell, this fact, though known to every woman who has had children, has been ignored by men of science, even by authors who have specially written on this very subject.

The *third group* includes the *two lower lateral incisors* and the *four first molars.*

The *fourth group* is formed by the *four canine* teeth.

The *fifth group* consists of the *four last molars.*

This is the usual order of appearance; but from it the exceptions are numerous. Though nine times in ten, the lower middle incisors are the first to appear, they are sometimes preceded by the upper middle incisors; but in these rare exceptional cases, the lower middle incisors immediately follow the evolution of the others. The simultaneous evolution of the upper and lower middle incisors is a still more rare occurrence. Likewise, in very exceptional cases, the small molars show themselves before the appearance of the second group—that is to say, the upper incisors. With reference to the evolution of the third group, it may be stated that, pretty frequently, the two small molars are seen before the lower lateral incisors. There is seldom any irregularity in the evolution of the fourth and fifth groups.

Notwithstanding the anomalies now stated, there is much more regularity in *the order* than *the epoch* of the appearance of the teeth.

The common opinion is that girls are more precocious than boys. This remark, which is perhaps true in respect of intelligence, would seem to be applicable also to the appearance of the first tooth. I find, as the result of my statistical inquiries into this subject, that in girls, the extreme limits at which the first tooth appeared were the second and the fourteenth months, the sixth month representing the average date; whereas, in boys, the extreme limits were the third and fourteenth months, the seventh month representing the average.

Before proceeding farther, gentlemen, let me remark upon the absurdity and inapplicability of *averages* when applied to matters of this kind. Among the boys who furnished me with the statistical table to which I have been referring, seven months was the average date at which the first tooth appeared, but not even in one of the cases from which this average was deduced did the first tooth appear at the seventh month, so that the average of the facts is not applicable to a single individual fact of those furnishing the average. In respect of the girls, the average of six months applies to three of the facts only, that is, to one-fourth of them.

Although this application of statistical results is profoundly absurd, we must not on that account reject statistical inquiries altogether, as some would wish us to do. A statistical result has this advantage, that it expresses no more than it ought to express; that is to say, a mass of individual facts which group themselves in numbers more or less considerable, and from which we may draw our conclusions, but which conclusions never can be *general*, inasmuch as they do not admit of application to all the individual cases; and, logically, no conclusion which is not applicable to each individual instance can be a *general* conclusion.

Here, gentlemen, let me once more raise my voice against the system of averages which has been so extraordinarily abused, and which, though overthrown by every rule of the most common logic, attempts to give as truths an average which are only abstractions, not expressing the fact which is most common, but the fact intermediate between extremes, and which itself may seldom or perhaps never exist. I protest against the mad attempt which has been made to base therapeutics upon averages, and to ask from statistics formulated truths which statistics cannot furnish.

Let me now return to the dentition of children. From a collection of cases which I have attentively observed, it would appear that girls are earlier than boys in cutting the first tooth.

Experience has shown, that variations are so great as to render it impossible to fix the exact limits of age at which this event may occur. Some infants are even born with teeth: of this phenomenon many examples have been recorded. When teeth exist at birth, they are generally the middle incisors: then we have the other extreme, some children not cutting the first tooth till eighteen months or even an older age than that has been reached. Between the two extremes, all the intermediate ages are met with—two, three, four, five, six, seven, nine, ten, fourteen months. I take these figures from the statistical tables of which I have been speaking.* It is possible, however, by making an abstract of all the observations made on this subject, to fix a period between six and nine months; and to be still more precise, let me name six months and a half as the period at which the first milk tooth generally makes its appearance.

My former pupil, Dr. Duclos,† one of our most distinguished physicians, now in practice at Tours, has come to the same conclusions; and he has also shown, that the first group of teeth, the lower middle incisors, appear between the sixth and ninth months.

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* Trousseau: Journal des Connaissances Médico-Chirurgicales, for November, 1841.

† Duclos (de Tours): Bulletin Général de Thérapeutique for April and May.

There is no great difficulty in determining the exact epoch at which the first tooth has appeared; for mothers, from whom alone on this point we can derive our information, are seldom mistaken. The cutting of the first tooth is an occasion of great maternal rejoicing, and is watched for with peculiar solicitude. But in respect of statements as to the date at which the second and still more the third tooth has appeared, I have much less confidence in the mother's memory.

The cutting of each group of teeth occupies, however, a certain period which, although generally of limited duration, is in exceptional cases more extended.

The evolution of the lower middle incisors generally takes place within a period of from one to ten days.

The four upper incisors are usually cut within a period of from a month to six weeks.

The lower lateral incisors, and the four molars, are cut within a period of from one to two months.

The evolution of the canine teeth occupies from two to three months.

The last molars occupy an equally long period in their evolution.

The evolution of the canine teeth is attended with the most difficulty, which probably arises from their having the longest roots.

The fact to be remembered as that of the greatest practical value in relation to weaning is that between the evolution of each group of teeth, that is to say, between the complete evolution of the last tooth of one group and the evolution of the first of the succeeding group, there is a period during which the progress of dentition remains in entire abeyance.

The duration of the pause between the completion of the first group and the appearance of the first tooth of the second group, is from two to three months.

The pause between the completion of the evolution of the superior incisors and the appearance of the first lower lateral incisor, or first molar, is two months.

From four to five months elapse between the complete evolution of the last molar and the appearance of the first canine.

There will then be an interval of from three to five months before the appearance of the first molar of the last group.

The periods are not, of course, always so precise as I have now stated, and as M. Duclos has described them; but for us, the important point to bear in mind is that, save in some few exceptional cases, there is a well-marked interval between the evolution of the different groups; the interval between the complete evolution of the last molar of the third group and the appearance of the first canine tooth is generally very prolonged; as is also that which separates the appearance of the last canine from the appearance of the first tooth of the last group.

When the entire evolution of a particular group takes place rapidly, the interval between the conclusion of that evolution and the appearance of the first tooth of the following group is more prolonged; and on the other hand, when the evolution of the group is exceedingly slow, there will be very little interval between its completion and the appearance of the first tooth of the next series.

It is not unusual for the first twelve teeth to be cut almost simultaneously without any very distinct pause between the different series.

These anomalous occurrences have for the most part no appreciable cause.

Irregularity in the order of dentition does not admit in general of any explanation; nor does it seem to have any significance whatever in respect of the general health of the infant. At the same time, there are certain diseases which almost invariably lead to irregularities both in the order and the time of appearance of the teeth.

There is no disease which exerts so decided an influence of this description as rickets. It rarely shows itself before the beginning of dentition; but when it does then appear, it retards dentition almost indefinitely. Should it supervene during dentition, about the age, for example, of ten or twelve months, it abruptly interrupts the evolution of the teeth, which then appear at distant intervals. Finally, should rickets supervene at an advanced period of dentition, or even when only a few teeth have appeared, it causes caries of the teeth, all, but particularly the incisors, being apt to become loose and fall out.

It is very important to remark that tuberculization, which has been for so long a time and so erroneously confounded with rickets, has a precisely opposite effect upon dentition. It is not unusual to see in children whose glands and pulmonary parenchyma are infiltrated with tuberculous matter, a regular and even a rapid development of the teeth, which, moreover, will probably remain undeteriorated during the whole duration of the malady.

Gentlemen, the facts upon which I have now expatiated would possess only a moderate degree of interest were it not that we can deduce from them practical conclusions relative to the subject now before us, and which may be expressed in the *absolute*, I say the *absolute* rule, never to wean an infant, unless some special circumstance render it imperative, till after the period during which the serious complications of teething usually occur.

There is a popular proverb which says—"*bel enfant jusqu'aux dents*"—signifying that the health of a child is most apt to undergo an unfavorable change at the period of dentition.

The possible dangers of dentition are greatest the nearer the period of the evolution of the fourth group; and the popular belief is well founded, that the time of cutting the canine teeth is a time of anxiety. This probably arises from the canine teeth having very long roots; and probably is sometimes also caused by the jaws not being sufficiently developed, for occasionally the sockets are so narrow that it is difficult to understand how the teeth can become developed within them. Then again, they are the only milk teeth which come forth within inclosed spaces—the spaces destined for them are between two teeth already developed, whereas all the other teeth are free, at least on one side, whilst they are piercing the gum.

The cutting of the last molars is most exempt from danger, a circumstance explained by the fact that at the period of their evolution the jaws have attained sufficient development; at this period also the infant has acquired strength sufficient to contend against complications which it could not have resisted at an earlier age.

Let me add, as a concluding general remark, that the manner in which a child cuts its first groups of teeth, affords no criterion by which to judge as to the way in which the evolution of the others will take place.

Having made these preliminary remarks, I now proceed to review the morbid conditions to which dentition may give rise.

The most common complication of teething is high fever, particularly at night, characterized by restlessness, insomnia, and cross temper. Along with these symptoms, the flesh becomes soft, there is a loss of natural color, and a dark areola appears round the eyes. This state of discomfort, which is a manifest effect of inflammatory action, precedes and accompanies the evolution of each tooth. It lasts from one to eight days; and it generally terminates on the very day on which the tooth shows itself, though it sometimes continues for one day or two days more. The symptoms really are those of a slight traumatic fever. They constitute the most common, and the least formidable complication of dentition. Nevertheless, when the teeth are evolved in very rapid succession, the symptoms now described are quite sufficient deeply to affect the health, and leave on the child's face the mark of the malady.

Sometimes, the disturbed state of the system declares itself by convulsions, which may depend partly on the pain caused by the process going on in the gums, and still more on the fever accompanying it. When speaking of the eclampsia of children, I gave you my explanation of these nervous attacks.

I only name stomatitis to remind you of it; it is very often of sufficient intensity to account for the fever, and is often accompanied by an ulcerative eruption causing intolerable pain as well as sometimes salivation and thrush [*muguet*].

I should say nothing about swelling and engorgement of the gums, which seldom occurs at the time of the evolution of the first teeth, but is more common at the cutting of the canine and molars, were it not that I wish to warn you against a practice which I consider as very objectionable. Mistaking the effect for the cause, many physicians attribute the difficult evolution of the teeth to this swelling, and under the influence of that idea, they scarify or make a crucial incision in the gums for the purpose of facilitating the exit. For that purpose, the operation is, to say the least, useless; and I very much doubt whether it even relieves pain by disengorging the turgid gums.

There is nothing in respect of the affections of the mouth which need detain us. The affections of the skin, so common during dentition demand more attention.

I do not refer to those transient fugitive erythematous eruptions, those red eruptions unaccompanied by pain, and irregularly circumscribed, which after appearing on different parts of the body (but particularly on the face), go away spontaneously, as soon as the influence of the process of dental evolution which excited them has moderated; nor do I refer to nettle-rash, which may also show itself: I speak at present only of cutaneous affections such as eczema and herpetic and impetiginous eruptions. These eruptions sometimes occupy a very considerable surface of the body—particularly the impetiginous eruptions which cover the face and hairy scalp, invade the trunk and limbs—causing dismay to families and often to physicians. This is not because they threaten life, but because they occasion great distress to the little patients, and obstinately resist all treatment.

The general inflammatory state of the system [*le mouvement fluxionnaire général*] may show itself by cutaneous affections or by catarrhal affections—by attacks of bronchitis, which ought to put us on our guard: but that for which we ought to be specially on the alert is disturbance of the intestines. Some infants are attacked with diarrhœa each time they cut a tooth, so that when in such subjects dentition proceeds too rapidly, or in a confused manner, exhaustion of strength is the result.

If the diarrhœa do not continue for more than four or five days, if it be not profuse, if the infant be under no bad influence through an unfavorable medical constitution of the season, the catarrhal phlegmasia, of which the diarrhœa is the expression, ought not to make us uneasy: it will cease spontaneously, leaving no trace behind. But if the diarrhœa be prolonged, the mucous membrane of the large intestine will become inflamed, and ulcerate superficially: the phlegmasia, acute when each new tooth was coming through, will at last become chronic, and may lead the infants to marasmus or the grave.

The sympathy which exists between the different parts of the digestive apparatus explains why the disturbance experienced by the stomach and intestines is responded to by their annexed organs. The formidable character of this disturbance is increased by the accompanying fever modifying the character of the gastro-intestinal secretions.

It is a prevalent belief of the public, and is likewise an opinion of physicians, founded upon a misconception of a proposition of Sydenham, that diarrhœa exercises a beneficial influence during dentition. This is an error respecting which I beseech you to be on your guard, and against which I implore you to exert all your influence. In sucking infants, diarrhœa is a symptom which must be looked upon in a very serious light. Though it cannot be denied that a very moderate amount of diarrhœa seems to diminish the general state of fever and the inflammation of the gums; yet it is equally true, that if the purging last for more than four or five days, or become too urgent, it must be treated with the greatest possible activity.

I anticipate, however, that objections to my views will be urged by some enlightened practitioners, who, in direct opposition to what I have now been telling you, will maintain that the suppression of this diarrhœa is a very frequent cause of serious mischief. It is necessary to establish a distinction. Let us suppose that an infant, during dentition, has pulmonary catarrh or hooping-cough, and at the same time diarrhœa. Should the excessive intestinal secretion be abruptly suppressed, the pulmonary inflammation will very often assume a corresponding increase of intensity: and the patients may evidently die from the imprudence of the treatment. But in such a case the question is not as to diarrhœa in relation to dentition: what we have to consider is a pulmonary affection which may be aggravated by suppressing the intestinal flux quite irrespective of the patient's period of life. Do we not see the cough of adult phthisical patients relieved by diarrhœa, and do we not see the severity of their cough and fever return when they become constipated? It would, however, be pushing the assertion beyond the limits of truth were I to argue that diarrhœa is always a salutary crisis in phthisis: still, you can understand that without maintaining any such proposition, I may hold that the diarrhœa if it do not proceed too far may be a favorable occurrence. Now this restriction is similarly applicable to the diarrhœa which, in illustration of the point before us, I supposed in the teething child, as in the case of the phthisical adult. This intestinal flux ought not in such a case to be abruptly suppressed: but it is quite different when diarrhœa is the sole complication of teething. Diarrhœa, under such circumstances, requires to be combated by the most active measures; and no untoward consequence will follow the adoption of such a course.

The intestinal complications of dentition are always most serious in children prematurely weaned. When the infant is unweaned, to give the breast almost always suffices to stop the purging, the administration of preparations of lime and bismuth being hardly necessary. When, however, the infant has been weaned, the practitioner finds himself in this cruel dilemma: he must either put the patient on low diet, which will ere long produce disastrous cachexia, or give such food as will daily excite new attacks of indigestion, which by the frequency of their recurrence will at last produce inflammation.

The infant, by being subjected to an unsuitable regimen, becomes the subject of enteritis. The affection is characterized by stools which are very profuse and frequent, consisting of a mixture of green and yellow matter, like chopped vegetables, to which the name of "*hachures d'herbes*" has been given: glairy and lienteric, they contain lumps of firmly curdled milk, which indicate that stomachal and intestinal digestion are not being performed, the food traversing the digestive canal without undergoing the normal changes. During the course of this chronic diarrhœa, even when the purging is not of more than ten or twelve days' duration, the infant is suddenly seized with bilious vomiting. A time soon comes when the food, be it what it may, whether soups made with milk or butter, panada, or even toast and water, are returned in the state in which they were taken, appearing to be no more acted upon in their passage through the intestines than if they had traversed an inert tube. The infant becomes perceptibly thinner: from morning to evening, and from evening to morning, it utters plaintive cries, and will not be comforted; and should a wet-nurse not be procured, to supply the sole description of aliment which agrees with it, death from inanition ensues. If it resist, its health is not the less seriously compromised; and when I shall have occasion to address you on the subject of rickets, I shall have to tell you that in most cases it is caused by unsuitable and insufficient food.

But when children in this deplorable state are subjected to the influence of the summer season, the diarrhœa assumes a special character, and becomes infantile cholera.

You now, gentlemen, understand my reasons for saying that children ought not to be weaned till they have passed the period during which formidable complications of dentition are of most frequent occurrence.

My rule, provided there be no serious obstacles to surmount other than the wishes of the family, is not to wean the child till after the complete evolution of the canine teeth, which is generally a more difficult process than the evolution of the incisors or first molars. My rule, therefore, is to wait, irrespective of age, till the infant has sixteen teeth.

When, however, as is unfortunately too often the case, circumstances render it impossible to continue lactation till sixteen teeth have appeared, I wait till there are at least twelve. Between the evolution of the third and fourth groups, there is generally a sufficiently long interval of rest for the digestive organs to recover from the fatigues to which they have been subjected, and to become more disposed to receive the new aliment to which they are unaccustomed.

Should pressing reasons relating to the health of the nursing mother, or considerations of a pecuniary or personal character necessitate the premature weaning of the child, an endeavor must be made to prolong lactation till the evolution of a group of teeth already commenced has been completed: if the infant has only three or four incisors, we ought to wait till it has six. It is specially important to wait should the time fixed on for the weaning be the hot season, for summer weather (contrary to vulgar belief) is the most unsuitable for weaning, inasmuch as it favors the development of diarrhœa in the terrible form of infantile cholera.

Under no circumstances ought weaning to be abruptly carried out. From the age of four, five, or six months, the infant will have become accustomed to take, in addition to the nurse's milk, farinaceous food and soups, the number of feedings and quantity of food being increased as time goes on. By and by, when the teeth have appeared, creams may be added to this aliment, also eggs beat up with milk, and pillars of bread soaked in the yolk of fresh boiled eggs: afterwards, there will be given some chicken bones to be sucked: a little meat will be then allowed; and so by insensible degrees, the stomach and intestines being sufficiently prepared, and dentition sufficiently advanced, the breast-milk may be wholly discontinued and the new diet commenced.

By regulating lactation in this manner, by thus accomplishing the weaning, we hold in reserve useful curative means should the infant become ill; for then the mother's milk will constitute, under all circumstances, the best tisane which could be administered.

LECTURE LXXIV.

DYSENTERY.

Most formidable of all Epidemic Diseases.—Its Causes unknown.—Eating Fruit blamed without reason.—Opinion of the ancients on this point.—Different Forms of the Disease.—Character of the Stools: Tenesmus.—Bilious, Inflammatory, Rheumatic, Putrid, and Malignant Forms of Dysentery.—Anatomical Lesions.—Treatment: Evacuant the most useful: Employment of Saline Purgatives, Calomel, Emetics, Topical Remedies, and Caustic Injections.—Dangers of Opium.—Sequelæ of Dysentery, viz., Dropsy, Paralysis, and Abscess of the Liver.—Intractable Diarrhœa.—Intestinal Perforation.

GENTLEMEN: The year 1859 will be looked back to as remarkable for the frightful epidemic of dysentery which we have just traversed. The disease has prevailed throughout all France in a more general manner than on the occasion of previous dysenteric outbreaks; and it has not spared Paris, where for the last hundred years isolated cases only have occurred. The epidemic, exhibiting its usual features, declared itself about the end of July: it attained its maximum severity in September: by the end of October, it had moderated greatly; and though it continued during November and December, it was much less prevalent.

You have had an opportunity of studying the disease in the clinical wards; and during the last few days, you have seen in bed 5 of St. Agnes Ward, a man, and in bed 11 of St. Bernard Ward, a woman, suffering from dysentery. The man is convalescent.

The woman died: and I showed you the terrible intestinal lesions which were found on examining her body—lesions which unfortunately testified to the uselessness of therapeutic measures in similar cases. The large intestine, throughout its entire extent, presented appearances of acute inflammation, there being also at some points ulcerations, and at others, gangrenous patches. The gangrene had in some places extended to the subperitoneal membrane. Traces of inflammation were found as high up as the small intestine; but let me call your attention to the remarkable fact that there was no lesion of Peyer's glands: this is contrary to what occurs in dothinenteria, in which ulceration of these glands is the anatomical character of the disease.

Here, in a few words, is this poor woman's case.

Eight days before her admission to the hospital, she was attacked by diarrhœa, the stools very soon containing blood and glairy matter. They became very frequent; and if the woman's statement is to be believed, she had had fifteen in an hour. According to the attendants, she had gone at least seven or eight times an hour, which would make the number of motions amount to 160 or 180 in the twenty-four hours. The dejections had the appearance of long-boiled flesh mixed with decolorized blood. This was the *lotura carnium* or flesh-washings described by Stoll, and by him considered as always of the worst possible augury. The general state of the patient was deplorable: the eyes were sunken, the skin was icy cold, and while it became colder and colder, it acquired a bluish tint: the tongue also was cold. Excepting that there was no change in the voice, the condition of this woman was exactly like the algidity of cholera morbus. Pressure on the abdomen produced only slight pain. Although on the second day after the patient was admitted to our wards, the stools had diminished in frequency, the general symptoms continued quite as formidable as before. The pulse was imperceptible at the wrist, and could with difficulty be felt at the carotids. The woman died on the twelfth or thirteenth day from the beginning of the disease.

I stated, gentlemen, that the epidemic of this year made its appearance toward the end of July, and was characterized by the usual features of epidemic dysentery. It is generally during summer, and principally during its greatest heats, that dysentery breaks out. At first, only a small number of persons are attacked; but up to September, there is a progressive increase in the number of seizures; and it is during the first fortnight of this month, that the ravages of the epidemic attain their maximum: after this, the number of new cases slowly decreases up to the end of autumn, by which time, generally speaking, the epidemic has disappeared. In some epidemics, however, seizures continue to occur up to January, as in the epidemic of 1765, described by Zimmermann.[*]

Of all epidemic diseases, dysentery is certainly the most severe and the most deadly. Outbreaks of dothinenteria, scarlatina, small-pox, diphtheria, and even cholera morbus itself carry off fewer victims. Desgenettes states that dysentery killed a greater number of our soldiers between 1792 and 1815 than fell in the great battles of the Empire.[†] This we can understand, for dysentery is not only very deadly, but it breaks out as an epidemic much more frequently than other diseases, and invades particular regions at very short intervals.

What are the *causes* of epidemic dysentery? The causes of this as of most other epidemics elude our observation: though the inquiry has been pursued very carefully, nothing positive has yet been established in respect of the conditions in which it originates.

In Tours there are two barracks, one in the eastern and the other in the western faubourg: they are similarly situated, and at an equal distance from the river which flows through the town. The same hygienical system is adopted in both; and in both also, the dietary of the soldiers is exactly similar. Nevertheless, during the twenty years which preceded, and the ten years which followed, the period during which I studied at Tours, it was always in the cavalry barracks that the disease first broke out. The few soldiers belonging to infantry regiments who were seized with dysentery at the beginning of the epidemic had contracted it in hospital, whither they had been sent for other diseases: and it was not till a later period that the epidemic showed itself in the infantry barracks.

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[*] ZIMMERMANN: Von der Ruhr unter dem Volke, 1765. Zurich, 1767—Traduction Française par Lefebvre de Villebrune; Paris, 1775.

[†] DESGENETTES: Notes pour servir à l'histoire de la Médecine Militaire de l'Armée d'Italie. [*Recueil de la Société de Médecine de Paris*, année 1797, t. ii.]

Here then is a case in which no charge can be brought against the local situation, the hygienical conditions, or the food. You are aware that it is very common to impute the causation of dysentery to the use of fruits: so general is this opinion, that one finds it rather difficult not to acquiesce in it. It is, however, a prejudice against which the greatest practitioners of former times have contended. Without going back to Alexander of Tralles, who taught that grapes and other fruits not only did not produce dysentery, but were, on the contrary, really preventive, and very often curative, I shall lay before you the views on this subject of Stoll and Zimmermann, two of the most illustrious physicians of last century.

Zimmermann says: "The majority of physicians and women-doctors [*commères*] regard fruits of the season as the true and special cause of all dysenteric attacks. It is an opinion which I have refuted in my treatise on practical experience in medicine; and the great physicians are on my side. Besides, the disease [the epidemic of 1765] appeared among our peasantry in June, when the only procurable fruit were the large cherries of Basle, and their high price placed them beyond the reach of these people: again, during the season in question, there was a great dearth of fruits. It is quite true that the unripe fruit of bad years may occasion colic, purging, as well as intestinal obstruction, and all the symptoms met with in nervous diseases; but still, no one has ever observed such a result as an epidemic of dysentery. I say, moreover, that cooling fruits even when not ripe, cannot cause dysentery."

Gentlemen, I attach no value to Zimmermann's reasons, which I join with you in condemning, as merely the echo of the humoral theories of his time; but that does not affect his clinical statement, which is quite applicable to what we are now seeing. Last year, for instance, when fruits were very abundant, there were hardly any cases of dysentery; and this season, when fruits are scarce almost everywhere, we have this formidable epidemic.

It cannot be denied that the spread of the disease is promoted by unfavorable hygienical conditions, such as hot weather, bad food, and crowding; but they are only proximate causes, to which we must add another something, and that something we call the *epidemic constitution*.

We cannot otherwise explain why dysentery does not always show itself in those years in which the heat is greatest; why it does not invariably appear where there is overcrowding: and why, for example (not to go beyond this line of argnment), it so generally spares Paris, so little spared by other epidemic diseases. Therefore, as I have just been saying, we are in absolute ignorance of its primary cause.

We know, however, that when once developed, it is exceedingly contagious; although Stoll denies the contagious character of [illegible] ll as of scarlatina. That both diseases, however, are [illegible]
In small places, it is easier than in great centres [illegible]
the disease to its source, and to follow its p[illegible]
invades. Have not our honourable collea[illegible]
dysentery, at intervals, co[illegible]
vails in a regiment, it [illegible]
halts, thus following [illegible]
when from the Al[illegible]
patients have b[illegible] on of
an epidemic [illegible] rrival of
these sick sol[illegible]

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* Haspel: M[illegible]

Gentlemen, before describing the *symptoms* of dysentery, I must tell you that the disease does not always assume the same forms in all epidemics. On this subject, read the accounts which have been left to us by Pringle,* Zimmermann, and particularly by Stoll.† There you will see that the disease is sometimes purely inflammatory, and at other times rheumatic or catarrhal, for, according to the distinguished physician of Vienna, there is no difference between rheumatism and catarrh except in the seat of the disease: dysentery, he calls rheumatism or catarrh of the intestines, or abdominal coryza. The form of dysentery which generally predominates is the bilious.

At the beginning of the attack, and without appreciable cause, the patients are seized with diarrhœa; in twenty-four or forty-eight hours, the stools change their nature and aspect, and become dysenteric. They contain glairy, yellowish-white mucosity, resembling a mixture of the white and yolk of an imperfectly cooked egg; or there is an admixture of transparent glairy matter, with thin streaks of blood; or there may be an appearance which recalls that of peripneumonic sputa.

The evacuations are preceded by frequent desire to go to stool, at times almost incessant, but which result in not more than a spoonful or half a teaspoonful being passed at any one time. They are accompanied by great pain in the anus, which sometimes extends to the bladder, producing dysuria. Tenesmus of an exceedingly painful nature is an essential characteristic of dysentery.

There is likewise colic, more or less acute, which is felt principally around the navel and in the course of the large intestine. The abdominal pain is increased on pressure, particularly in the left iliac fossa.

The tenesmus has been explained by alleging the existence of spasmodic contraction of the sphincter; but this explanation is at once refuted by examining the patients—which I have done many times in your presence—when we find that the anus, in place of being tight and closed, is sufficiently open to allow the five fingers to be introduced. The violent irritation, the acute inflammation of the intestinal mucous membrane, which is intensely red and turgid at the gaping orifice of the anus, quite accounts for the acute burning sensation felt by the patient, and for the painful constriction of the intestine, the lower sphincter of which is evidently inert and paralyzed. It is not unusual for this combination of paralysis of the sphincter with turgidity of the mucous membrane, to cause prolapsus of the rectum.

Along with the glairy frothy matter of which I have been speaking, dysenteric stools likewise contain pure blood, short thin shreds of false [illegible] greatly resembling burst boiled rice, and which, when somewhat [illegible] thicker, constitute that which the patients call the scrapings [illegible]

[illegible] ity of dysenteric matter evacuated at each effort to defecate is [illegible] the efforts are repeated at very short intervals, a patient may [illegible] four hours, as many as twenty, forty, fifty, or even [illegible] the total amount passed from the bowels may [illegible] two, three, four, or six litres.

[illegible] nd one specially characteristic of the malady [illegible] here are seen in the midst of the stools small

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[illegible] he Diseases of the Army: London, 1772
1793.]
[illegible] ecine Pratique (par Mahon, Paris, 18[illegible]
[illegible] Vindobonensi: Viennæ, 1783.

masses of fecal matter, which are moulded, and more or less hard; some of these masses are even scybalous, such as are passed by persons suffering from constipation. In point of fact, gentlemen, in accordance with Stoll's correct observation, dysentery ought to be considered as one of those disorders in which the bowels are confined. So much does it differ from diarrhœa, that although, in some cases, it is complicated with iliac diarrhœa as an epiphenomenon, for the most part the diarrhœal excretions which supervene in dysentery announce the termination of the attack.

About the eighth, tenth, or fourteenth day of the disease, dysenteric stools are horribly fetid, and contain almost no mucus; they consist of a reddish, serous liquid, in which float shreds resembling the débris of overstewed meat. These *flesh-washings*, to adopt Stoll's expression, almost invariably indicate gangrene of the intestines. Then also, and even sooner, the evacuations contain pus.

Having described the nature of dysenteric stools and the local phenomena by which they are accompanied, I now come to speak of the *general symptoms*, which vary according to the particular form which dysentery assumes in different epidemics, and which may also be met with in the same epidemic.

In the *bilious* form, patients complain of loss of appetite, of a bitter taste in the mouth, of nausea, and of vomiting a greenish matter. The tongue is covered with a saburral coat. The rigors which usher in the attack are of short duration, and there is not much fever. The abdominal pains are of moderate severity. In this form of dysentery, contrary to what generally occurs, there is diarrhœa. The stools, however, though frequent, are scanty: they consist of a greenish or yellowish liquid, in which float mucous, glairy, sanguinolent matters, and sometimes blood nearly pure.

Inflammatory dysentery is characterized by burning fever, a notable frequency and hardness of pulse, heat of skin, and sometimes copious sweating. The face has a more or less bright red appearance. The tongue, in place of being saburral, is red, dry, and clean. The patients suffer from headache. The abdominal pains, violent and *torminous*, to use the consecrated term, are aggravated by the least pressure. In some individuals, the abdomen is tympanitic. The stools are few in number. As they become more frequent, the febrile excitement soon subsides.

In *rheumatic dysentery*, the abdominal pains are most marked. Each time the patient goes to stool, his sufferings are depicted on his countenance, which is expressive of the most painful anxiety; and there is extreme tenesmus.

But the chief characteristic of this form of dysentery is the occurrence of metastatic affections of the joints, as was accurately pointed out by Stoll. Sometimes the metastatic affections are localized in one particular place; and it seems to me, that the knees are most frequently the elected situations. The articular rheumatic inflammation is generally rather transient, or at least is not severe; but sometimes it is of long duration, and of so severe a character that the great quantity of the synovial effusion causes rupture of the capsule. The rheumatic attacks are generally erratic, seizing first one place and then another. The chest may be attacked just at the very time when the dysentery is beginning to subside: the patients complain of pleuritic or simply pleurodynic pains: others suffer from oppression, cough, and all the other symptoms of catarrh. Generally, the catarrhal or rheumatic affections yield spontaneously within a very few days.

The transformation of dysentery into rheumatism was observed by Dr. Gondouin in an epidemic which prevailed in the department of Sarthe.

When dysentery prevails in a district in which palustral fevers are endemic, it is not unusual for the accompanying fever to become intermittent, assuming the tertian or double tertian type. The *intermittent* is considered the least formidable form of dysentery.

Under all its forms, dysentery follows a regular course. Putridity and malignity come athwart that course, complicating it and leading to a fatal issue, as you saw in the case of the unfortunate woman of bed 11 St. Bernard Ward.

Algidity is its predominating characteristic. The skin becomes cold, and covered with cold sweat: the complexion becomes clay-colored: the features are shrunken and the eyes sunken: the extremities, point of the nose, and the tongue are cold.

The patient has all the appearances of an individual in the algide stage of Asiatic cholera morbus.

He has, however, merely the appearances of cholera, and we have still only to do with dysentery; but it is hardly necessary to say that the disease really changes its nature when cholera is epidemic in a locality already a prey to dysentery. As the slightest diarrhœa gives cholera a pretext for attack, it is not surprising that cholera should strike with fury those prepared by pre-existing dysentery to receive it, and carry them off before the dysentery has had time to pass through its stages.

The most marked signs then of malignity in dysentery are algidity coincident with a feeling of great general discomfort; extreme, suddenly supervening feebleness, attended sometimes with fainting fits, and prostration to so great a degree as to render the patients almost indifferent to everything going on around them. The pulse is exceedingly weak, small, and compressible.

There are, however, dysenteric patients in whom the temperature rises in place of falling, and in whom the pulse is accelerated and less compressible; there is burning thirst: the tongue is dry, and a fuliginous coat covers tongue and gums: aphthous ulcerations appear in the mouth.

During these dangerous periods of the disease, the stools become smaller and less frequent, acquire a fetid cadaveric odor, and contain matter resembling flesh washings. There is almost no abdominal pain. The patient complains of a sinking feeling at the precordium, of nausea, hiccup, and vomiting.

Then also, *parotiditis* occurs as a complication. The patients complain of pain at the angle of the jaw, where, on examination, there are found swelling, redness of the skin, and a sort of deepseated fluctuation. By pressing on the parotid region, and on the cheek in the course of Steno's duct, pus is made to issue from the orifice of that canal. The suppuration invades the surrounding cellular tissue, reaching the neck sometimes, and dissecting its muscular masses.

These symptoms announce that a fatal issue is near. Stupor comes on, complicated with slight convulsive movements, subsultus tendinum, and low delirium, death speedily closing the distressing scene.

Upon opening the body after death, the depth of the anatomical lesions perfectly account, up to a certain point, for the severity of the disease and its fatal issue. The intestinal lesions, which existed chiefly in the large intestine, were the result of violent inflammation: the mucous membrane, of a brownish deep-red, a color derived from blood mixed with intestinal secretions, is thickened, turgid, and softened: this turgidity and thickening extended to the other tunics, and even to the subperitoneal cellular tissue.

Here and there, ulcerations were observed, varying in size and depth according to the period of their commencement.

When, at its onset, dysentery strikes down individuals, the ulcerations, about the third or fourth day, are quite superficial, and covered with a muco-sanguinolent fluid: by the fifth day, they assume a very varied aspect: during the course of the second week of the disease, the mucous tunic is more or less destroyed in extent of surface and in depth, so as to expose the muscular tunic. Sometimes, even, the ulceration destroys likewise the muscular fibres and reaches the peritoneum: under such circumstances, there may occur peritoneal perforation leading to peritonitis, but this is a rare occurrence.

At other times, there are a multitude of small ulcerations, the orifices of an equal number of small abscesses formed in the submucous cellular tissue.

In other cases, or in other situations, there are seen gangrenous sloughs, completely detached at some points, and mixed with a sort of magma, a black, bloody porridgy matter, which covers the surface of the mucous membrane, and is in other places adherent to the parts whence it proceeds.

These gangrenous lesions may dissect a great part of the large intestine, so as to present an appearance of a great portion of its mucous coat being entirely destroyed.

When, from accidental causes, death does not occur till a remote period —till four or five months after the invasion of the first symptoms, and when recovery from the dysentery has taken place—the ulcerations are found to be cicatrized or nearly cicatrized; but then the cicatrices have given rise to other lesions which may have occasioned death. I refer to strictures of the intestinal tube caused by the contraction of the cicatricial tissue. These strictures explain the pains which often continue long after the disease. They explain the intestinal obstructions and occlusions, in which originate the attacks of subacute peritonitis under which the patients sink.

Buboes occur in dysentery, as in all other pestilential diseases. The mesenteric glands are swollen and inflamed, while some are in a state of suppuration.

The parenchymatous tissue of the liver, kidneys, and spleen, is softened. The gall-bladder is distended by black, pitchy, grumous bile. In some cases there are true hepatic abscesses.

Gentlemen, I showed you a patient who at the close of an attack of dothinenteria had had the symptoms characteristic of purulent infection. At the autopsy, as you will recollect, we found a large metastatic abscess in one of the psoæ muscles, and numerous abscesses of the same kind in lungs and liver. When speaking to you upon that case, I explained to you that my understanding of the way in which dothinenteric ulcerations become the starting-point of purulent infection, is the same as in the external wounds which we see in the surgical wards, or the placental wound of the womb which occurs after delivery. I likewise told you that, in all probability, the hepatic abscesses and the articular suppurations met with at the close of an attack of dysentery, proceed from the same cause. Henceforth at autopsies, it will be necessary to search carefully for metastatic abscesses of the lungs and kidneys, and to examine minutely the state of the veins leading from the large intestine to the liver.

Gentlemen, you will remark that I have said nothing about the lesions of the small intestine. I have not thought this necessary, because when lesions of the small intestine do occur, they are quite secondary. Spots, more or less red, and traces of existing inflammation are met with, and (as in the patient whose autopsy you witnessed), the glands of Brunner and Peyer are exempted from attack in a ratio the opposite of that met with in dothinenteria.

The disease has attacked the large intestine principally if not exclusively; let me now add, that the inflammatory lesions which I have pointed out to you are much more extensive when situated nearer the lower extremity of that part of the intestinal tube and become less and less formidable the nearer they get to the cæcum. In conclusion, dysentery is nothing more than *colitis*, but a colitis of a peculiar character, the special characteristics of which do not allow us to confound it with non-epidemic colitis—with that form, for example, of colitis which supervenes after an excessive dose of a drastic purgative, such as jalap or colocynth. Dysentery is also quite a different disease from those attacks of colitis so common in young children and old people, which occur irrespective of any epidemic constitution of the season.

To mention some symptoms only, the different kinds of colitis are characterized by sanguinolent, glairy, mucous stools, by tenesmus, which latter, however—and this is a point of differential diagnosis—is never so severe in simple colitis as in dysentery. The lesions, though never so profound and extensive in the one as in the other, are of similar nature in both—they are thickening of the large intestine accompanied by turgidity, redness, and ulcerations more or less serious. But the feature which essentially distinguishes dysentery from colitis is that the latter is a disease pertaining to the individual, for the most part mild and transient, generally yielding without its being necessary to resort to treatment of any great energy; while epidemic dysentery presents that assemblage of general symptoms of peculiar character and varying severity, which I have described to you; and dysentery, moreover, when left to itself, has a tendency to become aggravated, its gravity in some epidemics being so great, that it often baffles all our therapeutic efforts.

What are the measures by which we ought to oppose this formidable disease? The importance of this question makes it incumbent on me to give a certain degree of development to the reply.

Having seen at Tours, Versailles, and Paris, several epidemics of dysentery, which carried off men in the prime of age and strength, as well as old people and young children, I am able to speak, and I wish to speak, from my own personal experience. Having been intrusted by the committee on epidemics to give an account to the Academy of Medicine of the reports annually received from the departments, I have had to compare observations brought together from all sides, comparing them at the same time with observations collected by myself. Finally, in reading the accounts left to us by our predecessors, I have been able to complete my own experience by adding to it that of others, and to form, on a sound basis, opinions as to the treatment which presents the greatest prospects of success.*

Thirty or forty years ago, we seemed to have quite lost sight of the traditions of past centuries. Broussais had made a *tabula rasa* of everything said prior to his day, and pretended to have re-established medicine on new foundations. Inflammation according to him was dominant everywhere, and was always of the same nature. In dysentery, he saw colitis only; and starting from that point, the treatment was necessarily antiphlogistic. Endowed with a great talent for exposition, influenced by an impetuous mind and a profound conviction of the soundness of his views, he proclaimed that no treatment except the antiphlogistic was right: his pupils "swore by their master's word," and spread his opinions everywhere, till they became accepted, without any modification, by so large a number of physicians that for a long time they dominated in medicine.

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* TROUSSEAU: Rapports sur les Epidémies qui ont régné en France, pendant l'année 1856. [*Mémoires de l'Académie*, Paris, 1858, t. xxii.]

In 1823, however, Bretonneau, a man profoundly clinical, dismayed at the non-success of a system of treatment, based upon a preconceived theory rather than upon sound observation, resolved to place himself in opposition to the deplorable practice which resulted from the doctrine of the Val-de-Grace.

Having before him, as exemplified in his own practice and that of many others, the sad results of indiscriminate resort to the antiphlogistic treatment on all occasions and without reference to the form of the disease, he set himself to make trial of the treatment by purgatives in accordance with the plan followed by Stoll, Zimmermann, and Pringle, all of whom stated that they had found it very useful.

The trial was attended by success. He then sought for an explanation of the successful results obtained; and he came to the conclusion that in dysentery, as in dothinenteria, the *quality*, the specificity of the local inflammation, plays a much more important part than its *quantity:* he likewise thought, that most probably the beneficial action of purgatives was due to their substituting for a specific local inflammation of bad type, another inflammation which, although it has also a specific character, has a natural tendency to cease.

While in respect of Broussais's doctrine, theory took the lead, and moulded facts to its service, Bretonneau's doctrine advanced, under the simultaneous and combined support of observation and theory. From that time, and in the different circumstances in which he was placed, the illustrious physician of the hospital of Tours, recognized that the purgative treatment was that most frequently indicated in dysentery.

In the account given by Dr. H. Parmentier and me of an epidemic which prevailed in 1826 in the department of Indre-et-Loire, you will find it stated that a really great proportion of recoveries followed the treatment just described.* I have long employed it; and it has rendered me signal services in the different epidemics against which I have had to contend.

Such was the case in the epidemic of 1848 in the garrison of Versailles, whither I went every morning to study the disease in the wards of the military hospital, then in charge of my honorable colleagues Drs. Perrier, Follet, and Godard.

In the reports communicated to the Academy of Medicine, to which I have just been alluding, there is expressed an almost unanimous opinion in favor of this powerful method of treatment. Nearly all the reporters state that the administration of purgatives was the chief means by which they opposed the disease; and that the purgatives which they principally used were the neutral salts, such as sulphate of soda, sulphate of magnesia, and the neutral tartrate of potash and soda, called *sel de Seignette.*

These are the medicines which you have seen me prescribe in the cases which have come under your observation. In my civil practice, I always have recourse to them, particularly in the commune in which my estate is situated, where this year dysentery has committed great ravages. My own household was not spared, several members have been attacked, and one child having died. My farm-bailiff was seized with the malady: I gave him the neutral salts; and though he committed imprudences, he recovered. Generally persons recover who are treated in this way, while those who neglect to call in medical aid, or are very late in doing so, and who consequently are not actively treated, either die or continue deplorable invalids for six weeks or two months.

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* TROUSSEAU et PARMENTIER: Mémoire sur une Epidémie de Dysenterie qui regna dans le départe-d'Indre-et-Loire. [*Archives Générales de Médecine,* pour l'année 1827.]

The evacuant method praised by the physicians of last century, and particularly the administration of the neutral salts in purgative doses once daily, or morning and evening, so as to induce diarrhœa, is, therefore, the best treatment of dysentery.

Does it follow that we are to confine ourselves to the use of the sulphates of soda and magnesia, and the salts of Seignette? Certainly not: there are cases in which other purgatives may be employed with advantage.

In 1812, there was dysentery at Gibraltar; numerous deaths from it had occurred, when Dr. Amiel, surgeon-major of the 12th regiment of infantry of the English army, conceived the idea of having recourse to sublimed calomel. This he gave in doses of one gramme, eighty centigrammes [27 grains] morning and evening, till the evacuation ceased to be mucous and sanguinolent, and had assumed a deep red color: the dose was then reduced; and afterwards, the calomel was discontinued, when lavements were used in its place. So great was the success of this treatment, that the director-general of the military medical service made obligatory its employment by all the other physicians.

In the epidemic of Touraine, which I spoke of a minute or two ago, Bretonneau and I tried this plan of treatment, and obtained similar results. We were, however, obliged to abandon it, on account of the salivation it occasioned in some patients, a complication from which the Gibraltar patients were exempt. This difference arose from the Gibraltar epidemic having occurred during the hot weather, and in a place where the temperature is naturally very high, so that the patients ran no risk of chills: at Tours, on the other hand, at the time we gave the calomel the bad weather was setting in, and the patients being obliged to pass several hours on the stool [*sur leur chaise percée*], were exposed to chills which favored the toxic effects of the mercury.

How does the calomel act? Is its action exclusively topical and substitutive, like that of the neutral salts? Or, is it more general, and does the benefit derived from this medicine depend on its action as an alterative? These are difficult questions to answer. I should, however, be rather inclined to adopt the former of the two explanations, and to accord only a very slight share in the beneficial results to its alterative powers, when I consider that calomel is never so useful as when administered internally, and that I have never heard it said (except by Boag as quoted by Gmelin),* that mercurial frictions of the skin are of any use.

Calomel was also the basis of the treatment of Dr. Leclerc, but our colleague of Tours in place of giving it in large doses, administered it at first in fractional quantities, that is to say in doses of one centigramme [$\frac{1}{7}$th of a grain] morning and evening, afterwards increasing the quantity by an additional centigramme on succeeding days. Simultaneously, and with a view to moderate the tenesmus, M. Leclerc prescribes inunction of the abdomen with an ointment of belladonna.

I have often had recourse to and have observed the good effects of these inunctions. Adopting at the same time the medication of MM. Amiel and Leclerc in a modified form, I have given calomel *fracta dosi* according to Law's method, that is to say, in doses of five centigrammes divided into ten packets, one of which was administered every hour.

This method has appeared to me particularly advantageous in the treatment of dysentery in children, as it is very difficult to get them to take saline purgatives. A similar remark applies to the purgatives to which it is necessary to add rhubarb, a combination in favor with some excellent physicians.

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* GMELIN: in Murray's Apparatus Medicaminum, Pars II: Gottingæ, 1793.

I have now to speak to you of *emetics*, which occupy an important place in the evacuant method of treatment, as applied to dysentery.

About the middle of the seventeenth century, Piso, the botanist, having, when in the Brazils, heard the praises of a root which was administered in powder, endeavored to introduce it as a medicine;* but the medical profession hardly paid any attention to his writings. It was in vain that Legros, who had made three voyages to America, brought a supply to France, and offered for sale "ipecacuan"—for that was the name of the wonderful plant. The new remedy received no credit save from the ranks of quackery. In 1686, nearly, in fact, at the date at which cinchona, the famous remedy of Talbot, had procured for its discoverer the patronage of Louis XIV and a large fortune, a French merchant, named Grenier, imported from the Brazils 75 kilogrammes [between 150 and 160 pounds] of the root of ipecacuan. Not knowing how to turn it to account, nor how to give celebrity to his new medicine, he assumed as a partner Adrian Helvetius, a Dutch physician practicing in Paris, whom he made acquainted with the antidysenteric virtues of his arcanum. Helvetius made his first experiments upon obscure persons, then upon persons higher in the social scale, and finally upon the Dauphin himself, whom he cured of a sanguineous flux: he then obtained permission from the King to make public experiments at the Hôtel-Dieu. His experiments having succeeded, he obtained a monopoly of the sale of his remedy, besides a money grant of a thousand pounds. Helvetius, however, acting the part of an unscrupulous partner, kept to himself all the honor and profit: Grenier then tried to be reinstated in his rights, and with that object instituted in the Parliament a suit, which he lost. Grenier, indignant at the bad faith of Helvetius, divulged the secret; and from that time ipecacuan became public property. Afterwards, by one of those reactions so common in the history of opinion, a tendency arose to abuse the remedy which had had so much trouble in making itself at all accepted.

The utility of ipecacuan in dysentery is, however, incontestable; it is chiefly beneficial in the bilious form, at the beginning of the attack, when the coated tongue indicates a very marked saburral state. It acts, on the same principle as purgatives, as a powerful modifier, and its action is equally beneficial on the entire digestive canal as on the stomach.

To sum up: here is the plan I generally adopt in the treatment of epidemic dysentery! At the beginning of the attack, I prescribe ipecacuan in emetic doses, according to the formula which I have given you so often: three grammes [46½ grains] are divided into four powders, one of which is taken every ten minutes till vomiting is induced. Next day, and often even on the evening of the same day in which the ipecacuan has been thus administered, I give one of the neutral salts in a dose of from 15 to 25 grammes [231½ to 386 grains], which ought to be repeated during the following twenty-four hours. I go on giving the saline medicine till there is an obvious modification in the nature of the stools, or in other words, till they cease to contain glairy sanguinolent matter and become diarrhœal.

But concurrently with the use of the means now described, I attack the disease by topical agents, which have a still more direct action on the affected parts. I use styptic and caustic lavements with sulphate of zinc, sulphate of copper, and nitrate of silver. For a child, I use nitrate of silver in the proportion of from 5 to 10 centigrammes [¾ of a grain to 1½ grain] of the nitrate to 125 grammes [4½ fluid ounces] of water: for an adult the proportions are from 20 to 75 centigrammes [3 to 11½ grains] to 200 grammes [7 fluid ounces] of water. The sulphates of copper and zinc are used in the proportions of about 5 centigrammes [¾ of a grain] for a child and one gramme for an adult. The lavements are repeated two or three times in the twenty-four hours. They ought to be retained within the bowel as long as possible: to promote this object a lavement of pure water ought in the first instance to be administered, and then the medicated lavement must be slowly injected.

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* Piso: De Medicina Brasiliensi; et Historia Rerum Naturalium Brasiliæ. Lugduni Batav., 1648.

Lavements of the acetate of lead have been recommended, and I have employed them: without inducing any toxic effects, I have used a solution of from 30 to 60 grammes [460 to 920 grains] of acetate of lead to a litre [rather more than 35 fluid ounces] of distilled water.

Gentlemen, you will be surprised that hitherto I have said nothing of opium, which in the opinion of some physicians seems to be a remedy essential in the treatment of dysentery. I have only to mention it that I may raise my voice against the lamentable manner in which it is too often abused; and I shall recapitulate what I said at length on that point in my lectures on diarrhœa.

When opium is indicated it is not for the purpose of stopping the dysenteric flux, but for moderating the accompanying pains and particularly for checking the vomiting, which renders the administration of other medicines impossible. In all such cases, the opium must be given in very small doses, beginning with one drop of the laudanum of Sydenham, which may be repeated every hour according to the persistence of the symptoms for which it is administered: the doses of opium must be small, for by giving large doses, the malady will become complicated by formidable typhoid symptoms.

There is another point in the treatment of dysentery upon which I must make some remarks. Every day you hear me prescribe soups for our patients, even for those who are in a very bad state: you observe that I insist upon their taking three or four times a day a small quantity of thick panada. As a tisane, I order barley-water, rice-water, albuminous water,* or the white decoction of Sydenham, which is simply a form of toast and water. To this practice I attach extreme importance. In dysentery, as in typhoid fever, I look upon alimentation as a matter of absolute necessity; and this opinion has been confirmed by long experience.

You thus understand, gentlemen, that I abstain from antiphlogistic treatment because it would be completely opposed to my alimenting the patients, which I regard as an imperative indication. It is only when the predominating symptoms are of a purely inflammatory nature that the application of leeches is right: such cases are unusual; but when they do occur, they ought without delay to be met as I have stated.

The treatment which I have recommended to you, based on my own practice and on the experience of numerous physicians, though that on which we ought to rely, is unfortunately not infallible, and is much less successful in some epidemics than in others.

In conclusion, I repeat, that dysentery is the most formidable and dangerous of all epidemic diseases. Even when patients have resisted its first assaults, and seem to be reaching convalescence, danger is not past, and there are evil consequences to be dreaded after the disease.

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* See page 457 of this volume.

I do not now speak of the dropsical affections of a more or less general nature, or of the paralytic seizures which supervene during the course of the disease, such as supervene in the course of serious fevers, in dothinenteria, for example, which profoundly implicate the whole economy. However alarming these complications may be, they can be got rid of by tonic regimen, restorative diet, and hygienical care. Nor do I speak of purulent infection, or of hepatitis with abscess of the liver, which, though rare, have nevertheless been noted among the sequelæ of dysentery.

I speak of intractable diarrhœa caused and kept up by lesions, more or less deepseated and more or less extensive, of the large intestine, lesions which are always accompanied by an inflammation, an irritation, which makes itself felt throughout the rest of the intestinal canal, disturbing its functions, exhausting the strength of the patients, and causing them to sink with every symptom of hectic fever, against which all therapeutic measures are unavailing.

Finally, the intestinal adhesions may lead to *perforations*, as I stated when describing the pathological anatomy of dysentery. They may occur at a period more or less remote, giving rise to rapidly mortal peritonitis. Attacks of peritonitis may also originate in *intestinal obstruction*, caused by the contraction of cicatricial tissue bringing the walls of the tube into mutual proximity, so diminishing its calibre, and constituting stricture.

LECTURE LXXV.

CONSTIPATION.

Constipation is not necessarily a state of impaired health.—Causes. Treatment: Influence of Will and Habit: Cold Lavements: Suppositories of Cacao-nut butter, Soap, and hardened Honey: Mucilaginous Lavements: Belladonna, with or without small doses of Castor Oil.—In Obstinate Constipation have recourse to Drastic Purgatives.—Hygienical Measures: Regimen: Bran-bread.

Gentlemen: I have already, when speaking of dyspepsia, made a few remarks regarding constipation: but the subject is one which merits being treated at greater length; and, morever, I ought to state to you the reasons which induce me to treat by means so very different, a symptom which, in all patients, seems identical.

For the present, I exclude from consideration, mechanical obstacles to the passage of the fæces. I exclude tumors, and physical obstacles, using the word constipation in its ordinary meaning. I consider that constipation exists whenever the stools are few in number, irrespective of any mechanical impediment to the passage of the fecal matter.

You must bear in mind, gentlemen, that in certain persons constipation is not an infirmity—that it is a state of the system—that unless it go beyond certain limits, it cannot be looked upon as a disease.

When you recollect that there exists in the stools, in addition to the residuum of the aliment, a large quantity of juices secreted by the salivary glands, liver, pancreas and glands situated in the intestinal mucous membrane, you will understand that these juices may vary infinitely in quantity, not only in respect of the nature of the food and drink, but also in respect of the idiosyncrasies of the individual. You do not say that a man is in a state of disease because he perspires very little; and you have no more right to say so, because his digestve apparatus is in an analogous condition.

Although, as a rule, every adult man ought to have a stool daily, there are some persons, who, from the peculiarity I have just mentioned, have an alvine evacuation only once in two or three days, and in whom constipation belongs to a state of health. So true is this, that should the individuals of whom I speak have a non-diarrhœal stool every day, they will experience pains in the bowels, borborygmi, a feeling of debility and general discomfort, precisely similar to the effects produced on other people by diarrhœa. The individual who is physiologically constipated—if I may use so incorrect an expression—has relatively diarrhœa when he has daily a moulded motion.

The contents of the intestines pass onwards in virtue of the peristaltic movement, and in no part of the canal is that movement more energetic than in the small intestine: in the large intestine, it is slower, or at least less efficacious, and the contractions easily exhaust themselves by acting on the fæces accumulated in the rectum, and on the sphincter. We can any day appreciate these effects, when we resist the sensation by which we are apprised of a need to empty the bowel. We can generally accomplish this without difficulty, provided there be no excessive accumulation in the rectum.

Habitual resistance of the peristaltic movement ends by enfeebling the excitability of the intestine, which exhausts itself in superfluous efforts, and becomes expended like all the other muscles; and which becomes so distended by gaseous and other contents, that the muscular tunic loses its contractile power just as do all hollow muscles when stretched beyond their normal distensibility.

However, the continual contact of fecal matter with the extremity of the intestine impairs the sensibility of the mucous and muscular coats, and the synergic contraction of the upper portions of the large intestine either does not take place, or takes place in a most inefficacious manner.

Gentlemen, I presume that you understand this mechanism. In the normal state, whenever you, in any way, irritate the lower portions of the rectum, you excite, in addition to the immediate contraction of the muscular coat of that portion of intestine, synergic contraction of the portions situated above, and throughout the whole intestinal canal upwards even to the stomach, there being produced by the augmented peristaltic motion: the entire contents of the intestines are propelled downwards, and there is thus established a state of diarrhœa. You see then that a solidarity exists between all the portions of the alimentary canal, a solidarity both in respect of excess and deficiency of action.

Here then, it is sluggishness of the intestine originating in and maintained by bad habits, which is the cause of the constipation. We shall afterwards see, when we come to speak of treatment, that the will, so powerful in causing the evil, is equally powerful in remedying it.

It is not fecal matter only which is accumulated in the large intestine: the gaseous contents play a not less active part, and contribute not less powerfully to destroy the elasticity of the muscular coat of the intestine.

This new anatomical condition, which in youth, and even in adult age, is so frequently the result of carelessness on the part of the patients, becomes in some degree a natural state at a more advanced period of life; for, with increasing years, the intestinal muscular tunic loses its tone, just as do the vesical muscular tunic, and certain muscles of organic life, those of the lungs, for example, which allow themselves to be distended so as to cause pulmonary emphysema. In the same way, constipation depending on dilatation of the large intestine is not an accident, but, in a certain sense, a normal state in old people.

Thus, gentlemen, habitual distension of the large intestine leads to its muscular atony, in consequence of which the fecal matter does not easily proceed onward, and to a certain extent may be said only to make way from the pressure of the new accumulations behind: this atony is unquestionably the cause which most certainly produces constipation. But the rectum is certainly the portion of the large intestine which is most essential to defecation. Provided with powerful fibres, strongly contractile, supplied with numerous nerves, and terminated by the anus, which is endowed with exquisite sensibility, this intestine cannot be normally filled with stercoral matter without having its contractility aroused, without trying to get rid of its contents. But when, with old age, the sensibility becomes blunted, and the muscular contractility enfeebled; or when the individual by obstinately retaining the fecal matter dulls sensibility, and accustoms the intestinal muscles to a state of constant distension, there is formed that dilatation of the rectum, that pouch, to which the term *ampoule rectale* has been applied. There, the fæces accumulate, and become agglutinated so as to form enormous boluses pressing on the anus, which are inexpulsable except by real parturient travail or by surgical intervention. This kind of constipation is more common than young practitioners suppose; and there is this difficulty in recognizing it, that it is sometimes accompanied by a diarrhœal flux depending upon two causes, viz., the local irritation excited by the presence of the excremental bolus, and the excessive contraction of the colon synergically induced by the irritation of the rectum—a form of diarrhœa belonging to the class of diarrhœal affections due to the excitation of the lower part of the intestinal canal, and which we have already studied together in detail.*

Although the muscular tunic of the alimentary canal may be regarded as the chief agent in pushing onwards the fecal matter to the colon—although in the child, and even in the adolescent, it is almost the sole agent in defecation, in the sense that no other agency is required for emptying the intestine—this is not the case in old people, and in persons habitually constipated. I may add, indeed, that in general, after getting beyond the age of maturity, it is so no longer.

The assistance of the expiratory muscles is then required. Now, gentlemen, these muscles are under certain circumstances liable to become enfeebled, which renders their assisting efforts in a great measure inefficacious. I am not speaking at present of senile debility, which has in the first instance suggested itself to you: I speak of the debility consequent upon repeated pregnancies. When the abdominal walls have been often distended by the product of conception they become extraordinarily flaccid, and unfitted to co-operate efficiently in the expulsatory efforts of defecation. There is a still stronger reason for this being the case when eventration exists. The same remark is applicable to hernia, which makes it to some extent a risk for the patient to strain energetically. In the latter case, the muscular power is intact, but the will restrains it from being called into operation. It is evident that practically a similar state of matters exists when the muscular efforts cannot be made without exciting violent pain: this occurs in rheumatism of the abdominal walls and diaphragm, and in painful affections of the abdomen: it occurs in those who suffer from piles, in those who have fissure of the anus, and whose bowels cannot be evacuated except at the cost of intolerable pain. These persons restrain the expulsatory action of the abdominal muscles, allowing the peristaltic action of the large intestine to accomplish nearly unaided the process of defecation. You perceive, gentlemen, how all these causes, like the will, act in producing constipation.

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* See p. 437 of this volume.

The diseases of the uterus and its annexes have a complex etiological action. If acute pain exist, as in metritis or in utero-pelvic phlegmon, the retention of the fecal matter is caused by a mechanism analogous to that which we have just been studying: the patient abstains from going regularly to stool because he is afraid of going, and ultimately he becomes habitually constipated. It is so likewise when there exists great prolapsus of the womb, which is always aggravated by the efforts of defecation, and which induces the woman to restrain the muscular efforts as much as she can. In extreme anteversion, and still more in retroversion, constipation is produced by a particular mechanism. The pressure exerted upon the rectum, which is flattened by being squeezed against the cavity of the sacrum, prevents the contents from escaping through the sphincter of O'Beirne, and the accumulation takes place in the horizontal portion of the sigmoid flexure of the colon. Now, in defecation, the colon acts with less power than the rectum, the contractile power of which is great: on the other hand, the matter accumulated in the colon does not provoke contraction of the rectum in the same way as matter immediately above the sphincter.

While great displacement of the uterus is a very effective agent in producing constipation, constipation will itself augment the displacement.

Reconsider, gentlemen, what I have now been saying. Suppose that the sigmoid flexure of the colon is filled with hard matter, and you will at once understand how it rests upon the floor formed by the anterior or posterior surface of the uterus, and how the muscular effort itself will augment the displacement and press the womb still more against the sacrum, and so render more formidable the obstacle to be overcome. Thus you see how it is that constipation is so obstinate and troublesome in women whose condition is such as I have now described. For them there is, in addition to the physical impediment, a sluggishness of the intestine, resulting from voluntary retention of the fæces, retention which is instinctively adopted by women to escape the pain and inconveniences which follow attempts to defecate.

The nature of the food and drink, and the sort of life which is led, have a remarkable effect in producing constipation: but in respect of these matters, the physician has no other guide than the idiosyncrasy of each individual. Speaking generally, however, we know that great abstemiousness, and a sedentary mode of life, are great predisposing causes. It is as unusual for great eaters as for persons who take a great deal of exercise to be constipated. It can also be shown that an exclusively animal diet predisposes to constipation, while the use of green vegetables and fruits produces rather an opposite tendency.

Gentlemen, the remarks which I have made upon the conditions which give rise to constipation ought already to have suggested to your minds some therapeutical notions. You must have already seen that, although in many cases the measures employed to counteract this infirmity are necessarily inefficacious, or at least only palliative, there are others by which immediate and durable success is obtained.

I have pointed out how constipation is produced by the habit of retaining the fæces; and in speaking of dyspepsia associated with constipation, I entered into some of the details of this subject.* It must be stated—for it is a fact—that when constipation is not constitutionally inherent in the individual (which as I have shown you is sometimes the case), *the will, patiently and regularly applied, will often triumph over this infirmity.* It is necessary that the individual go daily at the same hour to the water-closet. It is also necessary that for a considerable time he make powerful defecatory efforts: should these efforts be unsuccessful, he must wait till the following day, even though he should previously experience a desire to go. If on the second day, after a new attempt, there is no evacuation, a lavement must be taken immediately—not a lavement of tepid water, but, in the first instance, one of water with the chill off, and then one of cold water. On the following day, similar measures must be renewed, and repeated next day, should they have been unsuccessful—a second cold lavement must then be administered, should no stool have been obtained. The daily repetition of the attempt at the same hour to defecate, ends by causing a daily need to go to stool at that time: it seldom happens that persons who patiently pursue the manœuvres I have now described for eight or ten days do not ultimately obtain a daily motion.

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* See p. 401 of this volume.

There are, however, local adjuvants of some utility. I have spoken to you of the use of clysters of water with the chill off, followed by clysters of cold water. I have now to mention *suppositories*, which are more easily employed than clysters, particularly by men. In many cases, suppositories of cocoanut oil suffice: soap suppositories have a more sure and energetic action: and those made of honey hardened by heat are still more efficacious. A suppository of hardened honey ought to be in shape and volume nearly like a pigeon's egg. When it has been slightly moistened, it can be introduced into the rectum with great ease; and there are few cases in which it will not rapidly produce an evacuation. Quite understand, that I am decidedly opposed to the use both of clysters and suppositories, till energetic and fruitless efforts at defecation have been made on two successive days.

I must not, however, omit to mention, that the time of day at which the patient goes to the water-closet is a matter of considerable importance. The morning is certainly the most favorable time; one is then less in a bustle—on getting up one can devote more time to the water-closet than is possible during the course of the day. There are also other reasons of convenience in favor of the morning, which I cannot and do not wish here to set forth; but you can understand what they are.

It is worthy of remark, however, and the observation has been made by every one for himself, that immediately after a meal there is felt a somewhat urgent call to empty the large intestine. Perhaps the accumulation of food tends in a somewhat mechanical manner to expel the delayed residuum: or perhaps—and this explanation is the most reasonable—the renewal of digestion rouses throughout the whole alimentary canal a preparatory muscular action.

It is not only true that the best chosen time for going to stool is immediately after a repast, and particularly after the most ample meal of the day; but then also is the time when the local adjuvant means which I have pointed out are most advantageously employed.

It is evident, gentlemen, that irritant suppositories and even clysters cannot be employed without seriously disturbing digestion.

Before leaving the subject of clysters, I have still something to add. We have seen that a deficiency in the intestinal secretions has a great influence in producing constipation; and you can understand that by injecting into the rectum strongly mucilaginous fluids, such as infusion of linseed, decoction of marshmallows, or white of egg, the excrementitial bolus, and the mucous membrane of the intestine will both be lubricated, so as to allow the former to glide more easily over the latter.

When the use of irritant clysters is indicated, it is advisable, in the first instance, to try the emollient enemata of which I have been speaking.

Regimen is a matter of great importance. The surest plan to get the better of constipation is to make vegetable predominate over animal diet, to such an extent as the aptitudes of the stomach will permit. Herbaceous vegetables and fruits ought to take the first place in this regimen.

But, gentlemen, it is not easy to avoid going beyond or falling short of our aim. To produce diarrhœa is not to cure constipation, and is only substituting one disease for another. In respect of the vegetable diet, of the efficacy of which I have been speaking so favorably, it will only be useful if it be well borne.

Certain kinds of aliment derived from the animal kingdom, such as milk-food, have a slightly laxative effect upon some individuals. Milk-food, may, therefore, be given, whenever it has an aperient effect, without causing indigestion. Many persons find coffee with milk and others find tea powerful correctives of constipation.

Of drinks, beer and cider are most suited to persons of constipated habit. I may add, that I know many persons who, if they drink a tumbler of cold water in the morning fasting, are certain immediately afterwards to require to go to stool.

It would be difficult for me, gentlemen, to explain the mode of action of what is called bran-bread, a kind of bread made of three parts of flour, and one of coarse bran. I very often prescribe it: the patients eat it in place of ordinary bread, and in general the regular action of the bowels is greatly facilitated by its use.

We shall see immediately that belladonna is one of the most generally successful remedies for constipation: and you can thus at once see by recollecting the similarity of the properties of belladonna and tobacco how it is that many men cannot go to stool unless they smoke a pipe or a cigar immediately after a meal. Although, at least in our country, it is not considered very proper for women to smoke, I almost every week advise ladies to try the effect of smoking a tobacco cigarette, to aid in overcoming constipation which had proved inveterate under every hygienical treatment.

Following the example of Bretonneau, I constantly prescribe belladonna in constipation. I give it in the form of pills, each pill containing a centigramme of the extract, and as much of the powder of belladonna. One of these pills is taken daily, fasting, by preference in the morning on empty stomach, rather than in the evening. The number of pills may be increased from one daily to two daily within the first five or six days: they ought seldom to exceed four or five in the course of the twenty-four hours. Whatever number of pills are taken, they ought always to be taken at one time. I cannot tell you the manner in which they act. I can assert, however, that the majority of the patients who have perseveringly though fruitlessly followed the different counsels of which I have spoken to you, have at last obtained a satisfactory stool daily with the aid of belladonna. As soon as the stools become regular, the belladonna must be discontinued, and the organs be allowed to act without assistance.

Should the use of the belladonna prove unavailing, a teaspoonful of castor oil may be given with it: and to avoid disgusting the patient, this small dose of the oil may be administered in a gelatinous capsule. The intestine, prepared by the belladonna, yields to the purgative influence of the castor oil, which may be repeated twice a week if required. By and by, both the laxative medicine and the belladonna may be discontinued.

It is important not to go on using them too long, as that might lead to loss of appetite, and under insufficient alimentation, the constipation would return.

But, gentlemen, it will often happen that the constipation will resist the series of measures which I have pointed out. It then becomes necessary to have recourse to purgatives—extreme remedies, useful remedies, and indeed remedies which are indispensable, requiring, however, to be managed with certain precautions and with great prudence.

In general, the saline purgatives ought not to be employed. They have a rapid, almost instantaneous action, which, however, is not durable. After they have been used, the intestinal secretions, temporarily augmented, become dried up to some extent, just as the application of certain sapid salts to the mucous membrane of the mouth, after causing a profuse flow of saliva leaves a parched state of the mouth and thirst, proportionate to the intensity of the original effect.

For some years past, a remedy, which has taken the name of vegetable calomel, has been much used in England. I refer to *podophyllin*, the active principle of the *Podophyllum peltatum*. It is a very active medicine. I prescribe it in the form of pills, each pill containing one centigramme [$\frac{1}{7}$ of a grain] of podophyllin, and the same quantity of extract of belladonna. One, two, or three of these pills may be given, morning or evening, fasting. The result is one or two easy stools unattended by colic or heat in the stomach.

Generally, it is necessary to have recourse to the purgatives which are called *drastic*—particularly to aloes, extract of colocynth, gamboge, and extract of rhubarb. These are the substances which enter into the composition of all the purgative pills so much used by our English neighbors. I use the following formula:

Aloes, . . .	of each 1 gramme [15½ grains].
Extract of Colocynth,	
Extract of Rhubarb, .	
Gamboge, . . .	
Extract of Henbane, .	25 centigrammes [nearly 4 grains].
Essential Oil of Anise,	2 drops.

To be made into a mass, and divided into 20 pills, which are to be silvered.

The patient is to take, *always in one dose*, every second or third day, from one to three of these pills, the number of the pills for a dose being proportionate to the purgative effect produced. They ought to produce an easy and natural, or semi-diarrhœal evacuation.

The time at which these pills ought to be taken is not the same with all patients. Generally, it is best to give them at the beginning of the evening meal. In some persons, however, they produce a sort of indigestion, or act so quickly as to disturb sleep by causing evacuations during the night. When the pills act too rapidly, it is better to administer them to the patient in the morning fasting, or at the first meal in the morning. On the other hand, when they are slow in producing an effect, they should be given at the evening repast; or, if their administration at that time cause indigestion, let them be taken at bedtime, when the result will be a stool next morning.

You observe that I add henbane and oil of anise to the purgative ingredients of these pills. These additions, recommended by many English practitioners, are very useful: they prevent griping, and the henbane, in addition to its anodyne properties, exerts a beneficial influence, similar to that produced by belladonna and other active plants of the family solaneæ. Some prefer to give rhubarb in powder, immediately before dinner, in doses of from forty to sixty centigrammes [6 to 9 grains] and upwards. Whatever may be the influence of these purgatives, they ought never to be resorted to till the other means regarding which I have spoken at length have completely failed. The use of these pills is certainly less injurious than is generally supposed; and the abuse of them in England shows that we, on this side of the Channel, are inclined to exaggerate their evil effects. But it is not the less true, that regularity in obtaining relief from the bowels obtained by the observance of hygienical rules, by good and suitable food, and by habit, is always, in the end, preferable to that procured by artificial means.

Before leaving the subject of the treatment of constipation, let me refer to the application of *cold to the abdomen*, a minor method which I have seen recommended, and have myself prescribed, with astonishing success. On rising in the morning, let there be placed on the naked abdomen a compress of several folds soaked in cold water, and let it be separated from the clothes by a sheet of gutta-percha or caoutchouc. This compress ought to remain on for three or four hours.

LECTURE LXXVI.

FISSURE OF THE ANUS.

Treatment by Rhatany.—Constriction of the Sphincter of the Anus is the Effect and not the Cause of Fissure.—Fissure is very common in Women recently delivered: why it is so.—The Curative Effect of Rhatany depends on its modifying the character of the ulcerated surfaces, and tonifying the parts.—Its action ought to be promoted by Belladonna, which is a remedy for constipation.—When Rhatany fails, recourse must be had to a Surgical Operation; that which seems the best is Forcible Dilatation.

GENTLEMEN: That small portion of St. Bernard Ward which we reserve for nurses affords us frequent opportunities of observing an affection which, though apparently insignificant in respect of danger, is a source of great misery to the patient; I refer to fissure of the anus.

The history of this affection belongs rather to clinical surgery than to clinical medicine. I cannot, nevertheless, pass unnoticed the numerous cases which daily present themselves to your observation; and I feel the more bound to call your attention to them, that one of my plans of treatment which I have long used successfully is purely medical, if I may so express myself, and has not been sufficiently appreciated by surgeons. It consists in using clysters containing extract and tincture of rhatany.

The fissure or chap in the anus, which has been justly compared to cracked lips resulting from exposure to cold, consists of small, narrow, elongated ulcers situated between the stellate folds of the fundament. Women are much more subject to these fissures than men; and they are of far the most frequent occurrence in women who have been recently delivered. I shall tell you why it is so.

From the extreme frequency of this affection, it might be supposed that it has been known from very remote times: but my honorable colleague, Professor Velpeau, correctly wrote in 1838, that not more than twenty years had elapsed since fissure of the anus was first described as a distinct malady.

Boyer, who was the first to describe this affection in detail, advanced a theory in explanation of the mechanism of its production, and upon that theory based the plan of treatment which he adopted.

He rested his opinion upon the fact that he had never seen fissures unaccompanied by constriction of the sphincter of the anus, and that he had also several times observed all the symptoms characteristic of fissure without finding anything more than stricture; and also from the fact that section of the sphincter, even without touching the ulcers, immediately afforded relief, Boyer concluded that stricture is the sole cause of fissure. He said that the anal orifice is closed at the time of defecation by energetic spasmodic contraction of the anal orifice, and that the solid contents of the bowel, by forcing a passage against that impediment, cause tearing of the parts. Fissure in his opinion is only a complication or an accessory of the disease; and he thought that the only treatment required for the immediate cure of stricture and fissure was relaxation of the spasmodic stricture by section of the muscular fibres.

At the present time, a few surgeons hold Boyer's view, that the fissure is in itself of little importance, and that the preponderance of pathological importance belongs to the stricture; but generally, the only anxiety is to find means wherewith to modify the ulceration either by converting the wound, by an incision, into a simple wound, or by employing detergents, caustics, and the various ointments used in the treatment of obstinate ulcers in other situations.

The treatment which you see me employ is founded upon this principle. The chief efficacy of rhatany depends on the modifying influence which it exerts upon the morbid surfaces: but it possesses, in virtue of principles which it contains, the additional advantage of increasing the tonicity of the mucous membrane of the intestine and subjacent cellular network, which enables the parts to offer a more effectual resistance to the distending power of the excrementitial bolus, so that the solution of continuity not being torn afresh every day, is enabled to undergo natural cicatrization.

You perceive, gentlemen, that so far am I from being in dread of this constriction of the sphincter that I am not afraid to employ astringent medicines calculated to increase the stricture. The theory which I have formed from numerous cases to explain the treatment I recommend is, to my mind, quite satisfactory. The therapeutic method, however, is not mine; I got it from Bretonneau, to whom the healing art is indebted for so many means of cure.

Let us attend to the considerations upon which the illustrious physician of Tours founded the use of rhatany in the treatment of fissure of the anus, and how he was led to adopt it.

While on the one hand, constipation, and the expulsive effort, which, pressing the excrementitial bolus against the sphincter, distending and very often tearing it, are evidently in a number of cases the causes of fissure; on the other hand, constipation is the greatest obstacle in the way of a cure. Constipation is very often accompanied by a most remarkable structural change in the lower part of the rectum. Immediately above the sphincter, the rectum dilates into a sort of pouch, and again becomes contracted at the sacro-vertebral angle. In the pouch referred to, the fæces accumulate so as to form a very large ball; and every time the patient goes to stool, the expulsive effort is really like the straining of a parturient woman. In Bretonneau's opinion, whether the constipation is or is not associated with fissure, it is good practice to give to the lower portion of the intestine the tone in which it is deficient; and for this purpose rhatany seemed to him exceedingly well adapted. In cases of simple constipation, coincident with dilatation of the rectum, he was in the habit of giving lavements consisting of water holding in solution extract and alcoholic tincture of rhatany.

A lady treated by him had, along with the constipation of which I am speaking, fissure of the anus, which occasioned dreadful pain and seriously affected the health. He ordered her to have daily a quarter lavement of rhatany; and the result was a speedy cure both of constipation and fissure. Other patients presented themselves, suffering from constipation and affected likewise with spasmodic stricture of the rectum and fissure. The same treatment which was pursued in the case of the lady cured all these morbid conditions in them. He then thought that notwithstanding the existence of constipation, a symptom absent in some cases of fissure, rhatany might be employed, and the trial was crowned with success similar to that obtained in the other cases. By means of very legitimate induction, he accomplished the first step, and then facts to which he did not appeal awakened his attention: by simply verifying them, and pursuing a course of rational experiment, he was led to adopt a treatment which perhaps may not be "rational," but which is exceedingly good; and that is the principal consideration.

In point of fact, gentlemen, this plan of treatment would be truly rational, if in accordance with Bretonneau's view, constipation was always the cause or always a complication of fissure. But not unfrequently we meet with diarrhœa or soft stools in patients suffering from fissure of the anus: and we also find fissure in those who take clysters morning and evening to prevent pressure against the sphincter in defecation. When the state of constipation is very great, rhatany is by itself inadequate to accomplish a cure; and it then becomes necessary to aid its action by administering a laxative, so as to prevent tearing, by facilitating the passage of the fæces through the anal orifice. Bretonneau's treatment always gave exceedingly good results in his own hands, and in mine, as well as in those of all practitioners who have tried it with perseverance and in accordance with the indications given by its author.

Before giving you precise rules in respect of this practice, allow me to state what I believe to be the mechanism of the production of fissure of the anus.

There are some cases in which we cannot ascertain the starting-point; but we know that it may be occasioned by whatever excoriates or superficially tears the anus, such as the point of a syringe unskilfully introduced, sodomy, or other causes. We also know that hæmorrhoids and constipation are its two most common causes; and that they act with greater certainty when they find the parts in a special state, as in women recently delivered, who, as said before, are the persons most liable to suffer from this affection.

The pressure which in the latter stage of pregnancy, is made upon the parts within the pelvis, by the greatly enlarged uterus—particularly upon the lower portion of the intestine, in which it embarrasses the circulation—keeps up a constant state of congestion, and when in an extreme degree resulting in hæmorrhoids. If to this it be added that constipation is also a usual accompaniment of the latter period of pregnancy, you will perceive that women in that condition are peculiarly predisposed to fissure of the anus, because the different causes now pointed out are liable to coexist in them.

During labor, at that stage when the fœtus has descended to the floor of the pelvis, and presents at the vulva, the perineum is forced forwards by the expulsive efforts of the woman: the skin in the neighboring parts is chafed, and this chafing, which extends to the anus, may be such as to amount to little tears or abrasions of the mucous membrane, which will ultimately constitute fissures.

These minute solutions of continuity will have all the more chance of being converted into ulcerations by the lochial discharge flowing along the commissure of the vagina to the anus, and by the irritation so induced preventing cicatrization. I need scarcely add that this occurs most frequently in women who neglect scrupulous attention to personal cleanliness.

The lochial discharge itself may become a predisposing cause of fissure; the irritation which it induces and keeps up at the anus impresses on the tissues a peculiar modality, in consequence of which they tear and fret more readily when subjected to pressure by the passage of hard feculent masses.

The essentially characteristic phenomenon of fissure of the anus is a violent pain, which the sufferer compares to the pain caused by a tear or a burn, or, to use a comparison often employed by patients, to the sensation of a flame of fire passing over the affected parts. This peculiar pain is excited by defecation; and continues after the bowels have been evacuated for a period varying from some hours to a whole day or a whole night. So severe is the pain, that the suffering women, afraid of renewing it, dreading its return more than words can express, shrink so exceedingly from going to stool, that for eight, ten, twelve days or longer, they abstain from any attempt to evacuate the bowels. The constipation increases, the fæces become harder; and consequently the pains become more and more violent till the feculent matter is expelled. In many cases, the pain subsides a few minutes after defecation, to return with augmented severity, and in a few hours to assume a frightful intensity.

In some cases, the fissure is smeared with blood, which forms red streaks upon the excrementitial bolus: generally, however, there is very little oozing of blood from the ulcerated surface. It is a remarkable fact, that these pains are not only occasioned by the passage of hard masses: they are sometimes induced by soft, even by liquid motions, as has already been stated.

Upon making a digital examination of the anus, the sphincter contracts energetically; and if a forcible attempt be made to overcome the obstacle, the examination causes the patient greater suffering.

The best method of discovering the seat of the disease is to request the patient to bear down as if at stool: the anus then becomes prominent, and upon separating its folds, we are able to perceive a small ulceration at the bottom of the separating furrows: the ulcerated surface has a bright red appearance, which may be very well compared to chaps produced in the hands and lips by cold.

Many patients conceal from their medical advisers the existence of these fissures, in spite of the dreadful pain to which they give rise: you have often seen, in our wards, that after we have long remained ignorant that certain patients had fissures, the fact was at last discovered merely by chance. Some patients complain of having piles: when this complaint is made by women recently delivered, it is in itself sufficient to lead to the suspicion that there is fissure; and a more minute investigation will often show such to be the case.

When matters have advanced to a certain point, the obstinacy and intensity of the symptoms produce a prejudicial influence on the general health. The habit acquired by patients of restraining their desire to go to stool increases constipation, and so leads to dyspepsia: digestion ceases to be performed with regularity, and dyspepsia becomes all the more urgent that patients abstain from eating that they may not require to go to the closet.

However unimportant, therefore, fissure of the anus may be in itself, it may lead to serious consequences: even when the phenomena are only local, it is necessary to use measures to save the patients from excruciating pains. To modify the ulcerated surface is the object, and to attain it, we employ a variety of topical means which I need not here enumerate. There is one among them, however, to which I must call attention: I refer to the mode of treatment recently recommended by Dr. Chapelle (of Angoulême).

Dr. Chapelle's treatment consists in the introduction into the anus of a pledget soaked in a mixture of chloroform and alcohol in the proportion of ten parts (by weight) of the former to five of the latter. Dr. Chapelle has taken care to give warning that this proceeding at first causes acute pain; but he says that after the third application, sometimes even after the second, a cure is obtained. Having been nominated by the Academy of Medicine as a member of the committee appointed to report on this treatment, I tried it in several cases in our clinical wards. No untoward results occurred. However, the small amount of success which attended my trials caused me to resume the rhatany plan, which I find the best.

The following is my mode of using rhatany. To empty the intestine, I cause to be administered every morning a clyster, infusion of bran or marshmallow. Half an hour after this has been returned, a quarter clyster is given of which the following is the composition:

Water, ...	150 grammes [5¼ fluid oz.].
Extract of Rhatany, . .	4 grammes [61½ grains].
Tincture of Rhatany, .	4 grammes [61½ grains].

The patients ought not to retain the lavement more than a few minutes: it should be repeated in the evening. Sometimes, when the fissure is so situated as to protrude entirely when defecatory efforts are made, the desired object can be very well attained by using lotions of extract of rhatany.

Should the fissure be very deepseated and rebellious to treatment, the clyster must be administered with a syringe of continuous jet: the patient resists the injection, which is thrown back into the basin and taken up again by the pump, and may serve for an ablution which might be almost endless, and which ought to last for from two to four minutes or longer.

But it very often happens, that constipation which in a great measure was the cause of the malady is an invincible obstacle to its cure. Day by day, the hard and bulky excrementitial bolus tears the wound, and destroys the incipient cicatrization obtained by the rhatany. It then becomes necessary, during the whole course of the treatment, and even after the cure, to give daily a mild laxative, so as to keep the bowels freely open, and render the fæces less hard. In relation to this point, let me beg you to bear in mind the remarks which I recently made on the treatment of constipation.

It often happens, that during the first days of the treatment, the pains are greatly aggravated, to the great discouragement both of physicians and patients. The causes of this aggravation are easily understood. Persons who, from the incipient stage of the fissure, had got into the habit of going seldom to stool, to avoid the dreadful pain occasioned by defecation, now go several times during the course of one day: the result of this change is pain lasting continuously for perhaps several days. Such cases are fortunately very rare, but they do sometimes occur, rendering it necessary to administer during the first days of the treatment only one in place of two rhatany clysters, and to abstain from giving laxatives till the intestine has become less sensitive.

As soon as the pains are entirely subdued, one rhatany clyster a day is sufficient; and when there is reason to believe that the cure is complete, it will be necessary to continue giving, every two days for at least two or three weeks.

Persevering in the use of the remedy, even after its continuance may seem superfluous, is of great importance; for if it be abruptly discontinued, there will be a risk of a recurrence of the symptoms.

Thanks to this plan of treatment, I have been able to heal very painful fissures, and deep fissures with callous edges. The cure, it is true, proceeded slowly; and as an example of this, I may mention the case of a lady who, after refusing to submit to a surgical operation, was cured, contrary to all expectation, by pursuing the rhatany treatment for more than a year. This was certainly paying dearly for the cure; and in similar circumstances, I should certainly recommend an operation, which may either be paring the edges of the fissure—a proceeding which perhaps acts beneficially like the rhatany plan only by modifying the morbid surfaces—or forcible dilatation, to which more than once you have seen me have recourse.

It must, however, be admitted, that the rhatany treatment, particularly if prolonged, may be rather too costly. In consideration of this, I have endeavored to substitute for rhatany, sulphate of copper, a remedy, the price of which is so small as to be within the means of even the poorest. I order a half-lavement, containing 15 centigrammes [$2\frac{1}{2}$ grains] of this salt to be given morning and evening. You have seen the effects of this medication. Our night-nurse, a robust young woman, recently confined, had a very painful fissure from which she had been suffering for eleven days: her condition had been much ameliorated, and the fissure nearly cured by the use of rhatany lavements for ten days, when six days later, it was torn anew during defecation. She lost a great deal of blood, and experienced very acute pain during the whole of the night which followed this occurrence. Next day, March 17th, she had her first lavement of sulphate of copper, which was returned, a sensation being at the same time produced as of a hot iron traversing the anus. Two days later, she no longer had pain when the clyster returned, and she did not suffer for more than half an hour in the night from having had a stool. For three days, from the 21st to the 24th, she was absolutely free from pain on going to stool. But on the 24th, the fissure was again torn during defecation, and in consequence the patient suffered for two hours and a half. After using the sulphate of copper for three days, the pains had again entirely ceased: the patient only suffered slightly when the motions were hard. The sulphate of copper clysters were continued till the 15th April, that is to say for a month; and at the end of that period, the young woman passed hard stools painlessly. The cure was definitive.

You may have observed that since this case occurred, I have been using indiscriminately rhatany or sulphate of copper in the treatment of fissures of the anus; and that I substitute the one for the other when at the end of eight days, the amelioration (always obtained) remains stationary. By proceeding in this way, I always accomplish a cure. Sometimes, the cure takes place under the exclusive use either of rhatany or sulphate of copper, and in other cases, sulphate of copper completes a cure begun by rhatany, or rhatany completes the cure begun by sulphate of copper.

I now resume the subject of dilatation. I do not refer to the introduction of gradually increased pledgets of charpie, a practice which some adopt; I speak of sudden forcible dilatation performed by the introduction of the fingers. This operation would be extremely painful, were it not that we can perform it whilst the patient is in a state of anæsthesia from chloroform—a wonderful means of preventing pain.

I have still to mention some accessory means, which occasionally are in themselves sufficient to accomplish a cure. I order a sort of porridgy mixture to be prepared, consisting of one part of subnitrate of bismuth and five parts of glycerin; and with this I direct the patient to anoint the anal orifice five or six times a day, and to apply it to the ulcerated surfaces, taking care that whilst the application is being made, the folds of the anus are opened. Pomades of white precipitate and red precipitate *au trentième*, if very carefully employed, will produce equally good results. I also order the parts to be bathed morning and evening with very hot water, to which is added a sixth or an eighth of *eau phagédènique;*[*] or I order five grammes [77 grains] of corrosive sublimate to be dissolved in two hundred grammes [7 fluid oz.] of water: a teaspoonful of this solution is added to a litre or half a litre of very hot water, with which the parts are to be washed for some minutes morning and evening. Cauterizations with the solid nitrate of silver and sulphate of copper, though very painful, are also sometimes very useful.

Gentlemen, I cannot conclude my remarks on this subject without telling you, that careful ablutions three times a day with simple water—that is to say, minute attention to cleanliness—will sometimes supersede the necessity of any medication.

LECTURE LXXVII.

INTESTINAL OCCLUSIONS.

Their Causes.—Their Mechanism.—Their extreme Gravity.—Treatment by medical men.—Gastrotomy may be resorted to in serious cases.

Gentlemen: Last Tuesday you saw my honorable colleague, Prof. Jobert (of Lamballe), operate here, in accordance with my pressing solicitation, upon a man who came into our clinical wards with all the symptoms of intestinal strangulation. The patient died thirty-six hours after the operation. It is my duty to explain to you the reasons for my urgency with M. Jobert, who was opposed to the performance of gastrotomy: it is my duty to render you an account of this matter, because, notwithstanding the issue of the operation, I have still the complete conviction that it was right to have recourse to it.

The patient, was a man about fifty years of age. He stated, that for many years he had had bleeding piles, that he often passed blood and pus at stool; and that he was subject to alternations of constipation and diarrhœa. With the exception of these symptoms, his health was not bad. He had had no motion for thirteen days; and for ten or eleven days, he had had vomiting. The matter vomited, which at first was food, had become bilious. We found, in fact, in the spittoon and basin left beside the patient, not only bilious matters, but matters resembling those generally found in the lower part of the small intestine, and which in such cases are improperly called stercoraceous. The abdomen was very tympanitic, but painful only in a moderate degree. The countenance, however, was expressive of very acute pain, and the most distressing anxiety. In fact, there existed all the characteristic symptoms of strangulated hernia. My inquiries were first of all directed by that view of the case; and I searched in the groin and the fold of the thigh for a tumor. None, however, was found.

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[*] *Vide* vol. i, page 441.

The information derived from the patient's verbal statements evidently referred to an intestinal lesion; and with the greatest care I investigated into the possible seat of the malady. The hæmorrhoids of which he had spoken led me to ask myself whether there did not exist some affection of the large intestine causing an obstacle to the passage of the fæces. Both M. Jobert and I made a digital examination of the rectum, but discovered nothing as far as we could reach with the finger. Hence we concluded, that the obstruction existed higher up, as high up at least as the sigmoid flexure of the colon; that at all events it was beyond the reach of the finger. Exploration through the abdominal parietes was impossible from the enormous tympanitic distension. Whatever was the cause, whatever was the seat of the occlusion, the nature of the phenomena admitted of no doubt: the severity of the symptoms, the extreme anxiety, the smallness of the pulse indicated that danger was imminent, and that death must occur within twenty-four hours.

Too much time had already been lost to wait still longer for the doubtful results of the different measures proposed in similar cases; to count on puncturing the intestines distended with gas seemed to me equivalent to abandoning the man's life to chance; while gastrotomy, twice successful in my private practice, appeared to offer the only hope. Such were the circumstances under which I besought M. Jobert to operate. Along with me, he fully realized the gravity of the situation: he knew that though in itself gastrotomy was not more dangerous than herniotomy, it was in this particular case a much less hopeful undertaking than the operation for strangulated hernia is in ordinary circumstances.

Considering that after a too long delayed operation for strangulated hernia, it is not unusual to see the patient sink, M. Jobert was not unmindful of the serious consequences which we had to dread. Still, he yielded to my entreaties, perceiving very distinctly that it was only by his surgical intervention that a feeble and sole remaining chance of saving the patient was afforded.

Gastrotomy, therefore, was decided upon. It was proposed by this operation to form an artificial anus in the right iliac region, whereby to afford an issue to the matter impacted in the intestine; or, in other words, to afford immediate relief from the effects produced by the obstruction.

When the opening was made, a large quantity of gas and liquid escaped. The edges of the intestinal were united to the edges of the abdominal incision by some sutures, and a sound was introduced into the upper end of the intestine so as to secure a passage for the feculent matter. Notwithstanding these measures, there was only a partial diminution in the tympanites, and to a certain extent, the other complications also continued. During the night, the patient was attacked by a sort of choleraic diarrhœa; and a large quantity of a yellowish-white fluid passed through the gum tube left in the bowel. The patient had vomiting. There was an increase in the severity of the abdominal pains. The pulse became more frequent, the skin grew cold; and death occurred during the following day.

At the autopsy, we saw that the bistoury had opened one of the lower folds of the small intestine; and that that had occurred which is usual when the incision is made in the neighborhood of the cæcum—the lower end of the ileum was the part struck by the knife. This is an exceedingly important fact; for if the opening be made higher up, there will not be so long a portion of intestine between the stomach and the artificial anus for the performance of the digestive functions, and there will be a risk of the patient's dying from inanition.

The lips of the wound made in the intestine were perfectly adherent to the lips of the wound in the abdominal parietes. Notwithstanding the shortness of the time which had elapsed, the inflammation of the peritoneum, apparently dating back forty-eight or seventy-two hours, had promoted this rapid union, which had the patient lived, would in a few days have been perfect, leaving nothing to fear from the flow of matters from the digestive canal into the peritoneal cavity.

The cause of the obstruction was situated in the sigmoid flexure of the colon, which was enormously distended by gas, and seemed to be five times its usual length. It was folded upon itself in such a manner that its left curvature was pushed to the right and its right curvature to the left, while the inverted mesocolon formed a band which still more tightened the obstruction.

The small intestine was collapsed, free from fluids, which had probably escaped by the artificial orifice made by the surgeon. We also perceived another lesion; the free margin of the omentum was inserted in the ileocæcal appendix by an elongated false membrane, which dipping into the pelvis, tense, and fixed at both ends, formed a sort of bridge. As we shall see, symptoms similar to those experienced by our patient, are often occasioned by bands of this description entangling and strangling the intestine.

In the case now before us, however, this band had no share in producing what we observed, for on opening the abdomen with the utmost possible precaution, we were satisfied that the band did not grasp any loop of intestine.

After the death of the patient, when we had the parts before us, it was easy to perceive how, in this case, the opening made into the intestine might have proved of use. The result, for example, might have been to liberate gases enormously distending the sigmoid flexure of the colon, and to restore the contractility of its muscular tunic paralyzed by the distension resulting from the gaseous accumulation. It might likewise have been the means of restoring energetic peristaltic movements, by which the intestine might be brought back to its proper situation.

The remarks which I have to make to you to-day on the subject of *intestinal occlusion* will enable you fully to understand my views. In general, gentlemen, it is impossible to bestow too much care in ascertaining with the utmost possible precision the seat of intestinal obstructions. You will without difficulty understand—and of this the case which I have just related is a proof—that it is very important to discover whether the stoppage is in the large or small intestine. In the adult, it occurs in the great majority of cases in the small intestine, a fact which explains the success which usually attends the operation for artificial anus when the incision is made in the small intestine. The intestine quickly discharges the matters which had accumulated above the occlusion.

But in cases in which the bowel is opened far above the obstruction, the probability is that the operation will prove useless. This happened in the case which is the subject of this lecture. The obstruction was in the sigmoid flexure of the colon, and—the operation having been performed in the right iliac fossa, and the artificial anus made in the small intestine—the unfavorable state continued, and led to the death of the patient.

If Professor Jobert and I could have discovered beforehand the situation of the obstruction, we should not have hesitated to prefer Littre's method; or in other words, we should have made the artificial anus in the sigmoid flexure itself, and then probably we should have saved the patient.

Ileus, volvulus, iliac passion, and vomitus stercoris, were the names formerly given to the malady characterized by a complete cessation of alvine evacuations, accompanied by violent, incessant, and intractable vomiting, by tympanitic distension of the abdomen, acute pain—symptoms almost invariably leading to a fatal issue when the patient has been left without treatment, and often even when the most active measures have been resorted to. This disease was formerly considered as an affection essentially spasmodic. Even during last century, however, pathological anatomy had enlightened physicians as to its real nature: it had shown them the great similarity between ileus and strangulated hernia: it had shown them that the supposed spasm was something more material, and that the dreaded symptoms depended upon a structural obstacle in the passage. In our day, *internal strangulation* is the term substituted for the names I have just mentioned; and it more accurately describes the condition which generally produces the malady. Dr. Oscar Masson, a distinguished interne of our hospitals, has proposed to use in place of intestinal *strangulation*, the term intestinal *occlusion*, which has the advantage of being applicable to all cases in which there is accidental obliteration of the intestine, and also to all the varieties of hernia, the most common causes of occlusion;* but regarding which it is not incumbent on me to address you: here, we have to do only with occlusions originating within the abdominal cavity.

These occlusions, according to their causes, present numerous varieties.

The *causes* are themselves very numerous: some, independent of the intestine and developed external to it, act in such a way as to compress the bowel and diminish its calibre: others originate in the intestine itself, either in its cavity or in the substance of its tunics.

Among causes belonging to the first class, I first of all mention abdominal *tumors* when they acquire a certain bulk, and occupy a certain relative position to the intestines. The cause, for example, may be tuberculosis or cancer of the mesenteric glands, or a phlegmonous tumor in the iliac fossa; or, it may be the displacement or augmented volume of organs, as of the uterus or spleen in the cases to which Dr. Masson refers in his thesis.

The most common of the first class of causes, the causes external to the intestine, are those which induce internal strangulation properly so called. Such are adhesions formed between different organs by morbid exudations; and such likewise are the *pseudo-membranous bands* formed in the abdominal cavity by inflammatory action, generally by inflammation of a latent character.

Let us suppose that, consecutive to an attack of peritonitis, the ileo-cæcal appendix had contracted adhesions with another portion of the intestine, with the ovary, Fallopian tube, or broad ligament: let us suppose that these adhesions are formed between other parts of the intestine: the result will then be the formation of sorts of bridges under which a loop of intestine might easily become entangled. The same will occur in respect of pseudo-membranous bands, of which the number, extent, seat, and disposition, admits of infinite variety. In some cases these bridges are large enough to allow an intestinal convolution which has got under it to extricate itself. In other cases a convolution a little too large may be seized: it will at first be only slightly constricted: and were not the tube a living organ, the contained matters would pass onwards, though not so freely, but the tube being possessed of life, vital changes take place in it which in their turn produce obstruction. The embarrassed capillary circulation leads to the engorgement of parts, and consecutively a thickening of the intestinal parietes, the calibre of which diminishes on this account, and diminishes all the more that the muscular contractility is in its turn greatly increased under the influence of irritation. The intestinal fluids and gases, interrupted in their course, accumulate in the canal which contains them; the canal then becomes partially obstructed, and at last completely shut up.

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* Masson (Oscar): Thèse soutenue devant la Faculté de Médecine de Paris le 2 Mars, 1857.

In other cases internal strangulation arises from a very remarkable mechanism: the ileo-cæcal appendix twists round a convolution of intestine in a knot, which is sometimes double. The intestinal diverticula may also become a cause of internal strangulation; but, to enable this to take place, they must be some centimetres in length, and inflammation must have produced adhesion of their free extremity: thus it is, as in the case of the ileo-cæcal appendix, that the diverticula can embrace and strangle an intestinal convolution.

Internal strangulation may also be caused by a sort of hernia in a natural opening, such as the foramen of Winslow, or in an accidental opening, such as a chink in the diaphragm, or a tear in the mesentery or epiploon.

Occlusions originating in the intestine itself are also equally various in their nature. First in importance are organic lesions, particularly *cancerous affections*, and most frequently cancerous affections of the large intestine, which, as they advance, cause strictures and more or less complete obliteration of the canal. I may also mention syphilitic strictures, strictures resulting from thickening of the intestinal parietes succeeding chronic inflammation, strictures produced by extravasation of blood between the tunics of the intestine, such as Bretonneau speaks of having observed, and finally strictures determined by adhesions formed between the cicatrices of ulcers, by vegetations and by polypi.

Very severe symptoms of intestinal occlusion are sometimes induced by the accumulation of stercoral matter. You have certainly, more than once, in the hospitals, met with persons—generally women—having very large tumors of this description in the transverse and descending portions of the colon. These tumors, formed by masses of fecal matter, change their position or entirely disappear under the influence of a somewhat energetic purgative; but in other cases they give rise, I repeat, to the symptoms characteristic of intestinal occlusion. That which we so frequently observe in the rectum in cases of habitual and invincible constipation may also occur in the upper portion of the large intestine. Though this kind of obstruction is generally easily got rid of, it sometimes resists all our efforts; and cases are recorded in which it has caused death.

Undigested food may give rise to similar accidents. At an early stage of my medical studies, I saw a soldier who was seized with all the symptoms of intestinal occlusion fifteen or twenty days after gluttonously swallowing some pounds of cherries with their stones. He died: and on opening his body, we found near the termination of the small intestine, at the ileo-cæcal valve, a mass of cherry-stones almost half as large as the fist, completely obstructing the intestine.

Obliterations of the intestinal canal are likewise produced by foreign bodies accidentally swallowed, such as glass and ivory balls. Cases, too, are recorded in which similar results have followed biliary calculi passing into, and being arrested in, the digestive canal.

Of all the causes of intestinal obstruction, perhaps the most curious is the presence of lumbricoid ascarides. I saw a case of this kind in a woman, who died with all the symptoms of occlusion. At the autopsy, we found an enormous packet of these worms twisted one on the other, so as completely to obstruct the intestine.

Gentlemen, to conclude this enumeration of the mechanical causes of intestinal occlusion, I have still to speak of invagination, of volvulus, and finally of retroversion of the intestine, an example of which was presented by our patient.

You know the meaning of the term *invagination*. A portion of intestine becomes introduced within the portion below it in such a manner that the serous and mucous tunics are in apposition. The result of this intussusception or inclusion of one part of the canal within the other is necessarily a diminution of the calibre of the canal. You can understand how intestinal occlusion may be the consequence of this invagination; it is not, however, an inevitable consequence, for a vigorous contraction of the intestine may suffice to re-establish the normal condition. Unfortunately, however, it is not so always: by the persistence of the invagination, inflammation may arise, and glue together the two peritoneal surfaces which are in apposition. The inflammatory action being necessarily preceded and accompanied by engorgement of the tissues which it attacks, the already narrowed canal becomes still more obstructed, its calibre at last becomes entirely obliterated, and very soon the symptoms of intestinal occlusion show themselves. However, even in such cases, the issue is not so inevitably mortal as when the occlusion is dependent upon some other causes which I have mentioned. The occlusion may disappear in a way which I shall now explain. The inflammation of the invaginated portions may proceed to sloughing, and thus they may become detached from the living portions, fall into the intestinal passage, and be evacuated by stool. A reparatory process may at the same time organize a union of the two ends in mutual contact, and although the intestine remain somewhat contracted, the contraction is not sufficient to completely prevent the passage of the fæces. After a longer or shorter period, the cure is complete. Of this I have seen two examples.

Volvulus consists in the rolling or twisting of the intestines: and the case which has given rise to the present lecture may be considered as of this description. In this case, the *intestine was retroverted upon itself*. The sigmoid flexure, retained by a duplicature of peritoneum more than usually lax, and consequently more mobile, was retroverted, as you saw, in such a manner that the right curvature was placed on the left side, so as to form in the canal a fold by which the passage was obliterated.

As to the different kinds of ileus, of *miserere mei*, or *miserere* colic, they are generally attributable to the seat of the occlusion, and to the excruciating pains sometimes experienced by the patients.

Whatever may be the *causes* of intestinal occlusion, its *symptoms* are always those of strangulated hernia. For some days, the patients are without movement of the bowels, while at the same time they experience a dull pain in a limited part of the abdomen. This pain, caused by the retention of the contents of the bowel, increases in intensity and superficial extent: the intestinal convolutions become distended with gas: and then the patient has nausea, with vomiting of matters variable in their character. At this stage, the intestine has so great a tendency to peristaltic action, that the ingestion of fluids by the stomach, or pressure on the abdomen, is sufficient to cause vomiting, first of the fluid contents of the stomach, then of bilious and chylous matters pent up in the lower portion of the small intestine. *Fecal* matter, properly so called, that is to say, matter contained in the large intestine, cannot be rejected by the mouth, even when the obstruction is seated in the cæcum, the colon, or the sigmoid flexure, because the ileo-cæcal valve, if it has retained its normal structural relations, constitutes an insurmountable barrier to the passage of matters from the large to the small intestine.

But in these cases in which the sigmoid flexure of the colon is the seat of the occlusion, there is, from the very first, a great degree of meteorismus in the iliac, epigastric, and hypogastric regions; whereas, if the occlusion be situated in the small intestine, the meteorismus, for a certain time, does not extend beyond the umbilical region.

M. Laugier has in a special manner insisted upon the importance of ascertaining the locality of the seat of the meteorismus in the diagnosis of strangulated intestinal hernia.*

It is difficult to determine whether the obstruction be seated in the large or small intestine; but it is more than difficult—it is nearly impossible—to determine from the symptoms whether it be in the duodenum, jejunum, or ileum.

Whatever may be the seat of the internal strangulation, there is very soon inflammation of the intestine and peritoneum; and from the first, the symptoms sometimes derive a very marked severity of character from the peritonitis. The vomiting then becomes more frequent, the abdominal pain more general, and the meteorismus extends to the whole abdominal cavity. The pulse becomes very quick and very small: the skin becomes covered with viscid sweat; the very altered expression of the countenance is expressive of suffering; the eyes are sunken: the nose is pinched: the lips are bloodless: and the tongue is cold. The existence of peritonitis renders surgical interference useless. A state of excitement is followed by profound prostration, and death occurs without the intellectual faculties having become impaired. In some cases, so great is the prostration at the last, that the patients cease to utter any complaint.

When the physician sees the case at its commencement, or when he has had a precise account of the symptoms, there is hardly any risk of his committing a mistake: in point of fact, the total absence of stools, the pain localized in the first instance in the abdomen, then the frequency and persistent vomiting, unaccompanied by yellowness of the skin or conjunctiva, exclude the supposition that the case is either hepatic colic or simple peritonitis. But, on the other hand, if the history of the symptoms is imperfectly given, and if the physician examine the case for the first time, when the intestinal obstruction has become complicated with peritonitis, it is evident that there will be room for doubt as to the nature of the case; or a mistake in diagnosis may remain uncontradicted, till the absence of stools is a certainty. Thus, the symptoms of peritonitis from perforation have been seen to simulate the symptoms of intestinal occlusion. This occurred in a case recently treated in one of the wards of this hospital.

The *prognosis* is generally exceedingly unfavorable.

When the occlusion is produced by an accumulation of matter in the intestine, we may hope that energetic purgatives will cause the symptoms to disappear by inducing vigorous contractions of the intestine; but when it depends upon tumors situated in the walls of the intestine, we must admit our inability to cure, because these tumors, like cancerous tumors, are incurable.

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* Laugier: Sur un Signe Nouveau dans l'histoire des Hernies Etranglées, à l'aide du quel on peut reconnaître si l'intestin est compris dans le sac herniaire et à quelle portion du canal intestinal appartient l'anse étranglée. [*Comptes Rendus des Séances de l'Académie des Sciences*, 1840; t. x, p. 870.]

In strangulation by adventitious bands as well as in cases of invagination, of volvulus, and of inversion of the intestine, our intervention, however energetic, is generally of no avail: yet it is sometimes crowned with success.

Let us now consider the means of *treatment* which are available.

In strangulated hernia, the *treatment* which is first indicated is to employ the taxis; but, as can be readily understood, this operation is inapplicable in internal occlusion, even when we know the seat and cause of the obstruction, inasmuch as we cannot reach it by direct means. However, *malaxation* may to a certain extent serve as a substitute: it will induce peristaltic movements of the intestine, the tendency of which will be to restore the parts to their normal position. In performing malaxation very great prudence is required; and the older the date of the malady, the greater must be the moderation employed.

The application of a very large cupping-glass, or of several smaller ones, to the abdomen, has also been recommended. A bellows movement ought to be made with the cupping-glasses when they have been fixed on the abdominal parietes. The traction thus exerted is said to destroy the bridles of a false membrane and rectify the invaginations, so as to relieve the intestinal obstruction.

Leroy (d'Etiolles) proposed to excite the peristaltic movements by electricity, by establishing a galvanic current between the mouth and anus. To accomplish the same object, Dr. Duchenne (de Boulogne) has, in three cases, employed faradization; and in one of them only was the result a cure. Electricity, in fact, does not seem to me to be of any real use, whatever mode of applying it may be adopted.

Causing the patients to swallow mercury and balls of lead are modes of treatment which I merely mention to remark that they were formerly lauded and are now forgotten.

The administration of purgatives constitutes the chief means of treating intestinal occlusion. They act on the same principle as malaxation, that is to say, by causing peristaltic motion; but their action is much more energetic. Senna and the other purgatives which specially influence the muscular contractility of the intestine have the preference. For reasons which I do not require to state, these medicines ought to be administered in clysters.

In the work which I have quoted, Dr. Masson proposes to call the attention of physicians to the treatment of intra-abdominal intestinal occlusion by the external application of ice; but the number of cases which he adduces to show the utility of this measure is too small to justify satisfactory conclusions being drawn from them.

Notwithstanding the use of the different means which I have enumerated, the symptoms generally continue, and it becomes necessary at last to resort to a surgical operation.

The simplest operation is *puncture of the abdomen*, a proceeding indicated in cases in which there exists tympanites. The great accumulation of gas in the intestine, by producing excessive distension, paralyzes the contractility of the muscular tunic; for the same thing occurs in respect of the intestine, as in the bladder and all hollow organs. The paralyzed state is increased by inflammation of the parts. Such of you as have been present at operations for strangulated hernia may have seen, that the portion of intestine which is strangulated does not contract when pricked by the point of the bistoury, though in a normal state it would have contracted energetically if so treated.

The effect of puncturing the abdomen is to liberate confined gas; and to restore contractility by putting an end to the extreme distension. The punctures are made by inserting small exploratory trocars at the places where the distension seems greatest. Should the first puncture be insufficient, a second or third, or even as many as eight or ten, may be made. This little operation is neither painful nor dangerous. If you read the inaugural thesis of my colleague in the hospitals, Dr. A. Labric, you will be convinced that the punctures involve no danger, and that they may be productive of benefit—as they probably would have been in the case of our patient in St. Agnes Ward had they been made at an ealier period.*

After the distension has been got rid of, purgatives are useful to renew the contractility of the intestine.

The punctures must be performed at an early stage: the longer the delay in resorting to them, the less are the chances of success.

When all curative measures, including puncture, have failed, and the symptoms of occlusion continue—when the disease is of eight or ten days' duration, and there is either no diminution or a rapid return of the distension, with frequent profuse stercoraceous vomiting, and feeble pulse—when—to sum up in one word the state of matters—when there is imminent danger to life—there is only left the grave, the extreme resource of *gastrotomy.*

It is only within the last few years that this operation has been accepted as admissible in cases of occlusion.

As soon as it became an ascertained fact, that, in numerous cases, occlusion is occasioned by bands of false membrane, by a retroversion upon itself of a portion of the intestinal tube, by its invagination, it was thought, that by opening the abdomen, there would be a possibility of disentangling the intestines, and of so removing the obstacle to free passage through them. But the questions arose: Upon what grounds can so perilous an operation be justified? By what indications is the surgeon to be guided in performing it?

Numerous cases of accidental extensive injuries of the abdominal parietes by cutting instruments, and bull horns, had shown that penetrating wounds of the abdomen were not so dangerous as had been believed, as there were instances of complete recovery, even when the intestines had protruded from the abdominal cavity. It was concluded, that gastrotomy, if performed according to rational surgical rules, need not be fatal, since it was not necessarily fatal in that class of accidents to which I have just referred.

Gentlemen, I admit, that when we see surgeons (to search for and detach an ovary) making large openings into the abdomen with morbidly thickened walls and the seat of great morbid changes, without taking into account the temporary contact of air with the peritoneum, and the horrible mutilations necessary for attaining the object desired, there need not be any alarm at the proposal to make a large incision in the *linea alba,* so as to enable the hand to be introduced into the abdomen, there to seek for and destroy the obstacle, or to drag forward the particular intestinal convolution in which it is advisable to form an artificial anus. It appears to me, therefore, that the undeniable success which has attended ovariotomy would justify, for the cure of internal strangulation, recourse being had to an operation which, though perhaps more calculated to excite alarm, is surer, more rational, and less dangerous than ovariotomy. For this reason, it is incumbent on me to address you on the modes of performing gastrotomy for the treatment of intestinal occlusion.

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* LABRIC: Thèses de Paris, 1852.

In 1676, Paul Barbette, a surgeon of Amsterdam, very distinctly suggested opening the abdomen in obstinate volvulus or intestinal intussusception. He said: "When the ordinary means have proved unsuccessful, would it not be opportune to make an opening through the muscles and peritoneum, to disentangle the intestine, rather than allow the patient to die without an effort being made to save him?"

Some years later, Nuck, the expert anatomist, caused gastrotomy to be performed with success in the case of a woman affected with volvulus.*

For a very long period nothing was said about this operation; but after the lapse of a century, in 1772, Renault, an illustrious surgeon, performed the double operation of gastrotomy and enterotomy under the following circumstances. A young man had been operated upon for strangulated hernia under hopeful conditions, when, several days after the operation, without any external appearance of the hernia, symptoms of internal strangulation presented themselves. Renault, without hesitation, cut into the abdomen: having found that the small intestine was strangulated throughout a certain part of its course, he made an artificial anus. The operation was successful: on the twenty-eighth day, the fecal matter passed by its normal outlet; and the wound was completely cicatrized.

In 1776, Pillore (of Rouen), and in 1793, Duret (of Brest), successfully practiced gastrotomy both when the obstruction was in the large and when in the small intestine.

Dupuytren did not meet with similar success in 1818; but that did not prevent Mannoury, in the following year, from proposing as an extreme resource, in internal strangulation, the formation of an artificial anus, and the maintenance of the incised intestinal convolution in contact with the abdominal incision by a thread passed through the mesentery.

In 1838, Dr. Monod, in conformity with the rules of Dr. Mannoury, performed gastrotomy in a case of internal strangulation. At the autopsy, a serious lesion of the cæcum was discovered. In the same year, Professor Laugier called attention to the fact, that after the reduction of hernia, there is often a continuance of the symptoms of strangulation of the intestine: in such cases, he recommended enterotomy, and added that gastrotomy might also be appropriate in other cases than those resulting from the reduction of hernia.

All these facts were nearly forgotten, when, in 1844, M. Maisonneuve read to the Academy of Sciences the case of a patient upon whom he had operated for strangulated hernia, by dividing the upper ring of the inguinal canal. Immediately after the reduction of the hernia, the patient experienced relief; but next day, he had symptoms of internal strangulation. M. Maisonneuve, without hesitation, reopened the inguinal wound, and ascertained that the intestine was perfectly reduced, but that there was an adhesion of the neck of the sac to the intestine, which he considered was probably the cause of the retention of the fæces. At this point, in fact, the intestine was gorged with feculent matter; and the surgeon after having made himself quite certain that there was adhesion of the intestine to the neck of the sac, and of the latter to the opening of the inguinal canal, made an incision into the gorged portion of intestine, so as to form an artificial anus. There was perfect and rapid recovery. In the following year, M. Maisonneuve made this case the text of a very interesting memoir.†

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* NUCK: Operationes et Experimenta Chirurgica. Leyden, 1692.

† MAISONNEUVE: Mémoire sur l'Entérotomie de l'intestine grêle dans les cas de l'oblitération de cet organe. [*Archives Générales de Médecine*, 1845, t. vii, p. 448.]

Subsequently, MM. Denonvilliers and Nélaton declared themselves favorable to gastrotomy in intestinal strangulation.* According to my colleague, gastrotomy necessarily leads to enterotomy; but in my opinion, on the contrary, enterotomy ought not to be performed, except when it is absolutely necessary to make an artificial anus, that is to say, in the cases in which the intestine is gangrenous, or contracted in consequence of lesion of its tissues. When there is only volvulus, invagination of recent date, or strangulation caused by cellular bands, by adhesions of the ileo-cæcal appendix, or of an intestinal convolution, I believe that, the causes of the occlusion being known, and there being no serious lesion of the intestine, it is sufficient to destroy the different causes of the internal strangulation. A case reported by Valse shows that in cases of volvulus or invagination, sometimes all that is required to restore the intestine to its normal relations is to remove the symptoms; and so will it be in the cases of obstruction depending upon the other causes which I have enumerated. But to obtain such a result, it will be necessary to make, at some part of the abdominal parietes, an incision sufficiently large to enable the operator to search for the seat and cause of the occlusion both with his hand and eye.

Further, if there is to be a probability of success, the operator must proceed slowly, following the same rules which experience has sanctioned in ovariotomy. There are two questions which may present themselves to the mind of the operator when it is necessary to make an incision into the intestine in consequence of its condition from menaced perforation, or incipient gangrene. Ought he to form an artificial anus? or, would he be rash, after having emptied the intestine by the incision, to unite the lips of the intestinal wound by bringing into contact the cut edges of the serous membrane, and completing the operation as in the cases in which gastrotomy is permissible for the purpose of reducing a volvulus and breaking up abnormal adhesions? It is now an established fact in surgery, that in operating for strangulated hernia, one may successfully return into the abdominal cavity the hernial portion of the bowel, after having brought together by stitches the edges of the opening into the intestine.

It is beyond my province to say more on the surgical considerations connected with this subject; and I now propose to conclude this lecture by relating two successful cases which may be cited as encouragements to perform enterotomy according to the proceeding adopted by Nélaton in cases of internal strangulation.

Sixteen years ago, I was sent for to see in consultation a young painter of Hamburg. I ascertained the existence of all the symptoms of internal strangulation—vomiting of matters seemingly stercoraceous, which had been going on for six or seven days, great tympanitic distension of the abdomen, sunken eyes, and general coldness. Death seemed imminent. I was told that the patient was the subject of hernia: I constantly made an examination with the view of discovering whether there was any inguinal tumor, and I found that there was none. I then asked myself whether the symptoms did not proceed from strangulation at the neck of the sac. I asked M. Nélaton to see the case with me. My honorable colleague, like me, perceived all the symptoms characteristic of occlusion without being able, any more than I had been, to detect the cause. The danger, however, was urgent; and in our opinion it could only be obviated by gastrotomy. The operation for artificial anus was, therefore, performed. There was an immediate cessation of the symptoms. In eight or ten days, the young man was restored: eating with appetite, and digesting well what he ate. In three months, his recovery was complete: at that date, the artificial anus was closed: and four years ago, when I last heard of our patient, he was in perfect health.

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* Savopoulo: Thèses de Paris, 1854.

Nine years ago, one of my honorable professional brethren of Paris called me in, for the third or fourth time, to see his wife, who on several occasions had presented all the symptoms of intestinal occlusion. These symptoms were coincident with ordinary constipation, over which drastic purgatives are generally triumphant. MM. Requin and Beau were consulted along with me. Upon examining the patient, we all agreed that drastic purgatives ought to be insisted on; and that along with their employment recourse should be had to belladonna cataplasms, application of ice to the abdomen, long-continued baths, so as to overcome symptoms similar to those which had been previously experienced by the patient. Notwithstanding these different measures, the symptoms continued; there was great tympanitic distension; the matters vomited had assumed that aspect by which they are characterized in strangulated hernia, and it did not seem probable that life could continue for more than twenty-four or thirty-six hours. Under these circumstances, M. Nélaton was invited to meet us in consultation, and it was then decided to perform gastrotomy. The artificial anus afforded exit to a great quantity of gaseous and solid contents: immediate relief followed. The convalescence was rapid; and the lady is at present in perfect health.

These, gentlemen, are great facts which you ought to bear in mind; for though such cases are of rare occurrence, they are important, because the affection, if left to itself, is nearly always fatal.

What are the rules in accordance with which the operation of gastrotomy ought to be performed?

Perhaps I ought to leave this question to be answered by your surgical teachers; but as I am addressing medical practitioners, who, one day or another, may be called upon unaided to act by themselves in such cases, under circumstances of urgent necessity, allow me to tell you what I should advise and do if the emergency arose; let me say also whether I should not prefer to make a large abdominal incision, as is practiced in ovariotomy.

I begin the operation, as M. Nélaton advises, by making, in the right side, an incision two or three centimetres in length, a little above the crest of the ilium, parallel with Poupart's ligament: the length of this incision is subsequently increased to eight or ten centimetres. In dividing, layer by layer, the skin, the cellular tissue, the muscles and aponeuroses, tying, as may be required, the large vessels involved in the incision, we at last come to the most deeply seated aponeurosis. Proceeding always very slowly, and being very particular in sponging the wound carefully, this deep aponeurosis is cut through, when forthwith the peritoneum is reached. It is taken hold of by a small forceps and incised; afterwards, using the greatest possible precautions, a silver thread, by means of a curved needle, is carried, first through the intestine and then through the abdominal walls; four sutures are then made, two on each side of the incision; two others are made, one at the superior and the other at the inferior angle of the wound; but this time, the abdominal parietes are first perforated, then the intestine, and afterwards the abdominal parietes on the opposite side of the wound. In this way the intestine is fixed everywhere, laterally and from above downwards, to the walls of the abdomen: by this proceeding, no exudation can take place into the peritoneum. *It is then only necessary to make an exceedingly small incision in the intestine* by means of a sharp-pointed bistoury. The opening which M. Nélaton makes in the intestine for the passage of its fluid contents is even less than a centimetre in length.

Such is the operation. It requires more prudence than skill; although, of course, it is always better that it should be intrusted to experienced hands.

Such is the operation which the able professor at the Clinical Hospital practiced in a case which I shall now relate. Dr. Olliffe did me the honor to summon me in consultation with him in the case of a high dignitary of the Russian empire. This general suffered from great disturbance of the digestive functions. For two months his stools had been becoming fewer in number, and more painful. His appetite was impaired. Gradually and slowly the abdomen became distended; gas, at first inodorous and afterwards fetid, was discharged by the mouth. When I saw the patient he had become much reduced in flesh and strength. His face was typical of abdominal disease. There was nothing discoverable, however, indicative of very decided cachexia, nor did the cutaneous tint characteristic of cancer present itself. Through the thin abdominal walls were distinguishable the lumpy masses formed by the distended abdominal convolutions. There was a great degree of tympanites. No pain existed anywhere. For eight days the patient had had vomiting; at first, the matters vomited consisted of alimentary ingesta; they were afterwards of a yellowish color, horribly fetid, and very obviously stercoraceous. There was an exceedingly distressing hiccup. The patient was entirely without spontaneous stools. At rare intervals, and as the result of great efforts, he expelled some gas by the anus. In the first instance, I prescribed the ascending douche. There was administered in that form, twice a day, from four to six litres of liquid. The fluid passed in, and was returned; but it only brought back with it an exceedingly small quantity of fecal matter—small in calibre, and somewhat ribbon-shaped. The existence of an intestinal occlusion was certain; and most probably it was seated in the large intestine, judging from the peculiar form of the matters which were passed. The stricture could not be reached either by the finger or a very long sound introduced into the rectum. There was reason to hope that the stricture was fibrous and not cancerous, as that marked cachexia indicating hereditary taint was absent. Drastic purgatives, the ascending douche, and other means, having all failed, enterotomy was resolved upon, and was admirably performed by M. Nélaton, in the manner in which I have just described. There gushed from the incision in the intestine three large basinfuls of a yellowish, very fetid liquid.

The patient immediately experienced very great relief. The operation was performed on the 22d June, 1863: for the seven following days, gas and matters tinged with bile passed by the artificial anus: the patient was able to eat, and regained his usual strength and spirits. On the evening of June 29th, he had very severe colic; and then, soon after passing gas twice by the anus, he had a formed motion. During the night, he had another stool. On the following day, unfortunately, he had a violent paroxysm of fever, which continued nearly an hour. On the day after the next, he had another attack of fever. The wound, however, was not very painful; and there were no symptoms of peritonitis. Nevertheless, on the 1st July, nine days after the operation, the general died, after having given for seven days the best hopes of recovery.

Let me now endeavor to explain the mechanism of the cure of occlusion: I only refer to the cases in which the cure is definite, and not to those in which it is accomplished at the cost of an incurable artificial anus.

In cases of the latter class, the cure obtained is only very partial: it is he ate. In three months, his recovery was complete: at that date, the artificial anus was closed: and four years ago, when I last heard of our patient, he was in perfect health.

Nine years ago, one of my honorable professional brethren of Paris called me in, for the third or fourth time, to see his wife, who on several occasions had presented all the symptoms of intestinal occlusion. These symptoms were coincident with ordinary constipation, over which drastic purgatives are generally triumphant. MM. Requin and Beau were consulted along with me. Upon examining the patient, we all agreed that drastic purgatives ought to be insisted on; and that along with their employment recourse should be had to belladonna cataplasms, application of ice to the abdomen, long-continued baths, so as to overcome symptoms similar to those which had been previously experienced by the patient. Notwithstanding these different measures, the symptoms continued; there was great tympanitic distension; the matters vomited had assumed that aspect by which they are characterized in strangulated hernia, and it did not seem probable that life could continue for more than twenty-four or thirty-six hours. Under these circumstances, M. Nélaton was invited to meet us in consultation, and it was then decided to perform gastrotomy. The artificial anus afforded exit to a great quantity of gaseous and solid contents: immediate relief followed. The convalescence was rapid; and the lady is at present in perfect health.

These, gentlemen, are great facts which you ought to bear in mind; for though such cases are of rare occurrence, they are important, because the affection, if left to itself, is nearly always fatal.

What are the rules in accordance with which the operation of gastrotomy ought to be performed?

Perhaps I ought to leave this question to be answered by your surgical teachers; but as I am addressing medical practitioners, who, one day or another, may be called upon unaided to act by themselves in such cases, under circumstances of urgent necessity, allow me to tell you what I should advise and do if the emergency arose; let me say also whether I should not prefer to make a large abdominal incision, as is practiced in ovariotomy.

I begin the operation, as M. Nélaton advises, by making, in the right side, an incision two or three centimetres in length, a little above the crest of the ilium, parallel with Poupart's ligament: the length of this incision is subsequently increased to eight or ten centimetres. In dividing, layer by layer, the skin, the cellular tissue, the muscles and aponeuroses, tying, as may be required, the large vessels involved in the incision, we at last come to the most deeply seated aponeurosis. Proceeding always very slowly, and being very particular in sponging the wound carefully, this deep aponeurosis is cut through, when forthwith the peritoneum is reached. It is taken hold of by a small forceps and incised; afterwards, using the greatest possible precautions, a silver thread, by means of a curved needle, is carried, first through the intestine and then through the abdominal walls; four sutures are then made, two on each side of the incision; two others are made, one at the superior and the other at the inferior angle of the wound; but this time, the abdominal parietes are first perforated, then the intestine, and afterwards the abdominal parietes on the opposite side of the wound. In this way the intestine is fixed everywhere, laterally and from above downwards, to the walls of the abdomen: by this proceeding, no exudation can take place into the peritoneum. *It is then only necessary to make an exceedingly small incision in the intestine* by means of a sharp-pointed bistoury. The opening which M. Nélaton makes in the intestine for the passage of its fluid contents is even less than a centimetre in length.

Such is the operation. It requires more prudence than skill; although, of course, it is always better that it should be intrusted to experienced hands.

Such is the operation which the able professor at the Clinical Hospital practiced in a case which I shall now relate. Dr. Olliffe did me the honor to summon me in consultation with him in the case of a high dignitary of the Russian empire. This general suffered from great disturbance of the digestive functions. For two months his stools had been becoming fewer in number, and more painful. His appetite was impaired. Gradually and slowly the abdomen became distended; gas, at first inodorous and afterwards fetid, was discharged by the mouth. When I saw the patient he had become much reduced in flesh and strength. His face was typical of abdominal disease. There was nothing discoverable, however, indicative of very decided cachexia, nor did the cutaneous tint characteristic of cancer present itself. Through the thin abdominal walls were distinguishable the lumpy masses formed by the distended abdominal convolutions. There was a great degree of tympanites. No pain existed anywhere. For eight days the patient had had vomiting; at first, the matters vomited consisted of alimentary ingesta; they were afterwards of a yellowish color, horribly fetid, and very obviously stercoraceous. There was an exceedingly distressing hiccup. The patient was entirely without spontaneous stools. At rare intervals, and as the result of great efforts, he expelled some gas by the anus. In the first instance, I prescribed the ascending douche. There was administered in that form, twice a day, from four to six litres of liquid. The fluid passed in, and was returned; but it only brought back with it an exceedingly small quantity of fecal matter—small in calibre, and somewhat ribbon-shaped. The existence of an intestinal occlusion was certain; and most probably it was seated in the large intestine, judging from the peculiar form of the matters which were passed. The stricture could not be reached either by the finger or a very long sound introduced into the rectum. There was reason to hope that the stricture was fibrous and not cancerous, as that marked cachexia indicating hereditary taint was absent. Drastic purgatives, the ascending douche, and other means, having all failed, enterotomy was resolved upon, and was admirably performed by M. Nélaton, in the manner in which I have just described. There gushed from the incision in the intestine three large basinfuls of a yellowish, very fetid liquid.

The patient immediately experienced very great relief. The operation was performed on the 22d June, 1863: for the seven following days, gas and matters tinged with bile passed by the artificial anus: the patient was able to eat, and regained his usual strength and spirits. On the evening of June 29th, he had very severe colic; and then, soon after passing gas twice by the anus, he had a formed motion. During the night, he had another stool. On the following day, unfortunately, he had a violent paroxysm of fever, which continued nearly an hour. On the day after the next, he had another attack of fever. The wound, however, was not very painful; and there were no symptoms of peritonitis. Nevertheless, on the 1st July, nine days after the operation, the general died, after having given for seven days the best hopes of recovery.

Let me now endeavor to explain the mechanism of the cure of occlusion: I only refer to the cases in which the cure is definite, and not to those in which it is accomplished at the cost of an incurable artificial anus.

In cases of the latter class, the cure obtained is only very partial: it is the snatching of a patient from immediately threatening death, and that certainly is no small matter; but then, on the other hand, it is condemning him to live with a disgusting infirmity.

That is, however, the only solution of the case for which there is any ground of hope, when the occlusion depends upon compression by a tumor situated external to the intestine or on stricture resulting from organic disease of the intestine itself.

It is otherwise when there exists intestinal invagination, intestinal strangulation by a band, or retroversion of the intestine: under these conditions, there is a chance, though slight, of enterotomy leading to a complete and radical cure.

How then does this cure take place? It may be brought about in invagination in two ways which I have already explained to you. The state of invagination may spontaneously cease from the peristaltic movements of the intestine restoring the parts to their normal position: or, the invaginated portion of intestine may become detached by sphacelus, and fall into the intestinal canal, passing forth by the natural passage, leaving the two surfaces of divided intestine intimately soldered together by a reparative process originating in the parts themselves.

Gastrotomy then, on the one hand, causes a cessation of the symptoms which threaten to put a sudden termination to life, is the means of prolonging existence, and of contributing to recovery in cases in which the unaided efforts of nature might in time accomplish a cure; and, on the other hand, it promotes that cure in the manner which I have just explained.

If the occlusion continue, gas and solid matters go on accumulating in the intestines; and the inordinate distension so induced more and more confirms the occlusion. Should the cure proceed by elimination of the invaginated portion of intestine, it may happen that at the time of the elimination taking place, the distension of the bowel, and the stretched condition of its walls may be so great as to hinder the soldering together of the divided intestinal surfaces: this by allowing the contents of the bowel to pass into the peritoneal cavity, may occasion speedily fatal peritonitis. But, on the contrary, should gastrotomy afford an external exit to the contents of the intestine, the intestine collapsing to a certain extent, will allow the reparative process to proceed, and the two ends of the divided canal will become soldered together.

I shall now state what occurs in cases in which the strangulation is caused by bands, or by retroversion of the intestine on itself. When, in the dead body, we cautiously distend with air a portion of intestine thus coiled up, the air is perceived to pass onwards, and, as it advances, to gradually uncoil the involution. If, on the other hand, there exists a certain amount of resistance, and the insufflation be performed in a forcible manner, the air will accumulate above the obstacle, distending without unrolling the intestine, and thus augmenting the occlusion.

In the living subject, similar phenomena present themselves. A band exerts slight pressure on the intestine, or, in consequence of some mechanical cause which escapes our observation, a part of the intestine becomes temporarily put out of its normal position: the result is a certain amount of impediment to the onward passage of the contents of the bowels: then follow a slightly increased secretion of gas, which goes on augmenting so as to distend the intestine, thereby destroying its contractile power, till, from a disengagement of its gaseous contents, the intestine returns to its normal volume.

In these cases, the same thing may occur which sometimes takes place in hernia. A hernia may with facility pass out and in through an opening which is quite adequate, till, at some particular time, the hernia becomes engorged, and so cannot traverse the formerly sufficient passage.

In internal strangulation of the intestine, puncture, by affording exit to the gas, is sometimes sufficient to cause the symptoms to cease; but in the vast majority of cases, this measure is inadequate, and the resource which we must then look to is enterotomy. It is explicitly indicated when the symptoms of occlusion have existed for six or eight days—when there is great tympanites—when the matters vomited present that peculiar character of which I have spoken to you—and finally, when the persistence and severity of the symptoms presage imminent death.

Though the establishing of an artificial anus is unquestionably a serious operation, it is by no means so dangerous a proceeding as might be supposed. Certainly, the risks which attend it bear no comparison with those to avert which gastrotomy is resorted to. When all other means have failed, therefore, gastrotomy ought to be practiced. It was by this means that my able colleague, M. Velpeau, under desperate circumstances, saved the life of a patient, to whom he was called in by M. Briquet. During my medical career, I have five times recommended its adoption; and I have had the satisfaction to see two patients recover in consequence of the operation, who without it would have been hopelessly lost. I have related to you the history of both their cases.

LECTURE LXXVIII.

HEPATIC COLIC: BILIARY CALCULUS.

More common in Women than in Men.—Rarely occurs in Children.—Composition, Form, and Volume of the Calculi.—Biliary Gravel.—Cause of the Disease is not known.—Sometimes Hereditary.—May be coincident with Urinary Gravel, and be a manifestation of the Gouty Diathesis.—Hepatic Colic.—Diagnosis often very difficult.—May be mistaken for Gastralgia, Colalgia, and Hepatalgia.—Pain and Jaundice are not essentially pathognomonic signs, and may be absent.—They may be the symptoms of other affections, as of Hepatitis, Hepatalgia, or of the Hepatic Colic caused by ascarides or hydatids.—Presence of Calculi in the stools is the only positive diagnostic sign.—Symptomatic affections caused by the Calculi: Acute Hepatitis: Retention of Bile in the liver, in the gall-bladder: Dropsy of the Gall-Bladder: Rupture of Gall-Bladder and its excretory ducts.—Biliary Fistulæ.—Paraplegia, reflex and consecutive.—Treatment of Calculous Disease of the Liver.

GENTLEMEN: Listen to the language used by Morgagni in relation to biliary calculi: "I greatly fear," he says, "that what was true in the times of Fernel is true in our day, and will remain the same in the future, that is to say, that we shall continue to be without characteristic signs by which they can be easily and certainly recognized, and shall, as hitherto, have only conjectures to guide us in forming a diagnosis."* The progress of modern science has not in any way altered the accuracy of Morgagni's proposition: for us, as for our predecessors, the diagnosis of hepatic colic remains imperfect up to the time when the patient passes a calculus or a fragment of one. Till then, there are no data except probabilities—probabilities, in some cases, it is true, exceedingly strong.

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* MORGAGNI: His 37th Letter on the Seat and Causes of Diseases.

It is evident, for example, that when persons complain of having experienced on different occasions, and at intervals of longer or shorter duration, violent pains in the right hypochondrium, pit of the stomach, and round the navel—when these pains shoot through the whole abdomen, and up the chest to the right shoulder—when they are so excruciating as not only to cause the patients to cry out and to throw themselves into an almost convulsive state of agitation, but sometimes even to produce syncope—when they are accompanied by nausea and vomiting, and after continuing five hours are followed next day by jaundice—it is evident, I say, that when these conditions exist, we may almost unhesitatingly pronounce that the malady is hepatic colic, a diagnosis which sooner or later will be entirely confirmed.

Hepatic colic, however, is very far from being always characterized by so well-marked a group of symptoms. Generally, patients only complain of being subject at intervals—twice or thrice a year, perhaps—to what they call *cramps in the stomach.* They give no explanation of the cause of the recurrence of these attacks. The fact which they realize is that the attacks of colic are accompanied by a feeling of anxiety, discomfort, and sometimes a tendency to vomit—that after a crisis of from four to six hours, the symptoms wholly disappear till a new attack sets in. If you ask the patients whether they have remarked that the attacks were followed by jaundice, the majority are unable to reply; but if you are called in soon after an attack, you discover that their skin and mucous membranes have a yellowish color, which is particularly marked in the oculo-palpebral furrow.

The icteric tint is absent in some cases: in others, it is very general, and very decided: I should wish to give you the reasons for the differences which this phenomenon presents itself. It seldom appears till the day after the attack: then also, the stools, which have been few and more or less hard, become of a grayish hue or of an ash color, while the urine assumes the mahogany color peculiar to jaundice. Even during the attack, the urine, which is very copious, is limpid like water from the rock, being in fact what is called *nervous* urine.

Attacks of hepatic colic, though generally transient, are sometimes considerably protracted, there being during their continuance alternations of exacerbation and calm, the latter condition, however, being only partial. I saw with my colleague Dr. Bergeron of the Hôpital Sainte-Eugénie, a woman who had an attack of six months' duration: and, with my friend Dr. A. Joux of La Ferté-Gaucher, I saw another patient who had hepatic colics accompanied by green jaundice, which lasted almost uninterruptedly for three months. The duration of the colics was still longer in the case of a well-to-do Parisian merchant, whom I attended for more than a year without being able to recognize anything more than the symptoms of hepatitis characterized by turgidity of the liver (which was very painful on pressure), jaundice, an almost ever-recurring fever, loss of appetite, and general debility.

Gentlemen, I stated to you that the diagnosis of hepatic colic was of necessity incomplete, so long as the patient had passed neither a calculus nor a fragment of one. Consequently, it is indispensable, when the symptoms lead us to suspect the presence of biliary calculi, to examine each stool attentively, to cause them to be received upon a fine sieve, and to be dissolved and washed by a stream of water in such a way as to cause all except solid matter to pass through: the stools ought to be treated in this way for four or five days after the cessation of the colics, though the proceeding is very disgusting. You saw a woman at the Hôtel-Dieu, who has been many times under treatment in the wards, but who never passed calculi by stool till the third, fourth, or fifth day after the termination of the attack of colic.

Biliary calculi are observed much more frequently in women than in men; and, as you know, it is much more common to find them at autopsies of the old women of the Salpêtrière than at autopsies of the old men of the Bicêtre. The disease is much more common in old age, and in mature age between thirty and fifty, than during adolescence. Youth, however, does not confer an absolute immunity. Two young women between sixteen and seventeen years of age whom we had at the same time in St. Agnes Ward (beds 1 and 34), were remarkable proofs of this. Two years ago, I observed biliary calculi in a girl of nine years of age, whom I saw in consultation at Saint Germain-en-Laye. Lieutaud and Portal have mentioned facts that would show that they may be met with even in newly born children. These exceptional cases do not, however, at all weaken the general rule.

It is a mistake to suppose that biliary calculi vary in color with the age of the subject in whom they occur: their color is solely dependent upon the nature of their constituents. They are generally of a brownish-green color: sometimes, they are of a blackish-brown or are even quite black; in the fresh state, some have been seen to present a bluish and others a reddish tint: it is not unusual to meet with them of an ash gray color: some have been pointed out as white, transparent-like crystals, or, to use the better comparison of Heister, like gum arabic. These whitish calculi are spotted with black and red points, or they may present a yellow golden aspect, or points shining like talc. The different colors are due to the proportions more or less considerable which they contain of cholesterin and the coloring matter of the bile: the colors change as the calculi become dry, from the matters, which in the fresh state produced the colored coating, losing their properties with desiccation; they also lose the lustrous varnished appearance which they sometimes present, and assume that dull hue which some have from the first.

They frequently attain the size of a hazel-nut, and may become as large, or even larger, than a hen's egg: their size is in an inverse ratio to their number. When they are less in volume than a very small lentil, they are no longer considered calculi but "biliary gravel." The quantity of this gravel may be enormous; for, without speaking of the extraordinary cases related by Morgagni, in which the individual grains in the gall-bladder were from seven hundred up to one and two thousand, and even to upwards of three thousand, you will meet with patients who pass by stool spoonfuls of these small yellowish-green bodies. Dr. P. E. Chauffard lately described to me a case bearing on this subject: the patient was a magistrate, who had passed a quantity of small uneven gravel, the size of some of which was that of coarse river sand; their passage by the anus occasioned acute pain and a sort of laceration: the patient stated that the quantity he had passed was sufficient to fill both hands.

Biliary is perhaps more common than urinary gravel; but it is easy to understand why it should more frequently escape observation.

I shall not enter upon a long description of the physical characters of biliary concretions, for were I to do so, I should only be repeating what you have learned elsewhere. I shall merely recall one or two facts to your recollection. Their consistence is very variable: when recent, mere pressure is sometimes sufficient to crush them, and usually their resisting power is about equivalent to that of the stearine used in making candles: when placed in the flame of a candle, they melt, and burn like fatty substances. Their specific gravity is very little greater than that of bile; when dry, they float on water. Their form and size have relation to their number. When there is only one, it is pretty nearly round or oval; at some points, its surface may bear the mark of the parts within which it was formed, and which exerted pressure on it. When the calculi are numerous, they affect the most diversified forms, and are usually many-sided, presenting facettes which correspond with other facettes on other calculi; or they may become imbedded in one another, as if articulated like the heads of bones in their sockets.

Their structure nearly always consists of cortical layers of coloring matter: there is the middle portion consisting of thin triangular layers converging from the periphery towards the centre or nucleus. This central nucleus is generally composed of the coloring matter of the bile and mucus; but sometimes it is a foreign body: in a case mentioned by M. Nauche, it was a pin; in a case of which Lobstein gives a drawing, it was a lumbricus teres which had penetrated the biliary passages.*

It is all the more easy to give an account of the formation of biliary calculi that the coloring matter of the bile not entirely dissolved in the liquid bile, that the cholesterin, which is present only in a state of suspension, constitute, so to speak, microscopic nuclei: when, under these circumstances, there takes place a modification of the biliary secretion causing an abnormal increase in the suspended materials, a speck of coloring matter a little bigger, and a spangle of cholesterin a little larger than the rest, will become the centre of a calculus, particularly if at the same time the flow of bile is abnormally sluggish.

That everything which tends to disturb the secretions of the liver, to alter the composition of the bile, and to prolong its progress through the biliary passages, and the duration of its stay in the bladder, may be regarded as a proximate cause of biliary calculi is, it must be admitted, a very vague proposition; and again, we have not advanced one step towards the solution of the etiological question when we have spoken of the influence of the depressing passions, a sedentary life, office-work, and all the trivial causes which so often come to the help of our ignorance.

It is extremely probable that diet has an intimate relation to this affection; but there is diversity of opinion as to the nature of that relation. According to observations made by Glisson and by Peyrilhe, biliary concretions are found more frequently in the gall-bladder of sheep and oxen slaughtered in March, April, and May, after having been kept on dry forage during the winter, than in those killed during summer and autumn, after pasturing in the meadows; and from these statements it has been concluded that the first-mentioned kind of feeding causes the formation of these calculi. This explanation is open to dispute: for it may be asked, whether in the former case, want of exercise and air have not quite as great a disturbing influence, as nature of aliment on the functions of the liver, and consequently upon the formation of calculi.

Finally, gentlemen, we are baffled in our attempts to discover the real cause of this as of many other diseases. But, be the causes what they may, it is certain that they are dominated by a special predisposition existing in the individual. It is, therefore, the same in this respect with biliary calculi as with renal gravel. Some individuals, who though they lead an active life, and follow a temperate regimen in which vegetables predominate, nevertheless pass gravel almost daily with the urine. It is with difficulty that such persons get rid of the gravelly affection for even a few weeks by taking the iodide of potassium (that specially efficacious lithontriptic), or by drinking the waters of Pougues, Contrexéville, or Vichy. As soon as the treatment is discontinued, and often, even, during it, the malady returns with discouraging obstinacy. A similar statement is applicable to biliary calculi in some women. In virtue of an incomprehensible predisposition, there is a ceaseless formation of new calculi; and the malady is neither cured nor checked notwithstanding the best hygienical and medical treatment.

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* LOBSTEIN: Atlas d'Anatomie Pathologique. Paris, 1829.

It would appear from observations made by several physicians that this predisposition is hereditary.

It has also been observed—and Morgagni has quoted numerous cases in point—that biliary and renal calculi often coexist. So great is the importance which Morgagni attaches to this fact, that he admits, that when symptoms of hepatic colic show themselves in a person subject to urinary calculi, there is strong reason to suspect the existence of biliary calculi, particularly if the subject have passed the age of adolescence.

When we recollect that urinary gravel is very often the sign of the gouty diathesis, we see why the coincidence which I have just pointed out is to a certain extent the reason of another coincidence referred to by physicians as existing between biliary gravel or calculi and gout, especially when the latter, after having been frankly articular, localizes itself in the abdominal viscera.

Gout is an unusual disease in women; and yet it is in women that we most frequently meet with biliary calculi.

In nine out of ten cases of this affection, the gall-bladder is the seat of the gravel. This arises, as is obvious, from the gall-bladder being a reservoir in which the bile naturally accumulates, and in which the conditions of repose and concentration of the liquid are most favorable to that aggregation of molecules by which the calculi are formed. It is in the gall-bladder that they are occasionally met with in large quantity, and in which, when there is only one calculus, it attains an enormous size.

Sometimes, also, biliary concretions may form in the liver itself, that is to say, in the roots, or in the very radicles, of the excretory ducts of that gland. Generally, however, it is biliary gravel and not calculi which we find in that situation: but it occasionally happens that large calculi form and are moulded within the dilated ducts. When the concretions are situated near the periphery of the liver, they constitute tumors projecting from the surface of the organ. In such cases, after perforating the walls of the canals within which they have been developed, they become lodged in the parenchyma itself.

Save the exceptional cases, to which I shall have immediately to recall your attention, in which the calculi open for themselves an outlet from the place wherein they were formed, they find their exit by the intestine. To reach it, those formed in the branches of the hepatic duct must traverse the trunk of that canal, the gall-bladder, the cystic duct, and at last the choledoch duct. It is when the biliary concretions are passing through the excretory passages of the liver that they give rise to the symptoms which constitute hepatic colic.

The pain and the jaundice are symptoms which explain themselves; the first, by the irritation and spasm produced by the foreign bodies traversing the passages, which are narrow and provided with valves; the second, by the obstacle which these same foreign bodies present, from their volume or bulk, to the passage of the bile, when once they become impacted in the choledoch duct. The jaundice is also, and perhaps better, accounted for by the sympathetic irritation of the liver modifying the secretory functions of that organ.

The proximate cause of hepatic colic is not always appreciable when the affection shows itself after some effort, pressure on the hypochondriac region, rather violent exercise, or a powerful mental impression. There is one cause, however, and that, too, the commonest of all causes, which has been distinctly indicated by authors, particularly by Pujol: I refer to the influence of digestion. It is indeed after the principal meal that the hepatic colic usually supervenes, a fact which may be explained in the following manner. The gall-bladder, the cystic and choledoch ducts are muscular and contractile organs, intended to act during duodenal digestion, whilst the liver is going to secrete bile in large quantity to be poured into the intestine. In virtue of a stimulus produced upon the extremity of the choledoch duct, and which is transmitted by reflex action, the secretion of the hepatic gland takes place with a rapidity which is also observed in the secretion of other glands, as, for example, in the secretion of the salivary glands, when the appetite is excited by the sight of nice dishes, or in the secretion of milk, when the nipple is sucked. During duodenal digestion, the biliary secretion is similarly excited; and the bladder moreover contracts so as to pour its reserve of bile into the intestine. This ejaculation of bile, if I may use such an expression, will cause the expulsion with it of the concretions whether formed in the ramifications of the hepatic duct or, as is more usual, in the gall-bladder.

The pains are first felt at the pit of the stomach and around the umbilicus; and when they are localized in the right hypochondrium, this is a consecutive occurrence.

Patients employ all sorts of comparisons to give an idea of the sufferings which they endure; they speak of pinching, tearing, and burning; but that of which they generally complain is an acute, agonizing feeling of constriction, which sometimes extends to the back, epigastrium, and opposite hypochondrium, where it is increased by pressure, even by simple palpation. The pain goes down into the abdomen, in some cases simulating nephritic colic; more generally it ascends into the chest and even to the neck, and it is a remarkable fact that many persons experience it in the right shoulder.

The patients are sometimes exceedingly excited, utter piercing cries, roll on the bed or the floor, endeavoring by ceaseless change of position to moderate their sufferings. In some, the disturbance amounts to more than mere agitation, and consists in convulsive attacks; and in others, there are fainting fits, which occasionally, though very seldom, lead to death.

This kind of colic is frequently accompanied by nausea and vomiting. When it sets in soon after a meal, the food is forthwith rejected, after which a glairy substance is ejected; sometimes, at the end of the attack, there is vomiting of yellow bile. As I remarked at the beginning of the lecture, the urine is at the same time clear as water from the rock. It is not till twelve, eighteen, or twenty-four hours later that it assumes the reddish-brown, mahogany color characteristic of jaundice; and if jaundice is to show itself it is not till then that it will appear.

In hepatic colic, the pain probably depends on the same mechanical cause as in nephritic colic. In the latter, once the renal calculus becomes engaged in the urethra, it is constantly propelled onwards by the urine accumulating in the calices and pelvis of the kidney, and with every onward move which it makes in its passage through the narrow canal, it causes excruciating suffering. We may explain in a similar manner the pains produced by biliary calculi impacted in the choledoch duct. But how are we to explain the pains produced by calculi engaged in the cystic duct? I confess, gentlemen, that I have many times fruitlessly asked myself this question; but, nevertheless, calculi of the gall-bladder and consequently of the cystic duct are by far the most common and also the most frequently productive of hepatic colic. I can quite understand that in some particular movement of the body a calculus contained in the gall-bladder may present itself at the cystic opening and become engaged in the duct; but when it has got there, how will it make way? It certainly does make way; and in its progress causes agonizing paroxysms of pain which patients describe with extraordinary exuberance of language. You then require to bear in mind that the gall-bladder, like the urinary bladder, is provided with a muscle; and that this muscle must contract with increased energy when a calculus is painfully impacted in the neck of the cystic duct, just as the urinary bladder contracts with indomitable vigor when gravel or a fragment of stone is arrested in the prostate, or even in the canal of the urethra. It is for a similar reason that the uterine muscle contracts energetically at the term of gestation when we tickle the neck of the uterus or when the product of conception becomes more completely engaged in that passage. It is very obvious that the gall-bladder is full of bile in the intervals between the times during which digestion goes on, and that it empties itself by a somewhat powerful contraction whilst the food is being elaborated in the stomach and duodenum, but particularly during elaboration in the latter. We can understand that in a gall-bladder full of calculi the muscular tunic will become hypertrophied, just as happens in the case of the urinary bladder when it contains a stone; and this is not a mere rational hypothesis, for the hypertrophied muscular tunic can be demonstrated at the autopsy. The conclusion, therefore, is quite natural, that contractions of the bladder in the first instance propel the liquid against the calculus engaged in the canal so as to accelerate its progress, and that then contractions, irrespective of the bile, push it onwards. We can also see how it is that the paroxysms of pain may depend, to a certain extent, on these contractions, which, like the contractions of all hollow muscles, will be intermittent. I do not require to tell you that when the cystic canal is free, and the calculus is engaged in the choledoch duct, the action of the vesicular muscle may contribute to produce the paroxysms of pain, and aid in propelling the liquid accumulated behind the obstacle, so as to communicate an onward impulse to the calculus.

This rapid sketch which I have now traced embraces the most violent and most characteristic crises of the disease. But, as I was careful to tell you, there are many cases in which the symptoms now described are not so well marked. Generally, your patients will complain of cramps in the stomach; and you may be led to mistake hepatic colic for hepatalgia, gastralgia, or coxalgia. It must be admitted that in such cases it is not very easy to form a diagnosis merely from the nature of the pain. There are, however, certain considerations which will assist us in doing so.

If a patient, subject to attacks of neuralgia in other parts of the body, the face, for example, complains of cramps in the stomach recurring periodically —if he localize with precision the seat of the pain in the epigastric region, and tell you that it comes on at long intervals after eating, sufficient grounds will exist to justify your entertaining the idea that there may be *gastralgia*.

Finally, if the pains appear to be more localized in the left hypochondrium, while there exist at the same time constipation and the other symptoms which characterize *colalgia* (a disease to which I directed your attention in my lectures on dyspepsia), there will be reason to suspect neuralgia of the large intestine.

Should the right hypochondrium be more particularly the seat of pain, if it has been positively ascertained that no biliary calculi were ever passed, if the pains recur with tolerably precise periodicity, as in other forms of neuralgia, I conclude that the case must be one of *hepatalgia*, although that be very uncommon as an idiopathic affection.

But when the pains, whatever may have been their seat, are followed by jaundice, the diagnosis will be much less doubtful; and examination of the stools will show, sooner or later, that the case is one of hepatic colic.

The simultaneous manifestation of these two symptoms generally implies the existence of hepatic calculi, but it is necessary to recollect that one or other symptom, or even both, may be wanting, and also, that they may both be present, and yet there be no biliary concretion.

It is not unusual, perhaps, after an attack of colic induced by the passage of a calculus, for other smaller concretions, or still more probably for biliary gravel, to pass through the prepared passages without occasioning a renewal of the pains. Under other conditions, it is much more unusual for small calculi, or even for gravel, to pass through the excretory passages of the liver without leading to severe and characteristic suffering. The pains, it is true, may become more or less dull, and may only amount to a sensation of discomfort, as I have often observed.

In these very cases, the jaundice supervenes, though not simultaneously, at least within twenty-four hours. This symptom may, however, be absent. The most trustworthy authors have given examples of individuals who passed biliary calculi without ever having had jaundice. In these cases, the concretions were either very small, or if not very small had been too quickly expelled to obstruct the flow of bile, or excite that sympathetic influence on the liver which has so great a share in the production of jaundice. Again, colic has been occasioned by calculi contained in the gall-bladder, which, after being accidentally put in motion, have regained the place they originally occupied without remaining in the canals wherein they were temporarily engaged. I knew a patient who for more than four years had had attacks of hepatic colic, and yet in whom they had never been followed by jaundice. In the fifth year, the attacks became more severe, jaundice appeared, and the disease ceased upon the expulsion of a single calculus, shaped like an olive, the greatest diameter of which was two centimetres.

Absence of pain and of jaundice do not then necessarily imply the non-existence of calculi: more than that, I have said that these symptoms may, in some cases, supervene as manifestations of affections quite different from calculous hepatic colic.

True paroxysms of hepatic colic have been caused by *hydatids of the liver* becoming engaged in the biliary passages. For example, there died not long ago in the wards of Dr. Lasègue at the Hôpital Saint-Antoine, an individual affected with jaundice of a very deep shade of color, in whose body there was found, at the autopsy, hydatids obstructing the excretory biliary conduits. A similar case presented itself in a young woman, who, as you remember, died in our wards on the 20th September, 1863. When I come to speak of hydatid cysts of the liver, I shall have to recur to this case. I would for the present, only remind you, that the hydatid cyst of the liver with which this patient was affected, opened first into the biliary passages, then through the diaphragm into the pleural cavity, and that the first symptoms which showed themselves were violent hepatic colics, which recurred at remote intervals, and were accompanied by very deep jaundice.

These cases, I would remark to you in passing, completely negative an assertion of some physicians to the effect that hydatids in the liver are never accompanied by jaundice.

Dr. Bonfils has collected a very considerable number of facts relating to the symptoms which may be produced by the presence of lumbricoid worms in the biliary passages. These symptoms, which show themselves suddenly, are characterized by violent pains, accompanied by vomiting and jaundice, so similar to, as to be mistaken for, those which characterize hepatic colic depending on biliary calculi.*

Professor Andral has reported cases which appear to prove that hepatalgia, itself very rare as an idiopathic affection, likewise, in some cases, when complicated with jaundice, simulates hepatic colic.†

If I add, in conclusion, that *acute hepatitis*, which occasions sharp pain, recurring in paroxysms, and gives rise to jaundice, may also lead to an erroneous diagnosis, you will see how great are the chances of error, and why the only really sure element of diagnosis which we possess is the presence of biliary concretions in the stools. Up to that point, however well-founded our presumptions may be, they still are nothing more than presumptions.

You will not be surprised, then, gentlemen, that I do not attempt to give you any more precise information in regard to diagnosis, a subject upon which some light is pretended to have been shed by certain authors who describe signs by the aid of which they say, we can recognize the situation of the calculi in the different parts of the biliary apparatus.

It may happen, however, that the gall-bladder, from containing an accumulation of small calculi, projects from under the margin of the false ribs, so as to be recognized through the abdominal walls, in thin subjects. This was the case, as I showed you, in one of our young female patients in St. Bernard Ward. In exploring the abdominal region in women in whom the parts are very flaccid from repeated pregnancies, we can, by making firm pressure with the fingers whilst the patient takes a deep inspiration, reach a hard pouch within which a well-marked crepitation is perceived. But cases of this description are quite exceptional: the case now before us is only the second of the kind which I have met with.

The frequent recurrence of the symptoms, their persistence, their intensity, and the presence or absence of vomiting during the crisis, have by no means the diagnostic significance attributed to them.

When the calculi are engaged in the choledoch duct, there is no bilious vomiting, a fact which is explained by the position of the calculus preventing the passage of bile. Bilious vomiting indicates that that duct is free, and that the foreign bodies are impacted either at the neck of the gall-bladder or in the cystic duct. It is conceivable, however, that small concretions may traverse the choledoch duct and produce colic, without preventing the bile from reaching the duodenum and stomach. We can likewise understand that bilious vomiting is not a necessary phenomenon of hepatic colic, even when the colic is occasioned by calculi which have not preceded beyond the cystic duct.

The duration of the symptoms, their intensity, and more or less frequent recurrence, are exceedingly variable phenomena; and depend upon a great many circumstances, of which the most influential is assuredly the volume of the concretions by which they are caused.

The larger the concretions, the more slowly will they effect their transit: moreover, at any stage of their passage, they may be stopped, and pushed backwards into the gall-bladder, whence, under the influence of new proximate causes, they may again become engaged in the biliary passages, and again excite paroxysms of colic. Or, on the other hand, they may remain impacted, so to speak, in the cystic or choledoch duct; and if they do not produce colic, they will give rise to symptoms resulting from the distension of the gall-bladder or the accumulation of bile in the hepatic ducts.

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* BONFILS: Archives Générales de Médecine, for June, 1858.

† ANDRAL: Clinique Médicale.

You, no doubt, recollect an autopsy which we made in 1861. There was no symptom during life which had led us to suspect the existence of hepatic calculi; but, on examining the liver, we found two engaged in the cystic duct, one being as large as an olive, and the other a little smaller. The larger was closely adherent to the walls of the duct: prolongations of mucous membrane extending into the interior of the biliary concretion had to be broken before the calculus could be detached. I do not suppose that these prolongations were formed when the calculus had attained its full size: it is more probable that at a somewhat early stage, the presence of the calculus had set up acute irritation of the mucous membrane producing fibrinous exudations which became partially organized: afterwards new layers of cholesterin and coloring matter augmented the central nucleus, enveloping the bands of accidental cellular tissue. I can scarcely explain in any other way the incasement which I have described.

When hepatic colic has been of long duration, or has recurred at very short intervals, two symptoms, which I have already pointed out to you, are added to the feverish condition of the patient arising from inflammation of the liver. This inflammation (which has arisen under the influence of irritation extending to the gland itself, and under the influence, also, of a greater or less obstruction of the excretory ducts, by the bile temporarily impeded in its circulation retained in the passages which it traverses), shows itself by the organ becoming increased in size, and the seat of pain being rendered more severe by pressure. The increase in the volume of the liver is sometimes so great that it extends more than a handbreadth beyond the false ribs, and descends into the right iliac fossa. This kind of hepatitis, which is that most commonly observed in temperate climates, often continues after recovery from the colic in which it originated, and when there are no remaining biliary concretions. It becomes chronic: the enlarged state of the liver continues: it is the seat of dull pains, of which, at longer or shorter intervals, there are exacerbations; the biliary secretion is disturbed; and this functional disturbance causes dyspepsia, and sometimes extreme anæmia. This chronic inflammation, frequently, also, becomes the cause of cirrhosis and other organic changes, which sooner or later terminate in death.

The retention of bile in the liver, caused by the obstruction of one of its excretory ducts, also induces enlargement of the organ, dilatation of the larger and smaller ramifications of the hepatic duct, the calibre of some of which may become equal to that of the quill of a goose. These dilatations, sometimes partial, like aneurisms, form small fluctuating tumors resembling abscesses in appearance; but it is only on opening the dead body that we can recognize the nature of the lesions, for the retention of bile in the liver does not declare itself by any symptoms different from those of the hepatitis which accompanies it, and was likewise its cause. Distension of the gall-bladder does not lead to these consequences.

Distension sometimes proceeds so far, that the augmented volume of the gall-bladder causes it to project into the hypochondriac region. By palpation we can detect the tumor by which it is constituted—a fluctuating tumor, which, according to its size, occupies different situations. We may detect it under the margin of the false ribs; or, when the distension is greater, we may find that it extends into the epigastric region, and across the median line into the left side: or that it descends to the umbilicus, or sometimes even to the iliac crests. Cases are recorded in which it occupied the entire abdomen.

Distension of the gall-bladder is generally accompanied by more or less inflammation; and this inflammation leads to thickening of the different tunics; and particularly to thickening of the muscular tunic, a circumstance which explains the relative variety of rupture.

On opening the dead body, no bile is found in the gall-bladder; but, generally, it contains thin mucus resembling white of egg, and at other times a fluid resembling urine, or, it may be, a fluid which is limpid and colorless. There then exists that condition which has been called *dropsy of the bladder.*

This cystitis, which may terminate in suppuration, and which also produces more or less deep ulcerations of the walls of the gall-bladder, may supervene independently of any obstacle to the passage of the bile through the cystic and choledoch ducts, arising from the mere presence of the calculi causing permanent irritation. This, which is, perhaps, the most common cause of the cystitis, explains the persistence of pains during many consecutive years in persons subject to recurrent hepatic colic.

The cystic and choledoch ducts are sometimes the seat of a considerable accumulation of bile, producing dilatation of their calibre. The choledoch duct has been found so distended from this cause as to equal the small intestine in volume; and Morgagni (quoting Schenck) mentions a case of Traffelmann "in which the choledoch duct was as large as a stomach, and completely filled with calculi of different sizes."*

Though much rarer than distension, *atrophy of the gall-bladder*, is a structural change which has been mentioned as one of the consequences of the presence of biliary calculi. The pouch contracts upon the concretions which it contains: its walls become thickened, and adhere so firmly to the foreign body that they can hardly be separated at the autopsy. At other times, the calculus is imbedded in a part of the gall-bladder in such a way as to cause it to form two pouches, one of which contains the foreign body, and the other is filled with bile and mucus.

Inflammation of the gall-bladder is not unfrequently propagated by contiguity of tissue to the peritoneum. In this manner is produced more or less extensive peritonitis.

These peritonitic attacks, generally partial, give rise to adhesions between the gall-bladder and neighboring parts—the omentum, right kidney, stomach, duodenum, colon, and abdominal parietes. The false membranes constituting the adhesions are so thick in certain cases, that when they become lost in the middle of the mass which they form, it is difficult to dissect out separately the gall-bladder. These limited peritonitic attacks sometimes become general in a very sudden manner, assuming a subacute form which soon proves fatal.

However, a rapidly fatal attack of peritonitis does not generally supervene in that fashion. It is usually the consequence of rupture or perforation of the gall-bladder or biliary ducts.

I have observed that, when from obstruction of the excretory passages, the retained bile had accumulated in the choledoch duct, cystic duct, or gall-bladder, the walls of the latter were hypertrophied, and consequently more resistant, which explains why rupture of it is so rare an occurrence in such cases. If, however, acute cystitis supervene, the walls of the gall-bladder undergo softening, and then ulcerate. Thus we have a perforation which causes rapidly fatal peritonitis.

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* MORGAGNI: Letter 37th.

Eight years ago, I attended a retired notary who had been long subject to attacks of hepatic colic. Upon one occasion, I was sent for to see him on account of the paroxysms having assumed an unusual degree of severity. On my arrival I found that the patient had constant vomiting and a tympanitic state of the abdomen; there was total suppression of urine, an excessively feeble, almost imperceptible pulse, and a greatly reduced temperature of the body. To be brief, all the symptoms of subacute peritonitis were present. I pronounced the case to be hopeless, and the next day the patient died.

Although unable to obtain an autopsy, I am justified in saying that the case was one of peritonitis caused by effusion into the peritoneum consequent upon rupture of the gall-bladder or one of the biliary ducts; and that what took place was similar to what had occurred in another patient who died in nearly similar circumstances under my observation.

The case occurred at Tours. A rich inhabitant of that town, a patient of Bretonneau, was suddenly seized during an attack of hepatic colic (which had continued for five or six days) with intractable vomiting and all the signs of severe peritonitis, under which he succumbed in twenty-four hours. On opening the dead body, we found in the peritoneal cavity, a calculus the size of a hazel-nut; and we discovered in the choledoch duct a perforation, through which had passed a considerable quantity of bile along with the calculus.

I am indebted to an excellent pupil, Dr. Werner of Dornach, for the history of a similar case, which is particularly interesting from the difficulty experienced in the diagnosis of hepatic colic.

"Very soon after my arrival in this place," writes Dr. Werner, "I was called to a patient who had what he called very violent *cramps of the stomach*, following great mental emotion. I diagnosed the presence of biliary calculi; and instituted my treatment in accordance with that view of the case. On the following day, the pains having increased, and peritonitis having declared itself, I suspected that rupture of the gall-bladder had taken place; and requested that one of the principal physicians of Mulhouse should be asked to meet me in consultation. *Hepatitis* was the diagnosis of that gentleman, who sneered at my supposing that there were calculi. Dissatisfied with this opinion, I requested that a second physician should be asked to see the patient. He concurred with the former physician's diagnosis. Going home a little shaken in my opinion, I attentively reperused my notes taken at your clinical lectures; and the result was, that I became more than ever convinced of the soundness of my original view of the case. Your lectures on hepatic colic, it seemed, had not yet reached Mulhouse. In two days, the patient died. With permission of the family, I made an autopsy in presence of one of the colleagues whom I had met in consultation. I found twenty-five calculi, as large as hazel-nuts, in the gall-bladder, which had burst and allowed the bile to pour into the peritoneum: a calculus larger than any of the others was impacted in the choledoch duct."

Cases of this kind are not uncommon: and a considerable number have been reported by authors. In some cases it was pure bile, serosity, or mucus which escaped; in other cases it was pus, the inflamed gall-bladder being transformed into a sort of abscess: while in other cases, again, biliary concretions of greater or less magnitude were found in the cavity of the peritoneum.

These perforations and ruptures do not always lead to the formidable consequences of which I have been speaking. When the gall-bladder or its ducts have contracted adhesions with the neighboring parts, perforation may take place, without allowing anything to pass into the peritoneum, because the pouch has opened either externally or into the intestinal canal, urinary passages, or liver itself. It is in this way that external and internal *biliary fistulæ* are produced.

External biliary fistulæ may originate spontaneously, or be formed artificially by the surgeon in opening the tumor, which is sometimes of very considerable size, projecting beyond the abdominal parietes in such a way as to be mistaken for an abscess. Cases of this description are reported by authors, particularly by Jean-Louis Petit.

The following case was communicated to me by Dr. Léon Blondeau, who obtained the particulars from a patient whom he saw at Vichy in 1850.

A gentleman, aged 68, of vigorous constitution, who had had rather frequent attacks of urinary gravel, was seized with hepatic colic in 1843, and soon afterwards perceived a pretty large tumor in the right hypochondrium. It was painful on pressure, and was evidently fluctuating. The patient consulted Professors Rostan and Cruveilhier, and insisted that he should be operated on, as he believed that by an operation he could be cured. He was advised to wait; but during the following year, his malady continuing, a surgeon of Versailles consented to apply three cauteries to the tumor. After separation of the eschars, one of the cauterizations closed up, and two others, after giving exit to mucosity tinged with bile, became the orifice of fistulæ, whence issued about a dozen calculi, several of which were as large as the extremity of the little finger. From that time, the patient, without being aware of it, occasionally passed concretions: he found them amid the dressings of the wound in the morning. Sometimes, however, the expulsion of calculi a little larger than usual occasioned pain. He mentioned that on one occasion a considerable quantity of bile passed through one of the fistulæ, and that coincident with this flow of bile, which continued for about a fortnight, there was a certain degree of loss of flesh; the plumpness lost during the flow of bile was soon regained when the flow ceased. In addition to the wounds affording exit to calculi and harder concretions, they sometimes discharged blood and serosity. In other respects, the health of the patient was good.

I attended a similar case along with my honorable friend Dr. Laguerre. Our patient was a gentleman sixty years of age who had been often tormented with hepatic colic. Consequent upon an unusally severe and persistent attack of colic, there supervened acute pain in the right side, in the situation of the gall-bladder. There was soon felt a puffy state of the parts, the skin became red, and a real abscess formed, by opening which an exit was given to a muco-purulent fluid and calculi.

Dr. Guyon communicated to the Academy of Sciences a case in which recovery followed the passage of a biliary calculus through the abdominal parietes. The patient was a lady, who after presenting for some time a not very painful tumor in the region of the liver which only occasionally produced slight febrile action, passed by the tumor, which had been opened by caustic potash, a triangular calculus of about six centimetres in its greatest and of about four and a half in its smallest circumference. Consequent upon the expulsion of the foreign body, the local discomfort diminished day by day, the fever ceased, and the cure was complete.

Gentlemen, you are aware, that M. Petit held that it ought to be regarded as a rule in surgery, to anticipate by operation the opening of the gall-bladder, with a view to prevent fatal peritonitis from its contents passing by a spontaneous rupture into the peritoneal cavity. Establishing a similarity between retention of bile caused by calculi in the gall-bladder and retention of urine caused by stone in the urinary bladder, he came to the conclusion that lithotomy was applicable in both cases. After quoting Petit's opinion Van Swieten adds: *Forte prima fronte audax apparebit facinus talia moliri; sed certe audacior ille fuit, qui primus ex vesica urinaria sectione calculum educere tentavit.* Petit, however, held, that it was only when the gall-bladder had contracted adhesions with the abdominal parietes that the operation ought to be performed; he pointed out that if it was resorted to under other conditions, the very accidents we desire to prevent would be caused, by establishing a communication between the gall-bladder and the abdominal cavity. He indicates the signs by which the existence of adhesions can be recognized: but as Boyer justly remarks, these signs, which in reality are only two in number—immobility of the tumor, and puffiness of the integuments, have no certainty of character. According to Boyer, it would, therefore, be better to wait for clearer evidence of the existence of the affection, and till the tumor has shown a decided tendency to open externally.

Nevertheless, gentlemen, there are cases in which the imminence of the patient's danger obliges the physician to interfere as promptly as possible. That we may proceed with perfect safety in these cases, we must, following the practice of Bégin, endeavor to produce adhesions, by cutting through the abdominal parietes, layer by layer, till the peritoneum is reached. When this has been done, we wait twenty-four hours before completing the operation by cutting into the tumor itself. Proceeding in that way, the same results are attained as by Récamier's method of opening the tumor by the application of potassa fusa. That was the plan pursued in the case I have just related to you.

These proceedings are not free from danger. I have often mentioned another plan, which I have devised for accomplishing the same object. I use a multiplicity of acupunctures in the following manner. I insert thirty or forty steel-needles having large heads: I cause them to penetrate to the gall-bladder. In treating ovarian cysts, I proceed on a similar plan; and indeed it is principally for such cases that I have employed it. The needles are allowed to remain undisturbed for three or four days: they are then removed, when another set are introduced in the spaces between the punctures made by the first set: this system is renewed a third time. It is essential that the needles have heads of sealing-wax: it is likewise essential that a shield of glove-leather be applied over the part, and traversed by the needles at the time they are pushed into the skin: unless this double precaution be taken, the needles will very quickly penetrate into the tissues, there becoming lost, not without risk of dangerous consequences.

The practice which I have just described is certainly simpler, and freer from danger than any other as yet proposed for obtaining adhesions between a cyst and the abdominal serous membrane. I need not say that the small inflammatory areola developed around each puncture causes, from the proximity of the punctures, inflammation of the peritoneum sufficient to comprise all the surfaces which we wish to adhere.

You understand, gentlemen, that *internal biliary fistulæ* are quite beyond our means of treatment. As I have just been telling you, communications may be formed between the gall-bladder and the duodenum or colon.

An old lady, who lived in the Place Royale, was seized after an attack of hepatic colic, with violent pains, which were limited to the left side: along with these pains, she had obstinate constipation. These symptoms had led me to think that there might be pelvic abscess, when, in one day, the patient passed forty calculi by stool.

Expulsion of biliary concretions with the urine proves that a fistulous passage may form between the gall-bladder and the pelvis of the kidney.

Cases reported by reliable authors, such as Frank, show that similar communications may become established between the gall-bladder and the liver itself.

"A woman," says Frank, "who had suffered severely from hepatic colic, before her death, presented signs of gangrenous inflammation of the liver. At the autopsy, the concavity of that organ was found to be occupied by a large abscess containing fetid pus. Through one of the sides of this gangrenous pouch, there projected the point of a triangular calculus. The walls of the gall-bladder cartilaginous, a finger-breadth in thickness, adherent to the colon and duodenum, only communicated with the liver by several sinuses, whence flowed similar fetid purulent matter. It also contained two calculi as large as chestnuts, and many others of smaller size."

When biliary calculi once get into the digestive canal, whether they enter by the natural route, the choledoch duct, or by a fistulous passage, the peristaltic movements generally carry them on to the anus, whence they are expelled with the stools. There are cases, however, very unusual cases, it is true, in which calculi have ascended into the stomach, and been vomited.

You no doubt remember the history of a young woman who was admitted to St. Bernard Ward with symptoms of formidable peritonitis. At the autopsy, we found a biliary calculus impacted in the appendix vermiformis, in which it had caused a perforation. Similar cases are mentioned by authors.

You had an opportunity of observing one of the most curious examples of these internal fistulæ in a woman who lay in bed 28 of St. Bernard Ward. The following is a succinct account of the case: A woman fifty-three years of age became a patient in St. Bernard Ward, in 1863. She stated, that some years previously she had been under treatment by Dr. Béhier at the Hôpital Beaujon for paraplegia, with lesion of the vertebræ. The paraplegia was fortunately cured.

I was first of all struck with the deeply jaundiced tint of the skin, which at once fixed my attention upon the liver. The patient stated that some weeks previously she had experienced violent cramps in the stomach, which were accompanied by nausea, and sometimes by vomiting: on the following day, the skin became yellow, and the urine assumed a very dark color. On each recurrence of these symptoms, she had an attack of fever characterized by shivering, followed by a severe and protracted hot fit, but which was not succeeded by sweating. Some weeks after she came into the hospital, she had a much more violent attack of colic. The pain in the hepatic region assumed an exceedingly intense form; and for at least a week I thought there was severe hepatitis with inflammation of the gall-bladder. Then she had, at nearly the same hour, daily, for almost a month, shivering followed by heat and sweating: at the beginning of the paroxysm, there was no diminution in the jaundice, and always an exacerbation of the pain. I remained in the belief that the fever was caused by the hepatitis, and by the inflammation of the gall-bladder induced daily by the presentation of a calculus at the entrance of the neck of the cystic duct.

Examining the liver by palpation, I found that throughout its whole extent, it was hard and very sensitive. On the right side, the great lobe extended six or eight centimetres beyond the margin of the ribs; and as the abdominal walls were thin and flaccid, it was easy to follow the sharp edge of the liver to the point where it became lost under the left hypochondrium.

After following the cutting edge of the liver for nearly ten centimetres from right to left, I all at once found an interval occupied by a globular body having the liver for its base and projecting downwards: it was about the size of an orange, hard, without inequalities of surface, and very painful: then came another division, limiting on the left the projection of which I have just spoken: and after this, I could again feel the margin of the liver, and continue easily to follow it.

Every one frequenting the wards was able to repeat this exploration: and, like me, all thought that the tumor they felt on the margin of the liver was the distended gall-bladder. I was more than ever persuaded that it contained calculi, that a calculus obstructed the cystic duct, that there existed inflammation and extreme distension of the gall-bladder, and that there was also subacute hepatitis.

However after residence in hospital for two months our patient went out in a good condition. The jaundice had slowly disappeared: the paroxysms of pain had recurred at greatly prolonged intervals, and then wholly ceased. The fever subsided, the appetite and plumpness returned; and when the patient left the hospital, she only complained of a constant acute pain in the hepatic region, while in size the tumor had diminished nearly one half. It was quite evident to me that the calculi remained in the gall-bladder, and that it had become tolerant of their presence.

But at the end of December, 1863, this woman, who had enjoyed good health for six months, returned to our wards with symptoms in all respects identical with those for which she had been previously treated: the same hepatic pains, always coming on towards evening in an irregularly intermittent manner, and always accompanied by a true paroxysm of fever, with shivering, generally slight, and invariably accompanied by jaundice; the intensity of the febrile paroxysm and jaundice was always proportionate to that of the pains. During the intervals between the attacks, the woman's health was pretty good: her appetite returned: she left her bed, went down into the garden, engaged in the usual occupations of her sex, and gradually lost the icteric tint. The periods of remission were even sometimes so prolonged as to cause her to speak of leaving the hospital. She, however, always retained a bulky tumor in the right hypochondrium, presenting the same form which I described: it was painful during the paroxysm, and very slightly sensitive during the periods of remission.

Consequent upon straining in vomiting caused by the attacks of hepatic colic, an inguinal hernia on the right side was produced; and from time to time, this hernia was the cause of suffering. On the 20th May, 1864, the attacks of vomiting suddenly became exceedingly frequent, while at the same time, the hernia was the seat of acute pain; and on the following day, it was evidently strangulated. The operation of colotomy was performed; but two days afterwards the woman died.

Here is an account of the unexpected lesions of the liver which were found at autopsy:

There was no very remarkable increase in the volume of the organ: it extended about five centimetres beyond the margin of the false ribs, and presented a singular condition on its sharp edge. A deep hollow marked the separation between its right and left portions: a little beyond this, on the anterior margin, there was another hollow: situated between these two clefts, there was a large rounded lobule which looked like the gall-bladder distended with calculi. The general aspect of the liver was very much that presented by cirrhosis. Its external surface was studded with a multitude of yellowish-white granulations, among which was perceived the deep-brown color of the organ: the yellow tinge was, however, much less decided than in cirrhosis, and the tissue, when torn, did not present the granular aspect characteristic of that lesion.

On its inferior surface, the liver presented the appearance of a double gall-bladder: there were seen, in fact, in the situation of the fundus two pyriform tumors, both of which were evidently distended with calculi. The largest of the two pouches—that is to say, the gall-bladder—was situated internally, immediately below the isolated lobule which I have described. Its globular form, and the resistance which it presented, were sufficient to indicate that it contained solid bodies. From the neck of this *tumor* proceeded a number of very dilated canals, which were rounded off within the lobules. Conspicuous among these canals were two describing two concentric curves, the concavity of which was to the front: the one led to the upper and posterior part of the left lobe: the second sent a branch to the quadrilateral lobe, and became lost on the anterior part of the internal extremity of the liver.

There existed no trace, therefore, of the usual distribution of the biliary ducts. A fibrous cord, comprised within a peritoneal fold, passed downwards from the middle part of the curved canal I have described; but from its having been cut near its origin, I could not ascertain where it terminated.

The accessory pouch situated external to the gall-bladder, to which it was adherent at its summit, was of an oval form, and immovably fixed to the inferior surface of the liver by close adhesions.

After ascertaining the nature of the external appearances, the biliary reservoir and its afferent ducts were opened. From each incision made in the course of the dilated ducts, there came a gush of yellow bile, containing a great deal of minute gravel. The gall-bladder in its inferior part contained a round calculus the size of a large hazel-nut, yellow externally and internally. Above the place occupied by the calculus, was a large cylindrical pouch constituting a common reservoir, in which all the biliary ducts terminated. There was decided thickening of the walls of the gall-bladder.

At the base of this dilatation of small tubes, immediately above and external to the calculus, there was a series of lines indicating very well the spiral form of the cystic duct. Above this point was seen a rounded cicatrix, the centre of which was perforated by an opening leading to the accessory pouch. Another fistulous passage led to the duodenum.

The accessory pouch, filled by three large and three small calculi, pale externally and yellow internally, communicated with the gall-bladder by a small cavity leading into a rounded pouch, which exactly resembled a canal contracted at both ends. The bottom of this pouch was large, and was filled by the three calculi of which I have spoken. The walls, which were very thick, presented internally a membrane marbled by vascular arborizations filled by a whitish fluid, in which were seen, by the aid of the microscope, numerous pus-corpuscles, mingled with small globules of fat. Even the walls of this pouch, when microscopically examined, presented only the elements of adventitious tissue: the internal surface of the cavity was lined by pavement-epithelium.

The duodenum, otherwise in a healthy condition, had contracted intimate adhesion with the gall-bladder: by means of an oblique passage through these adhesions, a fistulous communication was established between the gall-bladder and the digestive canal. The intestinal orifice of this passage was quite smooth and rounded; it opened at the beginning of the second portion of the duodenum and easily allowed to pass the canula of a capillary trocar. The opposite opening was smaller, and seemed as if cut by a punch.

It was, therefore, by this indirect channel that the bile filtered into the intestine from time to time, in compensation for its inability to pass through the obstructed choledoch duct. We still had to make out the vestiges of that canal. The spiral lines situated near the orifice communicating between the gall-bladder and the neighboring pouch, seemed to indicate the position of the commencement of the cystic duct: and the enormous sac which surmounted it, and received all the biliary ducts was in reality the enormously dilated hepatic duct.

The choledoch duct was no doubt represented by the fibrous cord already mentioned. This cord occupied the anatomical situation of the excretory biliary duct; it presented a distinct cavity, two or three millimetres in length, and had become impermeable. Unfortunately, it was impossible to decide the question by following the course of this cord into Vater's ampulla. However, it seems pretty certain that the pouch glued on to the gall-bladder was an accidental cyst formed around calculi which had fallen into the peritoneal cavity.

This case, besides other remarkable peculiarities, was characterized by a series of attacks of hepatic colic in each of which there was a real paroxysm of intermittent fever, which always began in the afternoon. How are we to explain this intermittence in the phenomena, seeing that the lesions of the biliary passages were so deepseated, so inveterate, and so permanent?

Let me add, that it was impossible not to mistake for the gall-bladder distended with calculi, an irregular lobe which was felt extending across the abdominal walls. Here we see what would have been the embarrassment of a surgeon, upon rashly opening this supposed gall-bladder to evacuate its supposed calculi. I only allude to this point because some of you who attend the daily visit discussed the propriety of operating, a proposition which I emphatically rejected.

The woman of whose case I have been speaking was paraplegic; and her paraplegia seemed to have resulted from an affection of the vertebræ. I regret that this question was not elucidated at the autopsy; and I regret it all the more that I wish now to speak to you regarding a very unusual symptom, a symptom, moreover, which is perhaps even more remarkable for being misunderstood than for being uncommon: I mean *reflex paraplegia*. You are aware that much has lately been written upon paraplegia consecutive upon affections of the genito-urinary passages, and in particular on that which follows uterine diseases;* but so far as I know there has not yet been published any description of the paraplegia which is a sequel of hepatic diseases. In his excellent work on diseases of the liver, Frerichs has mentioned this subject.† Here, however, are the details of a very remarkable case of this kind.

In November, 1863, I attended along with my friend and pupil, Dr. Peter, a lady (Madame d'O.) who was sent to us from the country by an excellent physician, Dr. Levavasseur, of Blanc. She was some years beyond thirty, and had married at the age of sixteen: she had had seven children and a miscarriage without suffering from any consecutive ailments. She was remarkably fat in early youth. She had several times been attacked with an eczematous eruption on the ears, neck, and cheeks.

"Till 1862," said Dr. Levavasseur, in a letter which he sent to us, "she never had any serious illness. For some years, however, she had suffered occasionally from epigastric pains, which seized her suddenly, lasted some hours, and ceased under the influence of calmatives. There seemed a coincidence between the first manifestation of these pains and the cessation of the herpetic affection. About four years previously, Madame d'O. had lost a sister, who was the subject of Addison's disease. Some members of her mother's family had had gout.

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* See R. LEROY (D'ETIOLLES): Des Paralysies des Membres Inférieurs: Paris, 1857.—Also, BROWN-SEQUARD: Paralysis of the Lower Extremities: London, 1861.—Also, JACCOUD: Les Paraplégies et l'Ataxie · Paris, 1864.

† FRERICHS: Traité Pratique des Maladies du Foie. French translation; Paris, 1866.

"A year ago, about the middle of November, Madame d'O., being then between the fourth and fifth month of pregnancy, after two days of fatigue from long walks, was suddenly seized with violent epigastric pain, apparently of the same nature as that which she had before experienced from time to time, and which had generally been of short duration. On this occasion, however, it was otherwise: the pain continued, took possession of the entire hepatic region, and irradiated backwards to the dorsal vertebræ: it was sensibly increased by the slightest pressure. There was intense fever, and the pulse was 120. Universal jaundice showed itself; and the general volume of the liver became notably augmented. There was pain in the right shoulder, and slight epistaxis. After eight days of active antiphlogistic treatment, there was a diminution in the intensity of all the symptoms, which in the opinion of the physicians who had seen the patient —Dr. Mascarel of Chatellerault, Dr. Arnould of Blois, and others—were the characteristic symptoms of acute *hepatitis*. The fever subsided; the pulse fell to 100, and then to 90; the yellow color of the skin began to fade; and all seemed to be going on towards speedy and complete recovery. But such really was not the case: from the time that there was an abatement in the original acute symptoms, at first quite localized in the hepatic region, that is to say for a period of eleven months, there occurred a series of symptoms, various in character, and unusual in respect of the original nature of the disease.

"For several months, there was complete loss of appetite, disgust at every kind of aliment, frequent vomiting of undigested food, great thirst, the tongue denuded of its epithelium, and obstinate constipation. During this period, the pulse ranged between 90 and 100.

"Subsequently, there supervened a general condition of pain—*hyperæsthesia of the skin of the whole body*—when the slightest pressure was made, and this was most manifest all round the chest and in the superior extremities. After a time, the patient had attacks of acute pain *coming on spontaneously:* they extorted cries from her, and gave her no respite: their violence, however, varied irregularly in paroxysms: their seat was principally in the extremities and specially in the fingers and toes: by slow degrees, this state—always coincident with the disorders of digestion already mentioned—became modified: the paroxysms of pain became less violent, occurred at longer intervals, and then entirely ceased. They left behind them, however, a peculiar state of *muscular impotence*, which still exists from the waist to the toes.

"I ought here to mention that, in respect of this want of muscular power, there has been an amelioration in the state of the patient. Her ability to move the superior extremities was not always as great as it now is: for a long time, she had great difficulty in using her hands: for a long time, also, she was unable to change her position in bed, to move herself from one part of it to the other, to turn from one side of the body to the other, or to bend and extend the lower extremities without assistance. Now, however, she can perform all these movements.

"For some mouths, the great disgust at food has gradually disappeared; and at present, the patient eats with an appetite which it is often necessary to restrain. She has long since ceased to vomit her food; and the constipation has become less obstinate.

"Madame d'O. went to Vichy; on her return home, the pulse had entirely lost its frequency, and before her departure for Paris, it was between 60 and 65.

"Amid all this succession of symptoms, there was great variation in the state of the liver: after the disappearance of the original acute symptoms, it returned to its normal condition; but on several occasions, it again became enlarged, though the increase in size was not great. Likewise also there were several returns of the jaundice with a temporary icteric character of the urine. These returns of jaundice were consequent upon paroxysms of pain experienced at the epigastrium and in the region of the liver, which recurred about two months prior to her leaving Vichy, *for the first time since the beginning of the malady*. The pain had the same character as at the beginning: that is to say, it was epigastric and hepatic, characterized by acceleration of pulse and by jaundice, and excepting a short continuance of the jaundiced appearance of the skin it was completely at end in fifteen or twenty hours.

"During her sojourn of two months at Vichy, there was a more or less frequent recurrence of the paroxysms of pain: since her return home, they have been more frequent, happening several times a week. They have acquired a character different from that which they originally possessed. At first, they were characterized by pain in the epigastric and hepatic regions, which soon invaded the trunk, loins, shoulders, and spine. Any attempt to speak occasioned struggling and difficulty from constriction of the jaws. There supervened, after a longer or shorter period, natural or provoked vomiting of a thick, stringy, glairy fluid; but there never was any food thrown up, even when the vomiting occurred immediately after a meal. The pain then suddenly ceased: a general relaxation succeeded: and the patient experienced no subsequent effects of these violent paroxysms, except a feverish state for twelve or fifteen hours, and a sensation of general bruising.

"About a month or three weeks ago, after the patient had not had an attack for more than the usual interval of six or seven days, she was seized for the first time, with embarrassed movements of the tongue and an extreme difficulty in pronouncing words, a state which continued till next day, and then disappeared, an access of pain occurring at the same time.

"Embarrassment in moving the tongue has returned since then on different occasions, but to a less decided degree.

"The lady's accouchement took place in January, without leading to any notable change in her state of health. The infant lived eight days. Before delivery, she had albumen in the urine, and puffiness of the extremities.

"Biliary calculi were never dicovered in the stools."

To sum up this case: Violent attacks of hepatic colic occurred in a lady descended from a gouty father, and who herself had had eczema, which according to Dr. Bazin's doctrine must have been arthritic. During the course of these hepatic attacks—and this is the point which I wish to set forth in relief—many nervous symptoms in succession showed themselves, involving both motion and sensation, consisting first in general hyperæsthesia, and terminating in paraplegia.

Here is the lady's state when I saw her for the first time with Dr. Peter: There was an intensely jaundiced appearance with bronzing: the liver, which extended four finger-breadths beyond the false ribs, was hard, slightly painful on pressure, and free from nodulation: the hypochondrium was covered with the cicatrices of wounds produced by cauteries applied on account of supposed chronic hepatitis which some physicians had diagnosed. There existed emaciation in a marked degree, anorexia, difficult digestion, great general debility, but no fever. Along with these symptoms, the lady had great difficulty in moving the inferior extremities, which were not only weakened, but somewhat contracted. I tried to make the patient walk. I observed that walking had not merely become very difficult in consequence of the feebleness of her limbs, but was impossible from the vicious position which the feet had taken. They were in a state of forced extension; and as they had been for a long time in an anomalous situation, they had become stiff, and in a condition approaching pseudo-anchylosis. As this vicious position of the feet involved an incapacity to place them at right angles with the legs, standing was impossible.

My diagnosis was hypertrophy with chronic hyperæmia of the liver, and without any alteration in the hepatic tissue. I looked upon the hypertrophy as the consequence of the hyperæmia which was a consequence of a series of violent attacks of hepatic colic. Bear in mind that I attributed the attacks of hepatic colic to the presence of biliary calculi. The formation of these calculi, I was inclined to attribute to the gouty diathesis (the first manifestations of which were eczematous eruptions), and which was hereditary in Madame d'O. As for the paraplegia, it was in my opinion dependent upon the hepatic affection, the case being analogous to those cases of paralysis which are called *reflex*, and which supervene in certain persons after affections of the bladder or uterus—the only difference being, that paraplegia consequent upon disease of the liver is a much more uncommon occurrence, and one which has not hitherto been described. Finally, Madame d'O. was, from time to time, affected with a sort of paralysis of the tongue which either prevented her from uttering a single word, or caused her to stammer. These symptoms, which were transient, supervened under the influence of even slight mental emotion.

We proceeded energetically with the treatment. Madame d'O. came to us as an infirm person, who was looked upon by her family as an incurable paralytic. For the paralysis of the motor powers of the inferior extremities, I daily employed electricity: for the vicious position of the feet, I ordered, at Matthieu's, steel-jointed boots, so constructed that one could daily, by means of a trigger-spring, gradually bring the feet nearer and nearer to a right angle. Electrization showed that there was diminution of the electrical sensibility of the muscles of the lower extremities, and almost complete abolition of the electrical contractility of the same muscles: there was, however, a partial return of the voluntary contractility. After fifteen times employing electrization and shampooing (which occasioned great pain), there was a partial return of sensation and electrical contractility: and the voluntary movements became a little more extended.

The patient leaves Paris, to place herself again under Dr. Levavasseur, whose treatment I recommended in all its details; and which was intended to bear simultaneously on the articular rigidity, the paraplegia, and the calculous affection of the liver. Here is an extract from a letter written by Dr. Levavasseur to Dr. Peter some time after her return to Dr. Levavasseur's care.

"Since the return of Madame d'O. from Paris, her state has been always improving, particularly in respect of the paraplegia. Under the use of electrization and shampooing, there was a speedy return of sensation and electrical contractility in the muscles of the lower extremities. For the last two months, the electrization has been discontinued on account of its having become insupportable by Madame d'O. Since that time, she has, without any other assistance than a short stick, walked distances of some hundred metres, and moved about her house, going from one story to another without any other aid. Her steel-jointed boots have been long discontinued.

"The large muscles of the thighs and calves of the legs have not yet regained their natural size; but, nevertheless, they feel, when handled, as if they had a much better development: the adipose tissue constitutes a layer very thin compared with the obesity which existed prior to the malady.

"There was very little change in the special symptoms of the hepatic affection. The paroxysms, which were perhaps rather less frequent, occurred about once every fifteen days: they were as protracted and as violent as before: no calculi were passed.

"The speech, also, was frequently embarrassed: not a day passed during which the symptom was not produced several times by the most trifling emotion.

"There was no fever: sleep was excellent, and the appetite was good. For about a month, however, digestion has been somewhat difficult: after meals, there was distension of the abdomen, an uneasy feeling at the epigastrium, with flushing of the face and head. I had for some time discontinued the use of ether and turpentine capsules. The catamenia had not reappeared."

In June, 1864, I saw this lady: she was then walking very much as she did before her illness. To confirm her restored health, Madame d'O. went to Néris, where she remained in a most satisfactory state till August. At the end of that month, Dr. Levavasseur wrote to me to say that there was "a continuance of her state of general amelioration, a gradual restoration of plumpness and strength, a return of the menstrual function for about three months, with a more and more complete disappearance of the different paralytic symptoms, except, perhaps, that from time to time there was some embarrassment of the tongue in speaking. There was, however, a recurrence, pretty much as before, of the hepatic attacks. In conclusion, let me add, that for a long time past the cessation of the paralytic symptoms has been complete, and that the attacks of hepatic colic have been less frequent."

This case, gentlemen, if I be not mistaken, is a very remarkable example of "reflex" paralysis occurring as the sequel of a calculous affection of the liver. The unusual nature of the case has induced me to enter fully into the details: and I feel assured that once attention has been directed to the possibility of paraplegia occurring as a sequel of affections of the liver, additional examples will be detected by observers.

The manner of recovery from hepatic colic has not always been by the evacuation of calculi. I have frequently called your attention to the fact that we very often find, at the autopsy, numerous hepatic calculi in the bodies of individuals who for a long period had ceased to suffer from the symptoms which arise from the presence of biliary calculi.

When the cystic duct is closed by the impaction of a large calculus, inflammation of the gall-bladder is produced, and it becomes distended by the accumulation of mucus secreted in consequence of the inflamed condition of the mucous membrane. But the duration of this inflammation has a term: the secreted mucus is reabsorbed: the gall-bladder shrivels up, contracting upon the calculus: the pain, at first acute in the region of the gall-bladder, becomes more and more blunted: and the bile flowing freely through the choledoch duct, the health is perfectly re-established.

There are other cases in which we find the cystic duct obliterated by a pretty large concretion, and numerous calculi floating in the greenish mucus by which the gall-bladder is distended: tolerance is established: the inflammation of the gall-bladder comes to an end, and the calculi, ceasing to be engaged in the neck of the cystic duct, cease to cause pain. In cases of more unusual occurrence, such as that of which I have just been giving you the particulars, the distended and inflamed gall-bladder contracts adhesions with the omentum or the intestines, and becomes ruptured, whereupon the calculi, accompanied by the pus and bile, fall into, and become encysted in the cellular tissue of the new formation, where they remain, in the midst of the tissues, without causing any untoward symptoms, forming an accidental pouch having a fistulous communication with the ruptured gall-bladder. This was the state of matters in our patient of bed 28.

I have now to speak of the treatment of hepatic colic and biliary calculi. But a preliminary question presents itself.

An individual, let us suppose, has biliary calculi. Can we prevent him from having attacks of hepatic colic? When these attacks have declared themselves, can we hope to prevent them, by acting on the concretions which occasion them, so as to reduce them to fragments sufficiently small to traverse the cystic and choledoch ducts, without occasioning disagreeable consequences?

Were I to base my answers to these questions on my personal experience, I should reply in the negative. I am anxious, however, to add that my honorable colleague Dr. Barth, whose scientific authority is of the greatest weight, has published, in illustration of this subject, interesting cases apparently opposed to my views. Dr. Barth, indeed, believes that he has demonstrated that by the aid of particular medicines capable of imparting certain characters to the bile, the calculi in the gall-bladder may be acted on in such a way as to be disintegrated, and their passage into the intestine facilitated sufficiently to prevent hepatic colic being thereby produced.

This proposition has been maintained by other physicians, who, with the object of attaining the same result, have recommended the use of alkalies, which, if they have not, they say, a solvent action on the cholesterin, at least combine with the fatty constituents of the blood, and by saponifying them, carry them away, so as to prevent their being deposited from the bile; the alkalies, and mercury, they say, by dissolving the pus and mucus, prevent the formation of concretions, and disintegrate them if already formed, by depriving them of these two elements, so as to isolate the cholesterin and reduce it to small fragments.

Dissolving the calculi is the principle on which the famous remedy of Durande is based: it consists in giving the patients a mixture of sulphuric ether and essence of turpentine, in the proportion of three parts of ether to two of turpentine. Quite recently some physicians have seriously proposed the internal administration of chloroform, in consequence of M. Gobley having shown that hepatic calculi were more soluble in this than in any other menstruum.

You know, gentlemen, what I think of the application of chemical theories to the physiological operations of the living body. These theories are entirely fallacious, even in the opinion of chemists themselves, in respect at least of the action of ether and turpentine, which in a test-tube and in direct contact with biliary calculi either do not dissolve them at all, or dissolve them very slowly, and which when introduced into the stomach never reach the gall-bladder. We can easily perceive that although solution may be effected in the test-tube by therein bringing the calculus into direct contact with the menstruum at a maximum strength, it would be absurd to suppose that the same end can be accomplished by bringing into contact with the calculi a diluted solvent essentially modified before reaching the liver.

I reject the chemical theories of the solution of hepatic calculi: I reject the chemical theories of the solution of renal calculi by the waters of Contrexéville, Vals, Pougues, or Vichy. Consequently, I deny that medicine can act on either kind of calculi once they are formed: that which medicine can accomplish, is their expulsion by exciting the biliary or urinary secretion, the products of which will tend to entangle the concretions which are formed. And medicine can in a special manner do good by preventing the formation of calculi, by subjecting the patient to a regular plan of treatment, in which alkalies, chloroform, ether, and turpentine, are the most efficacious agents.

So long as the biliary secretions remain normal, there is no greater tendency in the bile to deposit the solid matter which it holds in suspension, than there is in normal urine to deposit the phosphates, oxalates, or uric acid which it contains. Consequently, in the treatment of hepatic colic, our object ought to be to regulate the functions of the liver, just as we endeavor to regulate the functions of the kidney, with a view to prevent the return of nephritic colic.

It is in response to this indication, that the waters of Pougues, Contrexéville, Vichy, Carlsbad, and Vals, are so undoubtedly useful in the treatment of biliary and urinary gravel. Under the influence of this potent medication, if well-directed, patients get rid of the troublesome aptitude which they had contracted. But the benefit, I repeat, does not arise from the alkaline waters dissolving calculi already formed: they act in another way—they modify the constitution of the patient, and perhaps also the organs, upon which they seem to have an action quite peculiar and special.

It is very necessary, however, to beware of abusing the alkaline system of treatment. When too long continued it impairs digestion, and exhausts the constitution. The alkaline remedies I call "long-range" medicines, because they continue to act long after their use has been discontinued. Thus, patients after passing a season at Vichy, Vals, Carlsbad, Pougues, or Contrexéville, remain for from six to ten months, or even longer, under the influence of the medication, and without experiencing any symptoms of their malady. It is, therefore, useless, to say the least of it, to keep up the alkaline treatment without intermission, a practice which I have too often seen.

Here is the manner in which I proceed. When an individual is subject to hepatic colic, I order him to take for eight consecutive days, once a month, one, or at the most two, glasses of the natural mineral alkaline water of Vichy or Pougues. After a week of the alkaline remedy, I direct him to remain for another week without taking any medicine. During the following week, he has to take immediately before each of the two principal meals of the day the capsules containing ether and the capsules containing turpentine of Dr. Clertan; or he may himself fill the gelatinous capsules of Lehuby with ether or turpentine, in the proportion of two-thirds of the former to one-third of the latter. Each capsule contains nearly twelve drops of ether and six drops of turpentine. Of these capsules, the patient takes from two to four; and, according to his tolerance of them, the dose may be increased to ten or twelve in the twenty-four hours. Then follow eight days of abstinence from medicines; after which period comes round the eight days of the alkaline waters. The treatment ought to be continued on this plan for four, five, or six months, even although all the symptoms should have disappeared.

My plan is, as you see, a combination of the use of alkalies with the remedy of Durande; the latter is modified only in respect of the mode of administration. The potion of ether and turpentine, in the form prescribed by Durande, has a very disagreeable taste, and turpentine administered according to his formula so greatly irritates the pharynx and œsophagus as to make its long-continued use impossible. Therefore, gelatinous capsules which are easily swallowed and do not dissolve till they have reached the stomach have undoubted advantages. Many physicians, relying on the experiments of Gobley, now substitute chloroform for ether; there is no difference in the mode of administration. I need not say that the relative proportions of ether and chloroform on the one hand, and of turpentine on the other, may be varied according to the varying aptitudes of patients.

Diet occupies an important place in the treatment of calculous affection of the liver. Gentlemen, while I insist on the necessity of vegetable alimentation, I do not think that it ought to be prescribed to the exclusion of animal food; my opinion is, that there ought to be a judicious combination of animal and vegetable fare. Patients will prefer to eat herbaceous vegetables, avoiding butter, oil, and fatty substances, which are digested with difficulty by persons in whom the liver is at fault.

Regular exercise must also be insisted on; it promotes organic decompositions and compositions, and favors the combustion of the fatty matters of the economy.

When a paroxysm of hepatic colic sets in, I do not know of any really efficacious means of suppressing it. The only remedies which seem to me to procure some relief are ether and chloroform in small doses, belladonna administered internally, frictions over the seat of pain with extract of belladonna, and prolonged general baths.

The inhalation of chloroform produces surprising effects upon some patients. You no doubt remember a woman (bed 7), who on inhaling chloroform for half a minute was immediately relieved from very severe paroxysms of pain. The sedative effect of chloroform will sometimes continue for half an hour; on the return of the colic, the patient must recommence the inhalation, and pursue the same plan till the paroxysm has come to an end.

LECTURE LXXIX.

HYDATID CYSTS OF THE LIVER

Case occurring in a child six years of age.—Two cases in which Hydatid Cysts opened into the Thoracic Cavity.—Hydatids: their mode of development.—Hydatids of the Liver.—Symptoms.—At first, nothing characteristic, except sometimes the appearance of a Tumor in the region of the Liver.—General Symptoms: Disturbance of the Digestive Functions: Tendency to Hemorrhages and Gangrene.—Functional Disturbance of Neighboring Organs.—Hepatitis.—Purulent Infection.—Spontaneous Opening of Cysts into different passages; through the abdominal walls; into the bloodvessels; into the biliary ducts; into the digestive canal; into the pleural cavity; and into the bronchial tubes. TREATMENT: *Simple Puncture with the Exploratory Trocar.—Puncture with the Permanent Canula.—Bégin's Method of Successive Incisions.—Récamier's Method of opening by Caustics.—Opening the Cyst by the Trocar, after establishing adhesions by Acupuncture.—Iodized Injections.*

GENTLEMEN: Within the last few weeks, three cases of hydatid cysts of the liver have come under your notice.

One of them occurred in a little girl, six years of age, who was brought to our out-patients' consulting-room. She had every appearance of a good constitution and perfect health; and according to her mother's account, never had had an illness. For some time, she had complained of pains in the right side, in which situation a certain amount of tumefaction had been perceived. The child, nevertheless, always appeared to be in her usual good health: there was no diminution in her natural cheerfulness: the appetite and digestion continued perfectly normal. The only indication of her being out of sorts was that her sleep, previously sound and calm, was disturbed by nightmare and precordial anxiety.

On examining, I ascertained that there was a tumor, limited on the left by the lower end of the sternum, and projecting under the margin of the costal cartilages. The size might be about that of a hen's egg. The skin over it was natural in color. The tumor was not painful, except when strongly pressed, when slight pain was excited in it. On attentively looking at the tumor, it was observed to be the seat of regular pulsations, which were much more sensible when the finger was applied; they were synchronous with the pulsations of the heart and arteries, and were not movements of expansion, but movements of lifting *en masse*. They ceased when the child was made to stoop forwards. During a deep inspiration the tumor rose, to fall down again during expiration, following, thus, the movements of the diaphragm; this circumstance, combined with the seat, enabled me to say that the tumor was connected with the liver, which did not otherwise appear to be augmented in volume. The tumor evidently contained liquid; deep fluctuation could be felt, and in its upper part there was very distinct crepitation.

I diagnosed a hydatid cyst of the liver—a diagnosis fully confirmed by the exploratory puncture which I caused to be made. The trocar gave exit to a liquid the first part of which was limpid, but the subsequent flow of which was sanguinolent, slightly turbid, containing gelatiniform foreign bodies which were the débris of hydatids.

The child having been at once taken away by her mother, I lost sight of her, so that the case has no other interest than the early age at which the affection presented itself.

You are aware that a hydatid affection—be its seat what it may—and the liver is the most common seat—is hardly ever met with except in individuals who have reached the middle of life or adolescence. It is equally rare in childhood and old age: so true is this remark in respect of early life, that Dr. Davaine,* in the most complete treatise on the subject which has appeared, has not been able to collect more than fourteen cases in subjects under fifteen years of age.

Half of the fourteen were cases of hydatid tumors of the liver developed in persons of twelve, ten, nine, and four years. In one case, which he quotes from Professor Cruveilhier, the subject was a child of *twelve days* old, in which were found only the débris of the cyst, which had opened into the descending colon. The seven other cases recorded in Dr. Davaine's work are cases of hydatids of the heart, pericardium, orbit, canine fossa of superior maxilla, kidneys, and lungs.

If to these fourteen cases you add two cases of hydatids of the thoracic cavity, which Dr. Henri Roger communicated to the Société de Médecine des Hôpitaux de Paris, on 9th October, 1861, another example of hydatids of the liver presented to the Société Anatomique by M. Descroizilles; and, finally, the case of our little patient, you have the sum total of the cases published or known of hydatids in children. They are in number eighteen, and nine of them are cases of hydatid tumors of the liver.

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* DAVAINE: Traité des Entozoaires et des Maladies Vermineuses de l'Homme et des Animaux Domestiques. Paris, 1860.

A few of you only, I presume, have seen the patient regarding whose case I am now going to speak. He was in another service of this hospital; I knew nothing of him when he was living; but I derived some knowledge of his case from being present at his autopsy. The particulars of his case furnished to me are sufficient to show how numerous are the difficulties surrounding the diagnosis of hydatid cysts of the liver, when there has not been an opportunity of observing their development and evolution for at least a certain time.

The man to whom I refer was for two or three months in the Ward Sainte-Jeanne; he presented all the signs of extensive effusion into the right side of the chest. The thoracic development of that side, the complete dulness, the blowing sound, the ægophony, and the broncho-ægophony, left no room to doubt the presence of fluid in the pleural cavity. After some weeks, the patient, feeling better, expressed his wish to return home, although there did not seem to be any real modification of the chest-symptoms. He left the hospital, but was very soon obliged to return. He was then expectorating yellow matter, in which bile could be recognized. Hence was inferred the existence of a communication between the lung and the liver. The symptoms assumed an exceedingly serious character, and proceeded to a fatal issue. During the last days of this man's life, his breath and sputa were horribly fetid, suggesting the idea of hydropneumothorax opening into the bronchi, and the contained fluid was altered in character by the presence of air.

At the autopsy, there was found in the liver an enormous cyst still containing some acephalocysts; it had opened into the bronchial passage through a gangrenous portion of pulmonary tissue. The pleuritic effusion recognized during life still existed; and, curious to relate, there was no communication between this effusion and the bronchi. You now understand why it was impossible, when the patient was alive, to diagnose what had occurred, to know that the pleurisy, occasioned in all probability by the cyst, was, nevertheless, independent of it. You are now also able to understand why it was impossible to ascertain the existence of the cyst of the liver, which occupied its convex surface, without causing that organ in the least degree to be abnormally salient.

I have now to speak of the young man whose case is the occasion of the present lecture. The patient occupied bed 12 of St. Agnes Ward. You recollect my remark in the presence of my honorable colleague, Dr. Legroux, just as I was going to make an exploratory puncture in the right hypochondriac region to complete the diagnosis which I had formed from the nature of the tumor projecting into the abdomen.

I said that there spurted from the puncture made by the exploratory trocar a transparent limpid fluid, which yielded no precipitate of albumen when treated by heat and nitric acid. This fluid contained the débris of hydatids, which by obstructing the canula prevented the flow from being as abundant as it might otherwise have been. This experiment incontestably demonstrated to us that the tumor, apparently belonging to the liver, and which occupied its convex surface, was really a hydatid cyst.

This man, who had just made the Crimean campaign, had been discharged from the army. Before his departure for the East, when in garrison at Auxonne, in the department of Côte-d'Or, he had complained of pains in the right side; but as these pains were dull, and did not wake up except when he made a forced march or had some kind of violent exercise, and as his health remained good, he was able to continue to perform his duty, and, consequently, went to the Crimea. During the whole of that trying campaign he remained at his post, and underwent the severe fatigues to which our expeditionary force was subjected. From time to time, however, the pain in the right side became aggravated; and then it was that the patient perceived a notable swelling in the situation of the pain. This caused him to consult the surgeon of his regiment, who did not attach great importance to what he saw, particularly because the man's general health was unexceptionable.

When the war was at an end, the young man received his discharge. Tormented by the inveteracy of the symptoms, which were complicated by attacks of fever recurring with pretty well-marked periodicity, he resolved to seek admission into an hospital; and thus it was that he came into our hands.

On the occasion of my first examining him, I was struck with the very decided fulness of the right side of the chest, which presented a globular projection, and occupied the whole of the corresponding hypochondrium, extending to the epigastric region. These appearances at once gave characteristic evidence of the existence of the hydatid cyst of the liver.

If not to a hydatid cyst, to what could the tumor in the right side be attributed? No doubt, the amplitude of the chest might be ascribed to thoracic effusion; but then it would be necessary to regard the effusion as completely encysted, as the fulness was exactly circumscribed below. Now, *encysted* is not the most common form of pleurisy. On the other hand, this theory would have made it necessary to assume that the walls of the cyst were so rigid that the pressure of the fluid had more easily overcome the obstacle formed by the thoracic walls than that formed by the lung—an inadmissible hypothesis. In encysted pleurisies, the lung, the mediastinum, the heart, the diaphragm, are pushed out of the way long before the ribs are interfered with. I repeat, moreover, that the bulge of the ribs is uniform throughout the whole extent of the thoracic cage of the corresponding side, and not merely in a limited space as in this case. The view that thoracic effusion existed was, therefore, inadmissible.

The development of the precordial region made it more probable that the affection was intra-abdominal, and its situation being the right side, the liver was clearly pointed to as the seat of disease.

What was the nature of the lesion? Was it cancer? The patient was of an age at which carcinomatous affections seldom occur. His general health seemed to be but slightly affected by the local disease, which, moreover, was so extensive that we could hardly suppose that, were it cancer, it would not have occasioned more severe pains. Finally, with the enormous volume which the tumor presented, we should, in cancer, have felt a nodulated surface of the liver, in place of finding the organ with so even an increase of size.

The fluctuation produced—particularly that produced on exploring the neighboring epigastric region—was not the false fluctuation sometimes met with in cancer, but was evidently due to the presence of a fluid. The progress of the symptoms, and the very disposition of the affected parts, did not allow me to be satisfied with thinking that that fluid was pus, and that we had to do with an abscess of the liver.

In fact, the diagnosis finally settled was that which I formed in the first instance, and which the exploratory puncture had amply confirmed. We found that we had to do with a cyst of the liver: this cyst occupied the convex aspect of the organ. When pressed between the body of the gland (which, supported by the abdominal viscera, was prevented from retreating beyond a certain limit) and the right lung (the elastic force of which also opposed a certain obstacle to its development), the cyst had exerted all its efforts upon the walls of the chest, causing them to bulge out in the manner we had observed.

Having established the diagnosis, the question was, What are the therapeutic indications? In such a case, I could not leave the patient to the unaided efforts of nature; for, although, in certain exceptional circumstances, hydatid cysts of the liver have undergone spontaneous cure, this result has assuredly not occurred in cases similar to that which we had under our observation. Sooner or later, cysts of the large size presented in the case now under consideration, lead to very serious consequences, and the event which we had above all others to fear in our case was rupture of the cyst into the abdominal or thoracic cavity, which would have led to a speedily fatal peritonitis or pleurisy. Interference was obligatory; and the only chance of useful interference was a surgical operation. I therefore proposed to empty the cyst.

Following the established principles of the surgical art, I first endeavored to establish adhesions between the tumor of the liver and the abdominal walls, so as to prevent the fluid from flowing into the peritoneum when I opened the cyst. I shall explain to you, gentlemen, the proceedings adopted in such a case. For the present, that I may keep to the case actually before us, I shall be satisfied merely to mention that I had recourse to multiplied acupuncture for the accomplishment of my object. This kind of acupuncture consists in burying in the tumor—piercing the skin previously protected by a small piece of linen, leather, or caoutchouc—thirty or forty needles arranged in a circle with about half a centimetre between each of them. These needles must be provided with sealing-wax heads.

I was waiting the result of this operation when complications supervened, excited perhaps by the proceedings which I had adopted. The fever, which from the time the man came into the hospital had been showing itself at intervals, all at once assumed a very formidable character, and was accompanied by acute pain in the right side of the chest. I discovered that there was pleurisy with effusion, characterized by dulness in the thoracic region and ægophony, phenomena which, day by day, became more marked. The dulness extended to the infraspinous fossa of the scapula: the ægophony reached as high up as the eighth rib: above, there was bronchophony. Still higher up, fine subcrepitant râles were heard. The expectoration was catarrhal.

On uncovering the chest, a great separation was perceived between the ninth and tenth ribs, with a bulging of the integuments in the same situation. When the patient coughed or made an expiratory effort, there was an increase in the bulging, just as if a liquid were raising the skin. On applying the hand, fluctuation was felt. I asked myself whether this fluctuation was referable to the cyst, which, after separating the muscular fibres of the diaphragm, had passed into the thorax, and had thus simulated the effusion of which I found the signs.

The subcrepitant râles, however, became finer and finer, and were heard on the left as well as on the right side: the sputa assumed the pneumonic character, so that if the chest-symptoms which I had seen become developed on the right side, could up to a certain point have been set down to the account of the cyst, those on the left side could not be similarly accounted for. I said to myself: The acupuncture has produced inflammation in the cyst, and also in the parenchyma of the liver itself, as may be inferred from the subicteric tinge of the skin, which had appeared coincidently with increase of the fever. The inflammation (the cyst having perforated the diaphragm) was propagated to the pleura; and perhaps there was some effusion into the chest in addition to that which I attributed to the cyst.

But I also said, it was surprising that the inflammation propagated to the pleura had respected the peritoneum, for I found no sign of peritonitis. In vain I sought to explain the bronchitis characterized by the subcrepitant râles: the solution of this question was all the more embarrassing that the left as well as the right lung was equally implicated.

The bronchial affection of the right side might be quite well accounted for by supposing that a communication had been established between the cyst and the lung; but no such hypothesis was admissible in respect of the left side.

For every reason, I saw that I could not operate upon the cyst. The case was exceedingly complicated by the thoracic symptoms whatever might be their starting-point. The symptoms continuing stationary, I decided upon opening the tumor, which was salient in the intercostal space. This I did by means of a pretty large trocar, giving issue to purulent fluid containing hydatids.

For two days, nothing occurred which could be regarded as announcing what was going to happen; nor was the patient's state worse. When seen at 4 o'clock in the afternoon, it had been ascertained that there was an abundant discharge of pus from the wound: at ten P.M., and again at midnight, the sister of the service found him very calm; but about one in the morning, he was seized with a fit of coughing which nothing could stop: in the midst of the anxiety and suffocation which accompanied the paroxysms of cough, he exclaimed that he was being suffocated and was dying. A few minutes afterwards he died.

I thought that the cyst had burst into the lung, and that the suffocation had been caused by the hydatids getting into the air-passages. The autopsy shows us that no such thing had occurred; and that disorders had existed which we had failed to recognize during life.

Here is the dead body. By the opening which I made in the intercostal tumor, I introduce a sound which you see passes into the liver through the costal and diaphragmatic pleuræ, which, observe, are firmly united by old adhesions. The liver is enormously enlarged, and I have to traverse its parenchyma to reach the cyst.

Above the diaphragm you observe, there is effusion; and the pleural cavity containing it communicates at one part with the cyst in the liver, and at another with the bronchi, the pulmonary tissue being perforated. Here, therefore, we have hydropneumothorax.

Gentlemen, in connection with the three cases which I have now related, I propose to-day rapidly to sketch the history of hydatid cysts of the liver.

It is not till the beginning of this century that we find in the writings of physicians the first tolerably accurate notions regarding this singular affection. In 1804, Laennec published his work upon vesicular worms, among which he classed hydatids, and called them acephalocysts.* In 1843, Dr. Livois,† a pupil of Dr. Rayer, arrived at the following conclusions: First, that hydatids ought to be excluded from the class of vesicular worms; and second, that they are simple pouches always containing echinococci in number proportionate to the size of the containing pouch.

These conclusions are now generally accepted: but there is not the same concurrence of opinion as to the relations which hydatids bear to echinococci. This point in natural history does not come within the limits of my present subject; and in relation to it, I cannot do better than refer you, for complete information, to the remarkable work of Dr. Davaine, of which I spoke at the beginning of this lecture. Let me merely add, that in the opinion of the savant whom I have just named: "*The hydatid corresponds to a phase in the development of an animal which lives a certain period, and may be produced a certain number of times under the vesicular form: the echinococcus presents a more advanced phase in the development of the same animal.*"

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* LAENNEC: Mémoires de la Société de Médecine de Paris.

† LIVOIS: Recherches sur les Echinocoques chez l'Homme et chez les Animaux.

The most important points upon which we, as physicians, require to be informed, are the phenomena by which hydatids betray their presence in organs, the symptoms to which they give rise, and the treatment which the affection demands.

In man, cysts may be developed in all the parenchymatous organs. The liver seems to be the favorite locality: when cysts are found in other organs, it is very unusual for them to be absent from the liver. After the liver, the next most frequent seat of hydatids is the lung: then come the kidneys, spleen, omentum, brain, and pelvis. There are, according to Dr. Davaine, some examples of their being found in the spinal canal, in the eye, and in the bones. I shall borrow pretty exactly from his work the description which I am now going to give you.

Whatever be the situation which hydatids occupy, they are "in their state of integrity, round vesicles formed of a substance similar to coagulated albumen, containing a limpid fluid, and free from any adhesion or connection with the organ in which they are inclosed. They almost invariably contain echinococci, which are either adherent to their internal surface, or floating free in the hydatid fluid."

Sometimes, they are scarcely visible to the naked eye: at other times, they are as large as the head of a fœtus at the full term. Generally, however, they vary in size between that of a pea, a large hazel-nut, or an orange.

Their form, at first spheroidal or oval, is often modified by the pressure exerted upon them by the parts amid which they originate: their walls, the uniform thickness of which is proportionate to the volume of the vesicle, are colorless, transparent, or of an opaline tint at some points, or throughout a greater or less extent of their surface. Accidental circumstances, such as contact with a colored fluid, the bile for example, may modify the color.

It is not unusual to find along with one large hydatid, several small ones. It is still more common for one large hydatid to contain small hydatids, free in its cavity, or sometimes adherent to its internal or external surface. Originating like granulations, they spring up, increase in size, become hollow, and ere long are detached.

When developed within natural serous cavities, or in veins, hydatids do not seem to have any other envelope than that formed by the walls of the cavity in which they are inclosed: when developed in parenchymata, they are surrounded by an adherent membrane, by a cyst formed at the expense of the cellular tissue of the parenchymatous organ, and the structure of which varies with that of the organ. This membrane, exclusively cellular at first, progressively assumes a fibrous, and fibro-cartilaginous consistence; and in old cysts may be seen disseminated nodules, cretaceous patches, apparently osseous. Their walls vary in thickness according to their age. The cysts are united to the neighboring parts, sometimes by very loose cellular tissue, and sometimes by fibrous adhesions which are solid and difficult to destroy. They may receive bloodvessels which spread over their surface, sometimes penetrate into their interior, and in old cysts reach their inner surface, there assuming a varicose aspect, or an appearance of being surrounded in their course by a real sanguineous injection: the inner surface is then like shagreen, wrinkled, and covered with exudation more or less adherent or thick, while in recent cysts it is white, to a certain extent resembling a serous membrane.

The hydatid cyst is generally globular, and is seldom composed of distinct compartments: when multilocular, this structure is derived from the fusion of many cysts, or by the hydatid cyst having encountered obstacles to its uniform growth, in which case, if the hydatid be single, it sends prolongations into the different compartments.

A very variable number of hydatids may be contained within a single cyst; and they have often been found to amount to five hundred, a thousand—even to seven, eight, or nine thousand. The tumor in such cases may attain a size equal to that of a man's head.

When the cyst only contains a single hydatid, that hydatid generally fills the cyst entirely, and forms a covering to its walls; when the cyst contains several hydatids, there is more or less fluid in which they float.

This fluid, which is transparent like that of hydatids, contains no traces of albumen, and is neither coagulable by heat nor nitric acid. However, when a hydatid cyst has been punctured several times, an albuminous fluid comes from the last punctures; but this is a new product secreted by the cyst itself, and is not the peculiar fluid of the hydatid. The fluid in the principal pouch may, like that of the therein contained hydatids, accidentally assume different colors, a yellow, greenish, or reddish color, from admixture with bile or blood. Not unfrequently, it becomes opaline, muddy, and thick, so as to resemble pus. Indeed, in many cases, it is a purulent fluid originating in inflammation of the cyst: in other cases, the fluid is only purulent in appearance, and is a serosity, holding in suspense sebaceous matter.

This sebaceous matter, which has also been compared to tuberculous matter, is deposited in layers on the internal surface of the cyst when the inclosed hydatid is single, or when, being multiple, its walls are directly applied, without the interposition of any fluid, to the walls of the cyst. By degrees, it grows thicker, assuming the appearance of concrete mastic, or sometimes of chalk. Under such circumstances, the hydatids become reduced to a few membranous shreds, and finally disappear: the echinococci, which have long before been destroyed, are then represented only by their tenacula.

Hydatid tumors thus transformed were, continues Dr. Davaine, formerly called *atheromatous*. The state resembling pus or tubercle, is, according to his view, merely a less advanced stage of atheromatous transformation, of which the cretaceous stage is the last; so that in cases of multiple hydatids, we are able to observe the different phases of change in the same individual.

Gentlemen, a minute or two ago, I reminded you of a fact admitted by all observers, that the liver is the favorite locality chosen by hydatids. It is, moreover, specially regarding *hydatid cysts of the liver*, that I wish to address you upon the present occasion. In one of my lectures [Lecture XXXIV, Vol. I] I spoke to you about hydatids of the lung, in relation to the case of a young man who was in St. Agnes Ward.

In the liver itself, hydatids prefer certain localities: they are more commonly met with in the right than in the left lobe, and in the convex, than in the concave part of the organ. There is sometimes only one; pretty often there are two, three, or more hydatids: but their number seldom exceeds five or six.

The cysts are developed very slowly; and as they often lead to no functional disturbance till they have attained a certain bulk, it is not unusual for the affection, which had never in any way showed itself during life, to be only accidentally discovered after death in persons who have died from totally different diseases. The cysts may take from two to twenty or even thirty years to be developed; and even then, though very large, they may only occasion feelings rather of discomfort, weight, and distension in the right side, than of real pain.

Our patient of St. Agnes Ward told us, that he had been able to go through the Crimean campaign, working at the trenches like his comrades, and taking part in the battles before Sebastopol, without ever having been in hospital. At that time, however, he was feeling a dull pain in the right side, which was sufficiently tumefied to cause, the clothes to press unpleasantly in that situation: this pain was increased by fatigue, but it had never attained great severity.

The painful sensations, then, were felt in the right hypochondrium, the epigastrium, and often in the right shoulder.

The symptoms, you see, are so little characteristic that it is very difficult, if not quite impossible, to diagnose cysts of the liver. But when the cyst has attained a large size, and has caused the side-wall of the abdomen to project, the form of the tumor and its concomitant phenomena often furnish the attentive observer with sufficient diagnostic data.

The tumor, growing slowly, occasioning no sensations strictly entitled to be called pains, accompanied by no state of fever, nor by any disturbance of the general health, is generally globular, and raises up in a uniform manner the thoracic and abdominal parietes beneath which it is situated. On percussion, it yields a dull sound: to pressure with the finger, it offers an elastic resistance, and a feeling of fluctuation, which is deepseated, and sometimes so obscure as to be very difficult to detect. Sometimes also, there is produced a peculiar purring, which has been called hydatid purring [*frémissement hydatique*]. It was first described by Dr. Briançon (of Tournon) in his inaugural thesis:* this is a sign of great value, and when it exists, may be looked upon as pathognomonic. Unfortunately, it is, in general, not to be found, however carefully it may be sought for: frequently, also, after having been perceived for some time, it ceases. Dr. Briançon announced his belief that the intensity of the hydatid purring was proportionate to the quantity of acephalocysts and of fluid contained in the cyst; and that the more numerous the hydatids, and the more abundant the fluid, the more sensible was the purring. Its cause is not quite understood; but this we know, that the purring may exist when there is only a single hydatid, as was ascertained by Professor Jobert in a case of tumor in the region of the deltoid.

I have said that hydatid cysts of the liver are slowly developed, and may sometimes exist in an organ without occasioning any disturbance of the economy. Such cases are reported in Dr. Davaine's work, but they are exceptional; and however slow may be the progress of this as compared with other chronic diseases, it is in reality rather rapid, because, as a general rule, its maximum duration seldom exceeds four or five years.

I have also told you, that cysts, even when of large size, may lead to no other symptoms than dull pains, a feeling of weight, uneasiness, and distension in the affected side. It is difficult, however, to understand how an organ of so much importance as the liver should be more or less implicated for a long period without the occurrence of serious disorders of the economy.

As the tumor, slow in its development, continues limited to a relatively small portion of the organ, the larger unaffected remainder is amply sufficient to perform the functions of the gland. But when nearly the entire liver is invaded by a single cyst (of which cases are reported), or by multiple cysts—when the cysts have rapidly attained a great volume—when this pathological change has caused its effects upon the system—when, finally, by their bulk they impede the passage of the bile through the excretory ducts—the result is the production of serious local and general symptoms.

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* BRIANCON: Essai sur le Diagnostic et le Traitement des Acéphalocystes. Thèse de 1828.

The general disorders produced consist in functional disturbance of the digestive organs. The appetite diminishes, and is lost: digestion is slow and difficult: at intervals, nausea, vomiting, and diarrhœa supervene. Emaciation and loss of color proclaim the cachectic state into which the individual has fallen. There has also been mentioned as occurring in these circumstances *a tendency to hemorrhages*, a very common complication of serious hepatic affections: it occurs in the form of repeated and profuse epistaxis—in women, of attacks of epistaxis and metrorrhagia. According to Dr. Davaine, a *tendency to gangrene* has also been observed. He says that gangrene of the lungs not unfrequently carries off patients who have large cysts in the liver.

Although *jaundice* is an unusual symptom in hydatid cysts of the liver, it is sometimes met with, though some physicians maintain the contrary. It may be more or less intense, more or less deep in color, the result sometimes of inflammation of the substance of the liver itself, sometimes of an obstacle to the passage of the bile through the biliary ducts compressed by a cystic tumor, which compression may also lead to partial or total atrophy of the gall-bladder. Jaundice may also be produced by the hydatids getting into and obliterating the biliary ducts, an occurrence of which I mentioned two examples when lecturing upon hepatic colic: one of these cases, I derived from the practice of my friend Dr. Lasègue, and the other, which I observed in our St. Bernard Ward, is one to which I shall have forthwith to call your attention. There may also be jaundice depending upon complete destruction of the biliary ducts and gall-bladder.

There are also other symptoms, which may be consequent upon mechanical interference with the play of the organs abnormally affected by the presence of hydatid tumors.

The growth of a large cyst in the abdomen may push the stomach out of its place, and press down the intestinal mass to the right iliac crest. Even when the tumor is not very large, it may compress the principal venous trunks, the vena porta and vena cava inferior, thereby producing ascites and œdema of the inferior extremities. These complications, however, are exceptional occurrences in the disease of which I am speaking.

A hydatid cyst forming on the convex surface of the liver, and attaining a great size, will squeeze up the diaphragm into the chest, displacing the lungs and heart: then, again, sometimes, by ascending as high as the second rib and the clavicle, it will simulate a pleuritic effusion, greatly embarrassing the respiration and cardiac circulation. I say nothing at present of those cases in which the tumor, separating or destroying the fibres of the diaphragm, penetrates directly into the pleural cavity: this is a subject to which I shall have to return when I come to discuss the communications which may be formed between hydatid cysts and the respiratory apparatus.

I have mentioned *hepatitis* as one of the complications of the hydatid affection of the liver. This inflammation, more or less acute, more or less extensive, is excited by the presence of a very large cyst, or by the very rapid development of the tumor: it may supervene either accidentally, or as the result of any external violence, such as a muscular effort of the patient, a blow on the seat of the disease, an exploratory puncture, acupuncture, the application of caustic, or any other operation performed with a view to accomplish a cure.

This inflammation often ends in suppuration, and in some cases invades the veins. The phlebitis is sometimes caused by the introduction of septic matter into the bloodvessels. In certain cases, indeed, the hydatid cysts have opened into the vena cava, and in others, they have not only opened into branches of that vein, but have likewise opened into vessels of new formation to be seen ramifying on the surface of the tumor. In these cases, the patients die from the effects of purulent infection.

Whether originating spontaneously, excited by accidental causes or surgical manipulations, the inflammation may remain confined to the cyst, which will then be transformed into a real abscess. This is one of the terminations, perhaps not unusual, of hydatid cysts of the liver.

When suppuration of the cyst takes place, it is either occasioned by a sort of putrid fermentation developed within its cavity by the presence of hydatids killed by an operation, or by the irritation which the instrument has set up in the parts occupied by the tumor, and propagated to its interior.

This suppurative inflammation is always announced by very violent fever, and by acute pains in the region of the malady, in place of the dull pains which alone were previously experienced by the patient. There is also, at the same time, in the majority of cases, a subicteric tinge, and sometimes a very deep-colored jaundice indicating that the parenchyma of the liver is involved in the inflammation.

Gentlemen, I attach great importance to suppurative inflammation as a termination of hydatid cysts of the liver; and in relation to this subject, I must relate a remarkable case communicated to me by Dr. Laboulbène, one of my colleagues in the hospitals; and afterwards I shall recall to your remembrance the details of another case—one to which I have already alluded, and which was observed by you in our clinical wards.

Dr. Laboulbène's patient was a man of fifty-two years of age, who, on 1st September, came into the Hôtel-Dieu, where he was placed in bed 23 of the Sainte-Madeleine Ward. He said that he had been ill for eighteen days, but that up to that date, he had enjoyed excellent health. When at his usual work as a day laborer, he was all at once—without having had a fall or a blow—seized with pain in the transverse arch of the colon. Thrice he purged himself, although he experienced neither gastric uneasiness, diarrhœa, constipation, nor vomiting. As the pain increased in severity so much as to oblige him to keep his bed, he came into the hospital.

On his admission, Dr. Laboulbène was struck with the typhoid aspect of his countenance. His decubitus was dorsal, and his eyes were injected. However, he neither complained of headache, nor of vertigo, even when he was made to sit up; but his tongue was thickly coated, dry, and blackish. He had burning thirst, and no appetite: he had no tendency to vomit. There was some fulness, but not much tension of the abdomen; nor any gurgling in the right iliac fossa. There was no trace of pink lenticular spots. The right hypochondriac region was slightly painful, and there could be detected enlargement of the liver, which ascended almost to the nipple, and descended about four finger-breadths below the false ribs, without presenting any projections or inequalities. The spleen only exceeded by nine or ten centimetres its normal volume. The patient was feverish, his skin was hot, and his pulse 100. Nothing abnormal was discovered in the condition of the heart or lungs.

The sclerotic had a slightly jaundiced tinge; but the urine was natural in color, and contained neither biliverdin, albumen, nor glucose.

Dr. Laboulbène prescribed tonic treatment, of which quinine wine constituted the most important part.

Some days later, the patient had a shivering fit towards evening. The typhoid condition was increasing: diarrhœa had supervened, and continued persistent: the abdomen was tympanitic: and the tongue was as dry as a bit of cork. The rigors in the evening, and the whole symptoms considered collectively, were much more characteristic of purulent infection than of typhoid fever.

On the 8th September, the patient lost a few drops of blood from the nose: his state had been getting worse and worse every day. He died during the night.

At the autopsy, Dr. Laboulbène found the liver enlarged, and adherent to the anterior and inferior surface of the diaphragm. The tissue of the upper part of the right lobe was soft: and in that situation, a cyst, which did not protrude beyond the surface of the organ, profusely discharged a whitish-yellow fluid having a purulent appearance, and containing very numerous bodies resembling transparent capsules of gelatin. Dr. Davaine found that this fluid was composed of white pus-corpuscles and very distinctive mucinous globules.

When the gelatinous bodies were cut in thin slices, they presented in the field of the microscope the characteristic appearances of the peculiar membrane of hydatids. Stratified layers were seen presenting an appearance like the transverse section of superimposed thin strips of oil-silk. Dr. Davaine found no trace of echinococci nor tenacula. The hydatids were destitute of the germinal membrane on which these entozoa germinate before becoming free. In this case, therefore, we had to do with *hydatids arrested in the first stage of their development.*

The internal surface of the cyst was lined by a slightly adherent false membrane, which was tinged with bile in several places. Some parts of this membrane were thick and fibrous. On removing them Dr. Laboulbène detected on the surface and in the thickness of the walls of the cyst, ramifications of veins and biliary ducts. The veins were large and tortuous.

On the anterior surface of the liver, near the falciform ligament, Dr. Laboulbène found several abscesses varying in size and resembling metastatic abscesses. Their internal wall was formed by the tissue of the liver itself. The purulent fluid which they contained was, in some cases, colored by bile, which exuded from bile-ducts, which M. Davaine ascertained opened into some of the abscesses. There were similar purulent collections in the left lobe of the liver.

One of the branches of the vena cava contained an adherent clot which extended into the most minute ramifications of the vessel; and in one of the afferent veins, there was a tubulated false membrane filled with pus, which exuded when the tube was pressed.

Throughout its entire length, the intestine was injected, but not at all ulcerated.

There were no metastatic abscesses in the lungs or spleen.

Should the hydatid cyst, from becoming an abscess, or from attaining a very large size, have a tendency to burst, the time will come when its contained fluid will open a passage for itself through the neighboring tissues. This passage will sometimes be outwards, through the abdominal walls, after the manner of hepatic abscesses and biliary tumors: when the adhesions naturally established between the walls of the tumor and the parietal peritoneum prevent effusion of the fluid into the peritoneal cavity, there exist the conditions which are most favorable for the ultimate cure of the cyst—the very conditions we endeavor to induce artificially as means of treatment.

Hydatid tumors situated on the concave surface of the liver may open spontaneously into the abdomen, either into the peritoneum (occasioning rapidly fatal inflammation), into the bloodvessels, biliary ducts, stomach, or intestinal canal, which latter is the most propitious mode.

I must now occupy a minute or two in calling your attention to these peculiarities.

I shall not revert to the communication existing between the veins and the hydatid cysts and the liver. Let it suffice to tell you the possibility of such an occurrence, and the nature of the results which may ensue.

Hydatid tumors open into the biliary passages by ulceration of their walls, consequent upon compression by the tumor. The vesicles become engaged in the biliary passages. The small hydatids first introduce themselves into the passages, and are then constantly propelled onwards by the bile secreted behind them, so that they pass on from the branches into the large trunks, and ultimately enter the intestinal canal. Should they be very small in size, they are easily expelled; but if larger, they make their way more slowly, and the accumulation of bile which they cause produces dilatation of the passages. This dilatation allows larger hydatids to enter, which in their turn perform the same transit. The same thing occurs which takes place in the case of biliary calculi; the symptoms are nearly the same in respect of pain, jaundice, and pale color of fæces, with this exception, that the pain is less acute. On examining the stools, we find that they contain the débris of acephalocysts, and even entire hydatids. Communication may be established directly between the tumor and the choledoch duct, or between the tumor and the gall-bladder, within which latter hydatids have been found. Like biliary calculi, hydatids engaged in the hepatic or choledoch ducts may cause retention of bile; but in these cases, the nature of the malady usually remains undiscovered till an examination of the evacuations has conclusively established the diagnosis.

It sometimes happens, that the bile passes into the cavity of the tumor through the communication formed between the biliary ducts and the hydatid cysts. At the autopsy, in such cases, we find that the hydatids are broken up, empty, and more or less yellow in color. It is probable, that prolonged contact with the bile causes death of the hydatids; and we shall see that it has been proposed to utilize this fact as a means of cure by injecting ox-bile into the hydatid cysts, a proceeding which (to say the least of it) is strange.

Finally, the spontaneous rupture of hydatid tumors of the liver into the biliary passages does not of necessity lead to fatal complications: it is sometimes a favorable termination of the affection. But fortunate issues are in other ways numerous when a communication has been established between the cysts and the intestinal canal, although in the one case as in the other, evacuation of the fluid contained in the tumor proceeds very slowly, so that several months may be required for its completion; although it may also happen, that the very narrow opening being insufficient to allow the contents of the cyst to pass, other openings form, by which the fluid is discharged simultaneously into the intestine, into some other organ, or externally.

To enable the communication to become established with the stomach—by far the rarest and least favorable mode of opening—or with the duodenum, ascending, or transverse colon—the most usual and also the most favorable mode of opening—it is essential that the tumor should have contracted adhesions with the organs into which it is to discharge itself. If these adhesions have not been formed, there will be danger of the cysts suddenly bursting into the peritoneum, and the production thereby of peritonitis proving fatal within a few hours. These adhesions are the results of an inflammatory process by which the serous coverings of the cysts and the intestinal tubes become glued to one another. They are formed exactly in the same manner as in cases of abscess of the iliac fossa, or of the broad ligament, when the abscess opens into the intestinal canal or bladder. On a future occasion, I shall have to tell you that this is a mode of termination of iliac and pelvic abscesses which is very frequently met with in practice: when left to the unaided efforts of nature, they almost aways undergo spontaneous cure.

The flattening, or disappearance of the tumor which formerly was prominent in the right hypochondriac region and towards the epigastrium, sometimes a peculiar sensation experienced by the patient, the vomiting of purulent matter at first free from fetor, but soon becoming fetid and containing hydatids or the débris of hydatids, are phenomena which indicate that the cyst has opened into the stomach. The presence in the stools of hydatids, or their membranes, announces that the opening has formed into the duodenum or colon. In the latter case, which, I repeat, is the most usual, matters proceed without any complication: if profuse diarrhœa supervene, it does not continue long, and on washing the excreta, there are found not only small hydatids, but sometimes also the mother hydatid, which resembles a false membrane of greater or less dimensions. The evacuation of hydatid cysts of the liver may take place simultaneously both into the stomach and into some other part of the intestinal canal; but again I repeat, that no mode of termination is more propitious than the entire evacuation taking place into the large intestine.

When the hydatid cyst is developed upon the convex surface of the liver, it is, to a greater or less extent, pushed down into the abdominal cavity, where it displaces the mass of intestines: the diaphragm is powerfully squeezed up into the chest. The tumor may in this way be caused to ascend as high as the fourth rib, as the second rib, or even as the clavicle, pushing aside the lung: respiration is greatly embarrassed, because both the diaphragm and the lung are peculiarly impeded in the performance of their functions.

Whether the muscle has remained intact, or whether its fibres, as a consequence of pressure, have disappeared from a more or less extensive surface, a perforation may occur, affording passage to the tumor: the presence of the hydatid cysts in the chest often induces effusion, physical signs of which are furnished by auscultation and percussion, viz., complete dulness, absence of respiratory murmur, and sometimes ægophony, if as very often happens, there exists at the same time, a little effusion into the pleura.

By an attentive examination, however, differences can be established which will assist the diagnosis. Thus, the dulness is usually limited to a certain space: its extent varies so much that on percussing, for example, along the vertebral column, the dulness is found to be complete, whereas beyond it, on the same level, on the lateral wall of the chest, a sonorous sound is heard whatever position the patient is made to assume: or again, it is beyond that that the dulness is absolute, while along the spine, the sound is clear and the vesicular murmur is audible. No doubt, this circumscribed dulness may be indicative of an encysted pleurisy: but in the first place, that is an unusual form of pleurisy, and secondly, it does not give rise to that peculiar kind of deformity which the chest generally acquires in cases of hydatid tumors—a circumscribed globular deformity extending to the hepatic region. There is no longer any ground for hesitation: besides the fact of the liver being down in the abdomen, deepseated fluctuation is perceptible on a line with the margin of the false ribs, particularly when there exists a hydatid purring tremor. An exploratory puncture will speedily remove any remaining doubts as to the nature of the affection.

We can understand that when a true pleuritic effusion takes place under the influence of the irritation caused by the presence of the tumor in the pleural cavity, the differential diagnosis becomes impossible.

In the cases of perforation of the diaphragm to which I have alluded, as being consecutive to disappearance of the muscular fibres under long continued pressure by the hydatid cyst—in these cases, I say, that the tumor of the liver may contract adhesions with the lung, and then, by bursting, form a communication with it. The symptoms of the pulmonary affection, or rather their meaning, is usually not understood until the accidental pulmonary cavity communicates with the bronchial tubes, and so enables the expectoration to furnish diagnostic proof. This proof consists in the presence of hydatids and débris of hydatids; or perhaps, the sputa are mingled with bile, which makes it manifest that the seat of the tumor is in the parenchyma of the liver.

The establishment of this communication between hydatid cysts of the liver and the bronchial tubes is frequently the happy means employed by nature for accomplishing a complete cure, as is shown by numerous cases which have been reported by physicians.

Under other conditions, the propitious termination has taken place in a similar manner,—that is to say by the elimination of the contents of the cyst of the liver through the bronchial tubes—although the tumor after perforating the diaphragm had burst into the pleura.

Nevertheless, gentlemen, rupture of cysts of the liver into the pleural cavity generally gives rise to extremely acute pleurisy, with a great amount of effusion, which declares itself by violent pain in the side, and intense fever soon leading to hydropneumothorax and death.

When matters advance less rapidly, a communication may be established between the cyst and the pleura on the one hand, and between the pleura and the bronchial tubes on the other; and then we encounter all the signs of hydropneumothorax, as in a case which has just come under my observation.

In a word, hydatid cysts of the convex surface of the liver may fill the thorax, and simulate pleuritic effusions: they may open directly into the lung, and may then eliminate their contents through the bronchial tubes: in these cases, the patients frequently recover. The cysts may open into the pleura, and give rise to a rapidly fatal pleurisy; or finally, they may open simultaneously into the pleura, and through the lung into the bronchial tubes, in which case recovery may take place, though generally the issue is fatal.

Already, in one of my lectures on the clinical study of hydatids of the lung, I have had occasion to speak to you of the manner in which hydatid cysts of the liver terminate by opening into the thoracic cavity, and into the lungs.* I had previously related to you a case which occurred in the hospital practice of my colleague Dr. Empis.† To that case, and to those other cases reported by different authors to which I have referred‡ you, I would to-day add one which occurred under your own observation, and which you had an opportunity of attentively studying in our St. Bernard Ward.

The case, interesting in every point of view, is peculiar from presenting an example of a hydatid cyst of the liver opening successively into the biliary passages below the diaphragm and into the pleura. Believing that the description of the case would lose much of its interest by being curtailed, I ask your permission to read its complete history as drawn up by my *chef de clinique*, Dr. Michel Peter.

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* Lecture XXXIV, Volume I, p. 638.

† Lecture XXXII, Volume I, p. 556.

‡ Lecture XXXVI, Volume I, p. 660.

"R. (Amélie), aged 27, was admitted to Dr. Trousseau's wards on the 11th September, 1863. She stated that three weeks ago she had experienced for two days acute pains in the epigastrium, and right hypogastrium, and that consequent upon these pains, jaundice appeared. From that date, the color of the skin became deeper and deeper. From that date, likewise, the pains recurred periodically in paroxysms, once in two days: the paroxysms came on in the evening, and were of about two hours' duration.

"From the time when these attacks showed themselves, the patient suffered from anorexia, dyspepsia, and pains at the epigastrium soon after eating or drinking. She had not had vomiting till within the last three days. In addition to the intense pains which recurred in paroxysms, there was also a constant but quite bearable pain.

"Three or four years ago, after a violent attack of epigastric pains similar to those by which she was seized three weeks ago, and which continued for twelve hours, jaundice set in, and continued for nearly three weeks.

"On admission to the hospital, the patient presented an exceedingly deep yellow color. She was thin: her face indicated suffering: and her general health appeared to be very bad. There was no heat of skin, and the pulse was but little quicker than natural.

"The *diagnosis* was: *Hepatic colic with great consecutive congestion of the liver.*

"During the evening the patient had severe and prolonged rigors, accompanied by increased pain in the hypochondrium and epigastrium. The fever continued during the whole night; and on the 12th September the patient was in a burning fever: the skin was dry, the face flushed, and the pulse 152 in the minute. The hepatic region was the seat of acute pain. On percussion, it was found that the liver was twice its natural size. The patient vomited everything she took. During the evening she had profuse epistaxis.

"*Diagnosis: Hepatitis.*

"Six leeches were applied to the anus, which produced a pretty abundant flow of blood, and afforded marked relief. This relief continued to be felt for three days. There was no diminution, however, in the volume of the liver, which descended almost to the umbilicus, and invaded the entire epigastrium. The fever returned with redoubled severity every evening.

"During the evening of the 14th, the patient was seized with exceedingly severe pain at the base of the right lung. This pain, which embarrassed respiration, extended to the right shoulder. Delirium soon set in, and continued all the evening.

"At the visit on the morning of the 15th, pain in the hypochondrium, irradiating to the shoulder and to the whole of the corresponding thoracic parietes, continued with the same intensity. On auscultation of the chest, however, no morbid signs were discovered. A bath afforded some relief.

"On the 16th the jaundice presented a saffron-yellow color. The state of the patient was most distressing: she groaned continually: there was a continuance of considerable pain, which prevented examination by percussion. Respiration was imperfect and anxious: there was no ægophony. The patient was ordered to take five milligrammes of calomel every hour.

"*Diagnosis: Diaphragmatic pleurisy, the inflammation having been propagated from the convex surface of the liver to the pleura.*

"On the 17th the pain was much less acute. There were heard for the first time a bellows-sound and ægophony in the middle third of the dorsal region. There was dulness in the whole of the inferior third, and skodaic resonance anteriorly in the upper third of the chest.

"On the 18th the pulse was 132, and small. The general condition of the patient was alarming. Dulness had invaded the whole of the right side of the chest posteriorly, and even occupied the infraspinous fossa: anteriorly, the dulness ascended as high as the fourth rib. There was no respiration in the lower half of the chest: in the upper half, and in the neighborhood of the vertebral column, a muffled bellows-sound and ægophony were heard, which were most intense in the supraspinous and infraspinous fossæ. The dulness in the hepatic region continued as formerly; but the pain on percussion had almost entirely disappeared.

"Respiration was excited, without, however, being too frequent. The nose was pinched, the countenance very much changed, and the cheeks cyanosed.

"Next day, the 19th, there was complete dulness posteriorly from the top to the bottom of the chest. It was only in the subclavicular region that there was sufficient resonance, but the resonance was skodaic. A bellows-sound and ægophony were heard posteriorly in the infraspinous fossa and vertebral hollow. Dr. Trousseau demonstrated to those present that there was distinct fluctuation in the intercostal spaces. This fluctation was produced by percussing the plessimeter with the hammer.

"The excessive profusion of the effusion, quite as much as the patient's difficulty of breathing, caused Dr. Trousseau to resolve to have recourse to paracentesis of the chest. The operation was immediately performed by Dr. Peter, the *chef de clinique*. It presented exciting incidents, and for that reason deserves to be described in detail.

"Having incised the skin over the fifth intercostal space in the axillary line, the operator introduced the trocar with a quick thrust. Upon withdrawing the trocar nothing issued from the canula; but on introducing through the canula a blunt probe, some drops of very fetid pus immediately flowed out.

"Dr. Trousseau seeing that there was an impediment to the free flow of the fluid from the chest, substituted for the ordinary canula, one of a much larger calibre. Some spoonfuls of pus then escaped, when the flow stopped: the reintroduction of the blunt probe allowed the exit of a gelatinous substance recognized as a shrunken hydatid. Dr. Trousseau at once concluded that there was *perforation of the diaphragm from rupture of a hydatid cyst of the liver*, with consecutive purulent pleurisy. Nevertheless, to evacuate the fluid from the chest, and relieve the patient, Dr. Trousseau, having adapted a double syringe to the canula, removed rather more than half a litre of pus. The hydatids were constantly choking the canula, so rendering the operation difficult, and at last making it necessary to discontinue before the chest was completely evacuated.

"Having removed the canula, and applied a piece of diachylon plaster to the wound, the patient was left. During the day, delirium supervened, the difficulty of breathing increased, and, in twenty-four hours after the operation, the patient died.

"*Autopsy.*—There was an enormous increase in the volume of the liver: and its left was at least twice as large as its right lobe. At the posterior and upper edge of the right lobe, and projecting from the thoracic, and not from the abdominal parietes, there was a cyst sufficiently large to contain the fist of an adult. Its diaphragmatic peritoneal surface was circumscribed by numerous false membranes which were thick and evidently of very old standing. The cyst was covered by false membrane, which in some places was fibrous, in others atheromatous, and incrusted nearly everywhere by calcareous deposit. It was filled by pus, in which floated shrivelled hydatids. Three perforations were visible:

"One of these perforations opened below the diaphragm, the result of which was the formation of a cavity between the convex surface of the liver and inferior surface of the diaphragm, circumscribed at its periphery by adhesions between the liver and diaphragm.

"The second perforation, the orifice of which might be sufficient to admit the index finger, communicated with the hepatic duct, by which it opened into the choledoch duct, which was very much dilated and contained three small shrivelled hydatids exactly moulded to the shape of the passage they obliterated.

"The third perforation opened into the cavity of the pleura, through a perforation of the diaphragm: it had an inferior orifice sufficient to admit the little finger, and a superior orifice in the form of an elongated slit.

"In the left lobe, were four abscesses, the largest of which was the size of a walnut. They contained a semi-concrete purulent-looking matter, which was found, on being examined by the microscope, to consist of pus-globules and fibrinous granules.

"The hydatids contained in the choledoch duct were situated at the junction of the hepatic and cystic ducts, which caused great dilatation of the latter. The gall-bladder, more than three times its normal size, contained a biliary fluid, oleaginous in consistence and of a very deep-green color. It did not contain any hydatids.

"In the pleural cavity, were nearly two litres of purulent fluid, containing hydatids in all respects similar to those removed by the paracentesis. The trocar had evidently penetrated into the pleura, and not into the cyst: the distance between the cyst and the wound made in the surgical operation showed that it would have been physically impossible to have reached the hepatic cyst by the trocar. Moreover, the diaphragmatic surface of the pleura and base of the lung were covered by thick downy false membranes formed of superimposed layers, which were easily torn, and were evidently of recent formation. They covered nearly the entire lung, as high up as the infraspinous fossa, gradually diminishing in thickness.

"The spleen was very large and did not contain any hydatids.

"Nothing noteworthy was observed in the other organs."

Dr. Peter follows the narrative of this case with some remarks which I wish to bring under your notice and in which I entirely concur.

"It is evident," he says, "that this patient had had three years previously a first attack of hepatic colic, and that even at the commencement of that illness which terminated in death she was suffering from attacks of undoubted hepatic colic; it is not less evident that the attacks of colic had been produced by the successive passage of hydatids through the biliary ducts. The communication between the cyst and the biliary passages produced in the first instance attacks of hepatic colic, which, though not very unusual, are far from being of frequent occurrence. These, however, were not the only consequences.

"1. From the cyst communicating with the hepatic duct, and through it with the choledoch duct, it followed that the cyst communicated indirectly with the small intestine; the result was the enabling the hydatids to make a passage for themselves through the intestine, thus allowing the cyst to be evacuated and the patient to be ultimately cured.*

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* FRERICHS thus mentions the fact: "Symptoms were observed similar to those which accompany the passage of biliary calculi through the choledoch duct." See

"2. From the biliary ducts being in permanent communication with the cyst two results ensued, one relating to the hydatids, and another to the cyst in which they were contained. The hydatids were killed, as often happens.* The other result was inflammation and suppuration of the walls of the cyst, which thus became a large depository of pus. It was in consequence of this inflammation, a partial peritonitis—slow and obscure, but nevertheless continued, and dating back apparently to the first manifestation of the symptoms—that adhesions were formed between the convex surface of the liver and the diaphragm; it is also as a consequence of this same inflammation that the cyst successively burst—1st, below the diaphragm, the purulent fluid being prevented from getting into the peritoneum by the adhesions between the diaphragm and the convex surface of the liver; 2d, through the diaphragm into the pleura, from successive perforation of the walls of the cyst, of the diaphragmatic peritoneum, of the diaphragm itself, and ultimately of the diaphragmatic pleura.†

"In this way, during the life of the hydatids, the cyst in the first instance opened into the biliary passages from their growth, and the necessity for a larger habitation consequent upon their increased size: afterwards, it opened below the diaphragm, and at a still later period, into the pleura, by ulcerative inflammation caused by the introduction of bile into its interior.

"That was not all. As a consequence of the communication between the hydatid cyst and the intestine, the intestinal gases were enabled to penetrate into the interior of the cyst, which explained the fetor, almost stercoraceous, of the fluid which issued from the puncture in the chest. By the existence of this fetor, Dr. Trousseau was at once informed that he had to do with an hepatic cyst which had burst into the pleural cavity. It might have been added, as Dr. Trousseau remarked at a later date, that the cyst was in communication with the intestine. This, as was pointed out by Dr. Trousseau, is an almost pathognomonic sign, upon which, for the future, great stress must be laid.

"It has been established by the observations of Velpeau that the contents of all the purulent collections in the neighborhood of the digestive canal acquire a stercoraceous odor. There is a still stronger reason for this odor existing when the cyst communicates with the digestive canal by an abnormal passage permanently established.

"It is worthy of notice that in spite of the permanent character of the hepatic lesion, the patient had *periodically evening attacks*, and at last epistaxis—symptoms all of which have been pointed out by Dr. Monneret as occurring in diseases of the liver."

Gentlemen, I have now sketched for your information the great outlines of the history of hydatid cysts of the liver; I have described to you the symptoms by which their presence may be recognized and the consequences they may entail; I have stated to you the difficulties which frequently arise in the diagnosis, in respect of which, in many cases, it is impossible to attain absolute certainty except by making an exploratory puncture, and so giving issue to a fluid having special characteristics, or better still, by containing hydatids or débris of hydatids. I now come to discuss the

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p. 593 of the second edition of the French translation of his treatise on "Diseases of the Liver:" Paris, 1866.

* FRERICHS admits that a cure may take place in this manner. See his treatise on "Diseases of the Liver:" *op. cit.*, p. 581.

† DAVAINE: "Traité des Entozoaires," p 478; DAVAINE offers no opinion on the subject. CRUVEILHIER and G. BUDD are of opinion that the introduction of bile into the cyst is the cause of its becoming inflamed.

question of most importance to physicians: What is the best *treatment* of hydatids of the liver?

Though admitted that medico-therapeutic means are indicated in the treatment of the complications which may arise; though granted that narcotic applications, cataplasms, ointments containing opium and belladonna, applied to the affected parts may subdue the violence of the pain and the inflammatory symptoms, medicine is absolutely impotent to cure or even to stay the progress of the affection. Surgery alone can afford useful succor.

I have described to you the progress of the affection, showing how it increases in gravity from day to day, till the tumor becomes so large as at last to burst. Sometimes, the rupture takes place through the skin, and sometimes into the intestinal canal or bronchial tubes; and in these cases, there is a spontaneous cure: unfortunately, cures of this description are too exceptional to be counted upon. In hydatid cysts of the liver, the physician ought always to give a serious prognosis; and be prepared to employ active surgical intervention, though well aware that his intervention will unfortunately cause formidable dangers, and may even lead to a fatal issue at an earlier date than if the case had been left to nature. He, nevertheless, feels compelled to act, because his intervention, however numerous the unfavorable chances may be, affords greater probabilities of radical cure than could be expected from the unaided efforts of nature.

The object of the physician, therefore, is to evacuate the cyst; to adopt measures for the prevention of its again forming; to endeavor, consequently, to destroy the hydatids, which, by their increase in bulk and number, produce the tumor. When once the hydatids are destroyed, the pouch which contained them collapses, and finally disappears.

Several *modes of treatment* suggest themselves for the accomplishment of this object. In the first place there is *simple puncture*.

This puncture is recommended to be made at the most projecting point of the tumor. As a general rule, however, it is preferable to operate in the hypochondriac region. The operation will be more easily performed in that region, as the abdominal walls which the instrument has to traverse are very thin. There will, moreover, be fewer dangers to dread, as the peritoneum alone will be involved, whereas, if we take as our only guide the precept to operate on the most prominent part of the tumor, we may run the risk of wounding several very important organs. Here, let me explain. In our patient of St. Agnes Ward, the hydatid cyst projected from an intercostal space. Now, in some cases of this description, the trocar would have to traverse the skin, parietal pleura, diaphragm, and peritoneum. There would then be a danger of pleurisy and peritonitis as concurrent consequences of the operation.

An *exploratory puncture* is made with a very small trocar. Without the employment of any other means, it may lead to a definitive cure; but it may likewise be the starting-point of fatal complications. This occurrence is quite exceptional; but still, it has occurred; after an exploratory puncture, peritonitis has supervened, and carried off the patient in a few hours. Dr. Moissenet, my colleague at the Hôpital Lariboisière, has related a case of this kind which you will do well to remember.* Let me recommend you to protect yourselves by a statement of possible untoward eventualities, whenever you have occasion to make an exploratory puncture in this class of cases: while you reassure the relatives of the patient as to the general harmlessness of the operation, also warn them as to the untoward accidents which may sometimes occur.

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* MOISSENET: Sur la Ponction avec le Trocar Capillaire, appliquée au traitement des Kystes Hydatiques du Foie. [*Archives Générales de Médecine*, for February, 1859.]

Dr. Boinet* has formulated certain rules, by following which it would always be possible to avoid introducing any of the fluid into the abdominal cavity, a common cause of rapidly fatal peritonitis. He says that it is necessary to be careful, on withdrawing the canula from the trocar, to press back with the fingers the abdominal parietes towards the cyst, so as not to allow any free space to exist between them. This pressure ought to be continued for a minute or two after the operation, and the relative state of the parts should be maintained for some days by means of graduated compresses and a bandage applied round the body.

In the memoir by Dr. Moissenet to which I have just been alluding, the author proposes to apply puncture with the exploratory trocar to the radical treatment of hydatid cysts—a means which hitherto had only been used for diagnostic purposes. He quotes some published cases in which exploratory puncture had led to an ultimate cure; but no one prior to my honorable colleague of the Hôpital Lariboisière had thought of turning these facts to account in practice. This method of treatment will be applicable to those cases in which the cysts "have a manifest tendency to advance externally, and when they impede the free exercise of the organs in the midst of, or in the neighborhood of which they are developed. Under these circumstances, and even in the absence of adhesions to the abdominal parietes, evacuant capillary puncture may be at once resorted to in cases of acephalous cysts, when there is no obstacle to complete evacuation of the cysts. But when from the extreme debility of the patient, and the enormous size of the tumor, it is evident that the contents can only be evacuated little by little, and at intervals, it is necessary to endeavor to produce firm adhesions between the cyst and the abdominal parietes, both for the purpose of performing the capillary puncture without danger to the peritoneum, and of being able afterwards to have recourse, under favorable circumstances, to such other means of treatment as may seen appropriate."

Experience has not given sufficiently decisive testimony in favor of this method of treatment; and I am unable to concur with Dr. Moissenet in believing that evacuant puncture with the trocar is less dangerous than exploratory puncture; for a larger proportion of cases can be adduced in which the exploratory operation was performed without any bad result, as a set-off to the other cases in which its issue was unfortunate.

No one denies the dangerous consequences which may result from making the evacuant puncture with a trocar the canula of which is sufficiently large to allow the passage of fluid, of small hydatids, and débris of hydatids. The *simple incision* is an operation which is always applicable when the tumor projects outwardly in such a way as to threaten to open; for then there is reason to hope that adhesions have become established between the tumor and the walls of the abdomen. Should such adhesions not exist, it will necessarily happen that the fluids contained in the cyst will escape into the peritoneal cavity, if the opening has been made through the abdomen, and into the pleural cavity, if the puncture has been made through the chest: rapidly fatal inflammations are almost inevitably the consequences of the effusions which thus take place.

To avert so formidable a complication, Jobert (de Lamballe) has proposed that several punctures should be made in succession, so as gradually to reduce the size of the tumor, giving the cyst time to contract; or better still, after making the puncture, to leave the canula in its place for twenty-four hours. The canula, which traverses the abdominal parietes and the cyst, determines, at its points of contact, an inflammatory action, which tends to establish adhesions between the parietal and cystic folds of peritoneum. In reality, Jobert, by this proceeding, obtains results similar to those obtained by the plan of Bégin, the plan of Récamier, and by my own method.

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* BOINET: Traitement des Tumeurs Hydatiques du Foie par les Ponctions Capillaires et par les Ponctions suivies d'Injections Iodées. Paris, 1859.

Bégin's proceeding, which I have already explained to you in my lectures on hepatic colic, consists in reaching the tumor by successive incisions.* In the commencement of the first stage of the operation, the skin and muscles are alone implicated; then the aponeurosis being reached, is opened with extreme caution: after which, the peritoneum itself is incised. The cyst is then seen at the bottom of the wound. A dressing is applied, which is kept in place by means of a very tight bandage, and the patient is told to move about as little as possible. When inflammation of the parts has produced adhesions between the cyst and the abdominal parietes, the second stage of the operation is proceeded with: this consists in penetrating the tumor with a large trocar, or (which is preferable) with a bistoury.

Upon the same occasion, I spoke to you of Récamier's method. It is not so quickly performed as Bégin's operation, but what it lacks in rapidity of execution, it gains in safety. In the first stage of the operation, caustic is substituted for a cutting instrument. To the skin of the place where the cyst is to be opened, there is applied potassa fusa, Vienna paste, or the caustic of Filhos, in sufficient quantity to produce an eschar of a certain size, involving at least the thickness of the skin. When this eschar is formed, it is detached, and a new supply of caustic is applied to the bottom of the wound. Proceeding in this way, by successive cauterizations, we at last reach the peritoneum, which we must be careful to respect. Inflammation takes possession of the serous membrane; and by keeping the parts tightly bandaged, we bring the tumor into contact with the abdominal walls in such a way that adhesions are formed between the peritoneal fold covering the cyst, and the peritoneum covering the abdominal walls, in consequence of the former participating in the inflammation of the latter; just as takes place when the plan is followed of leaving the canula in the puncture, or when the other method of successive adhesions is adopted. Thus, there is a possibility of opening and emptying the cyst, without being afraid of causing it to contract upon itself in such a way as to pour its fluid contents into the cavity of the peritoneum.

Récamier's method has been objected to, on the ground that there is often a difficulty in limiting the action of caustics: it has been said that they may give rise to more or less extensive, and even to general peritonitis; while also, an objection of an opposite character has been adduced, to the effect, that the desired result, the production of adhesions, sometimes does not occur. To the latter objection, it may be replied, that in those cases in which adhesions are not produced, the caustic has been badly applied, and has not reached the peritoneum; or that sufficient care has not been taken to maintain the abdominal walls in apposition with the tumor by means of suitably applied pressure.

Multiplied acupuncture is another method by which you have seen me endeavor to obtain adhesions between the cyst and the abdominal walls. I have already told you my manner of employing this method: acupuncture when practiced in this way seems to me to present the advantage of being always accompanied by an inflammation which is circumscribed within the limits of its origin: and another advantage consists in the adhesions being more rapidly obtained, because there exists no necessity, as when Récamier's method is employed, of successively destroying the different layers of the skin before reaching the peritoneum.

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* Begin: Mémoire sur l'Ouverture des Collections Purulentes et autres, développées dans l'Abdomen. Paris, 1830.

When a hydatid cyst has been opened, the suppuration which takes place within it from decomposition of its contents, may become, in some cases, the starting-point of a putrid or purulent infection leading to a fatal issue. To prevent such results, it has been proposed to inject water, or some other fluid, such as alcohol, or tincture of iodine, into the cavity of the opened pouch, with a view to modify the condition of the suppurating surface.

Iodinous injections, used for the first time in the treatment of hydatid cysts of the liver by Dr. Boinet, are those which up to the present date have yielded the best results. Beware, however, of supposing that they constitute an infallible system of treatment. No doubt, a somewhat imposing number of cures has been recorded as resulting from the use of iodinous injections; but still, it must be admitted, that the method is very hazardous even when skilfully employed.

The injections ought to be employed daily: the mixture should consist of equal parts—say fifty grammes—of tincture of iodine and distilled water, with an addition of a certain quantity—say four grammes, of the iodide of potassium. Should symptoms of iodism show themselves, the proportion of distilled water must be increased.

When the walls of the pouch are thin, they contract on themselves, and the cavity is at last closed: but it does not so happen when the walls are thick.

I shall only say a few words regarding *injections of bile*, a mode of treatment recently proposed, and used for the first time, in 1857, by Dr. Auguste Voisin. Before any opinion can be definitively pronounced upon this method of treatment, it will be necessary to have reports of a greater number of cases than has hitherto been recorded. Besides, while it is easy to understand how it is that the injection of iodine is useful, it is not so easy to form a conception of the benefits resulting from the injection of bile, particularly when we bear in mind the terrible consequences of the contact of bile with the peritoneum and subcutaneous cellular tissue.

LECTURE LXXX.

MALIGNANT JAUNDICE.*

Malignant Jaundice [Ictère Grave] is a general disease—totius substantiæ—analogous to Typhoid Fever, and the Bilious Fever of Tropical Climates. —Retention of Bile in the biliary ducts does not constitute Malignant Jaundice.—Typhoid Symptoms at the beginning of the attack.—Yellow color, and Green color of Skin and Conjunctivæ.—Hemorrhages from the mucous membranes: Epistaxis, Gastrorrhagia, Melæna.—Hemorrhages from the Skin: Ecchymosis, Purpura.—Decrease in size of Liver not constant.—Secondary Nervous Symptoms.—Death the most common termination.—Morbid Anatomy: Change in Structure of Liver not constant.—Primary Alteration of Blood.—Notice of the Fatal Jaundice of Infants. —Malignant Jaundice is not Yellow Fever.

GENTLEMEN: In a recent lecture on hepatic colic, I gave you the complete details of the case of a woman, aged fifty, who died in St. Bernard Ward under complications originating in strangulated hernia. You no doubt recollect, gentlemen, that this patient (who was in our wards at several different times for hepatic colic), had a very severe attack of jaundice during the latter weeks of her sojourn in hospital. The biliary ducts must have been obstructed during several weeks at each attack of hepatic colic, for the jaundice was long continued, and the urine had a decided mahogany color, while the fæces presented the characteristic appearances met with under similar circumstances.

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* Ictère Grave: Ictère Malin: Ictère Typhoïde.

This patient died, as I have said, from strangulated hernia; but the posthumous examination, made with very great care by Dr. Benjamin Ball, disclosed among other interesting facts, that there was complete obliteration of the excretory bile-ducts. The cystic duct and the first portion of the choledoch duct were so blended together, amid the surrounding products of inflammation, that it was impossible to conceive the bile flowing into the intestine by the normal passage. It was evident, that if the bile reached the duodenum, its flow must have been through a fistula, of which we could only find the intestinal opening. I do not propose to enter at greater length into the details of this autopsy, which are still fresh in your recollection: my principal object at present is to call your attention to the fact that when during many weeks, and even during many months, there existed a relative if not an absolute retention of bile, the liver presented no morbid structural alteration, the bile certainly continued to be secreted, but, being retained in the excretory ducts, was to a great extent reabsorbed, as was indicated by the gravity and persistency of the jaundice.

It results from the examination of this case, as well as from data founded on many other cases of an analogous character, that the retention of bile does not *necessarily* lead to special poisoning of the economy. Moreover, even the persistent retention of bile does not inevitably produce an organic change in the liver. It is, therefore, impossible to attribute the symptoms of the disease termed "*ictère grave*," "*ictère malin*," "*ictère typhoïde*," to retention of bile.

For at least the last ten years, in England, Germany, and France, the attention of pathologists has been specially directed to a malignant form of jaundice, which almost invariably terminates rapidly in death—a disease evidently general in its nature, existing either with or without structural change of the liver, and having jaundice and numerous hemorrhages as its principal symptoms.

Here is a case in point: A woman, thirty-four years of age, a patient who occupied bed 24 of St. Bernard Ward, had been complaining for seven weeks of pains throughout the body, but particularly in the joints: she had, nevertheless, continued her employment as a journeywoman, but four days before coming into hospital had experienced general discomfort, and remarked that a little blood was mingled with the sputa and nasal mucus. There was a continuance of pain in the continuity of the lower limbs, and particularly in the right haunch and knee; but there were no other signs of rheumatic arthritis. The patient was feverish, and had a small quick pulse. The tongue was foul, though there was no loss of appetite: there had been neither vomiting nor diarrhœa; and the abdomen was soft and sluggish. The liver extended several finger-breadths beyond the false ribs. There was complete sleeplessness. The skin presented a slightly yellow tinge: the urine had a red mahogany color, and became decidedly green on the addition of nitric acid or tincture of iodine.

Next day, the patient complained of suffocative sensations coming on at intervals: examination of the heart and lungs did not furnish any explanation of this intermittent dyspnœa. The articulations were still painful; the fever was high; the skin was moist; and the pulse was 120. The mental condition remained quite clear; but on the following night delirium supervened, and the pulse became more rapid. The jaundice was a little more decided; the sputa again became sanguinolent; though there had been neither epistaxis nor vomiting, the pulse was becoming smaller and smaller; the delirium continued, and the patient sunk on the fourth day after her admission to the hospital, that is to say, on the seventh day from the commencement of the hemorrhages, and the more decided feelings of general discomfort which obliged her to seek for hospital succor.

The autopsy showed that there was no articular lesion—no pus in the joints—not even a bright injected appearance, nor an abundant secretion of synovia in the articulations which had been painful. In no part of the organism could any evidence be found of either purulent collections or phlebitis. The stomach and intestines presented no morbid changes: Peyer's patches had their normal appearance: there were no ulcerations in the large intestine. The lungs were slightly congested at their base, and there were no traces of pneumonia or of hemorrhage. The heart was small; its valves were normal; and there was neither endocarditis nor pericarditis. The brain itself presented no change.

There was no enlargement of the liver; but it was flabby, and of a brownish-yellow tint. When cut, the section presented a deeper color; every trace of the lobular structure of the liver had disappeared. The usual granitic appearance of healthy liver could not be seen. The gall-bladder was contracted, and scarcely contained any bile.

A microscopic examination was made by Dr. Benjamin Ball, at that time *chef de clinique*. He found that the liver presented the following morbid changes:

1. Capillary vessels few in number, and diminished in calibre.
2. Complete disappearance of the normal cells of the parenchyma of the liver: there was no trace even of their débris. In their place were found brown granules of pigmentary matter, some very small, others pretty large, and of a polyhedral form: there was a very great abundance of minute fat-globules, which rapidly disappeared on the addition of a small quantity of ether: at some points, extravasated blood-globules were seen.

Hyperplasia of the cellular tissue presented itself in bands distinctly visible upon examining a thin slice of the tissue.

Clusters of isolated cellules were seen here and there, which were turgid, and infiltrated with fat; but nowhere were there any tyrosis or leucocythæmic globules.

The leading symptoms in this case were a long-continued state of feverishness, which was soon accompanied by feelings of general discomfort, an icteric tint of skin, and slight hemorrhages from the mucous membrane of the nose and bronchial tubes. The patient complained of dyspnœa, which was not accounted for by any lesion of heart or lungs: cerebral symptoms supervened: there existed delirium and excitement. Seven days after the appearance of the hemorrhages and jaundice, the patient died in a comatose state. The muscular and articular pains could not be attributed to the rheumatic diathesis, because the patient had had no sign, present or past, of that diathesis, so that they might be ascribed, as well as the other morbid symptoms, to a general disease, similar in its character to the pyrexiæ. The appearance of hemorrhage and yellow skin, speedily followed by formidable nervous symptoms, gave ground for diagnosing typhoid jaundice, that is to say, a pyrexial malady with a probable organic change in the structure of the liver. The autopsy has verified this diagnosis; but it must be remarked, that in this case there was no *atrophy of the liver*, although the cellules were entirely destroyed. We could not discover any probable cause of the disease.

Here is another case:

A. G., aged forty-six, was of frail health, nervous, hypochondriacal, and timid. He had had frequently slight disturbance of the digestive functions, but nothing to require active medical intervention. In the beginning of July, 1864, without known causes, he had a paroxysm of fever, which subsided about the fifth day, and then, when all was going on well, jaundice, unaccompanied by fever, declared itself. Up to that date nothing had occurred to alarm either the patient or his family; but two days later there was a new attack of fever, and a phlegmon appeared under the jaw-bone on the left side.

The yellowness became very intense: the skin was hot: the pulse ranged between 120 and 130: the tongue became parched, and slight attacks of epistaxis supervened. There was no pain in the region of the liver. The mind was clear. The patient was unable to sleep.

The intensity of the fever, and the acuteness of the pain caused by the phlegmon made the diagnosis difficult. Was the fever excited by the phlegmon, or was its chief cause the hepatic affection? The answer to this twofold question materially influenced the prognosis. In general, when jaundice is accompanied by fever, there is great danger in the case: but this is no longer exactly so when the fever can be ascribed to a concomitant disease, although experience shows that jaundice, even of the simplest kind, is often aggravated by the mere existence of fever, whatever may be the cause of that fever. Therefore although we hoped that matters might assume a more favorable aspect, we could not fail to be exceedingly anxious as to the issue.

The hepatic lesion, however, did not give rise to pain: there were no anxious nervous symptoms: the phlegmon advanced satisfactorily; and although the attacks of epistaxis continued to occur, although the icteric tint of the skin remained as deep as ever, it was justifiable to entertain the pleasing hope that all might still proceed to a favorable termination.

As a rule, the fever due to a phlegmon diminishes, when the phlegmon becomes an abscess. Pus accumulated, fluctuation was felt under the skin, the abscess began to open into the mouth; and yet the fever did not moderate. We were now at the tenth day from the first appearance of the jaundice. One morning, subsultus tendinum was observed, the tongue became more parched, and there was more constitutional excitement: the succeeding night was more agitated: next morning, the subsultus was more violent. In proportion as the nervous symptoms increased in severity, our anxiety as to the issue also increased. During the evening, the patient became weak, and expired in the night without having had delirium or convulsive movements.

This case, gentlemen, places before you the difficulties of the diagnosis when jaundice is complicated with an intercurrent affection.

Dr. Jules Worms lately communicated to me the following case:

A soldier of the 14th regiment of the *voltigeurs de la garde*, twenty-nine years of age, squat, strong, and very healthy, a man said not to have been of intemperate habits, one day when on guard complained of loss of appetite and uncomfortable sensations. Next day, for the benefit of his health, he took a walk with his comrades. On the third day, he felt more unwell, had rigor accompanied by prostration of strength, and was unable to leave his bed. He complained of pains in his limbs; and his comrades observed that he was jaundiced. On the fourth day, the prostration was at its height: bilious vomiting having supervened, he was sent to the Hôpital du Gros Caillou. The admitting physician observed that there was jaundice of medium intensity, and coldness of the skin. The pulse was slow, and hardly appreciable: the physical prostration and loss of mental power were extreme. During the morning of the fifth day of the attack, the patient died, having been in a state of torpor from the previous evening, without having had any evacuation from the stomach or intestines, or any hemorrhage.

The autopsy was made twelve hours after death. The icteric color of the skin was not very intense; but in both eyes, the sclerotic was as yellow as ochre. The body showed no traces of ecchymosis: the gums, however, were covered with a sanguineous crust. Both lungs were congested at the base. The right side of the heart was filled with clotted blood, part of which was hardly coagulated. The small clots were gelatiniform. The left side of the heart contained some fluid grumous blood. There were no coagula in the vessels. The spleen was fourteen centimetres in length, and ten in breadth; it was very soft and friable.

The stomach contained two hundred and fifty grammes of a fluid as black as ink, from which a black matter was deposited, which, on examination by the aid of the microscope, was found to consist wholly of altered blood-globules. Numerous very small ecchymotic patches were disseminated over the surface of the great curvature of the stomach: the mucous membrane was softened. The kidneys did not present any change of structure apparent or histological. The walls of the bladder were normal.

The inferior margin of the liver was found to be three finger-breadths above the costal margin. The liver was of a deep-red color: it was very small, being not larger than the liver of a child. Its transverse diameter was not more than twenty-four centimetres: the perpendicular diameter from the gall-bladder to the point of emergence of the vena cava was fifteen centimetres. The weight of the organ was only nine hundred and forty grammes, the average weight of an adult's liver being fourteen hundred grammes. The gall-bladder contained sixty grammes of very black, thick bile. The capsule of Glisson was in folds, and thickened at certain points, forming an arborescent appearance. The capsule was evidently too large as a covering for the shrunken liver. The hepatic tissue was very soft and friable. The different sections of the liver presented to the naked eye no striking difference from the usual appearances. Yellow points no doubt were visible; but this appearance fell very far short of that marquetry which is characteristic of cirrhosis.

The condition revealed by microscopic examination was as follows. The hepatic cellules were entirely destroyed. It was only here and there that shreds of the cellular envelopes were to be seen. There was a profusion of pigmentary cells. There were also found some nuclei of free cellules, which were beginning to undergo the fatty transformation: but there were scarcely any fat-globules.

The striking features in this case, gentlemen, were the abrupt onset and rapid progress of the disease, and subsequently, the nervous symptoms which proclaimed the existence of a serious organic change. This man's health was remarkably good, when, all at once, he complained of general discomfort and loss of appetite: next day, he had rigors announcing the general affection of the system, and immediately afterwards, he fell into a state of prostration which continued till he died. It was scarcely during more than a few hours before his death that he had a little transient delirium, which soon subsided into a state of torpor. The appearance of the jaundice from the second day of his illness proved that the cause was in the liver; and the typhoid condition of the patient gave certainty to the diagnosis. Although he had had neither nasal, stomachal, nor intestinal hemorrhage, the stomach contained two hundred and fifty grammes of black blood, showing that hemorrhage had preceded death. The blood, moreover, was altered, and the heart contained viscous diffluent blood: the spleen was soft and friable: and the liver presented all the anatomical characters of *diffuse hepatitis*, or the *acute yellow atrophy* of German authors.

These are cases, gentlemen, which Graves and Budd would have described under the name of *malignant jaundice* or *Irish yellow fever;* and which Frerichs introduced into his chapter on *acute atrophy*, or *yellow atrophy* of the liver.* Finally, these cases are similar to those described by my learned colleague Professor Monneret under the name of *essential hemorrhagic jaundice.*

What then are the characteristics of this formidable disease, so variously explained, and the subject of so many theories, though regarded as essentially a *general disease* by the majority of authors?

The icteric color of the skin, in all its shades of intensity, can only be looked upon as a symptom. Jaundice is not a disease; but is on the contrary the expression of numerous states, some of which are compatible with health, whilst others are invariably mortal. But although jaundice is sometimes symptomatic of organic lesion of the liver, it happens occasionally that no structural change can be demonstrated at the autopsy, and that the most competent histological observers are unable to recognize any important modification in the hepatic cellules.

In general, jaundice is only a temporary phenomenon which does not at all disturb the gastro-intestinal functions, nor prevent those in whom it exists from pursuing their ordinary occupations. This is the case even when it arises from a mechanical obstacle to the passage of bile: whatever may be its duration or intensity, there exists no malady (using malady in its vulgar acceptation), though the patients sometimes suffer intense pains, which are generally caused by the difficult passage of biliary calculi through the hepatic ducts. And, gentlemen, bear in mind the remark I made to you at the beginning of this lecture, to the effect, that jaundice may continue for several months without the economy showing any disturbance resulting from the unwonted circulation of bile in the blood. All the organs and all the fluids are colored by the bile; and yet none of these organs or fluids cease to fulfil their physiological functions. Finally, there are pathological conditions in which the excretion of the bile is impossible, as, for example, when the choledoch duct is obliterated: there are cases in which there is an almost complete suppression of the biliary secretion, as in atrophy from cirrhosis; and yet the retention or non-secretion of bile does not, till after long continuance, determine secondary general morbid changes incompatible with life.

But although it be true, as a general proposition, that jaundice is only a symptom of inconsiderable gravity, clinical observation demonstrated to Franciscus Rubæus,† Morgagni,‡ Boerhaave, and Graves,§ that though at first mild, and presenting apparently only the usual conditions, it might suddenly manifest general symptoms terminating in death. Indeed we all know how necessary it is to give a very reserved prognosis in cases of jaundice. As in cases of pleuritic effusion, so in jaundice, one can never say what is to be the termination. Graves states that he was always uneasy as to the issue when nervous symptoms showed themselves, symptoms moreover, which he remarked, were often coincident with a diminished secretion of urine, *the skin and sclerotic retaining their yellow tint:* he also states that, fearing the retention of bile in the system, he has often with success administered diuretics, endeavoring by that proceeding to imitate nature, which eliminates bile by the kidneys when there is an impediment to its exit through the intestinal canal. The Dublin Professor, however, has not described a specially malignant jaundice, and even in his lectures on the "yellow fever of Ireland," he only accords a secondary importance to the yellow color of the skin.

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* **Frerichs**: Traité des Maladies du Foie et des Voies Biliaires: traduit de l'Allemand par L. Duménil et J. Pellagot, 2me édition; 1866.

† **Rubæus** (Franciscus): De Ictero Lethali.

‡ **Morgagni**: Anatomical Letters, 10 and 37.

§ **Graves**: Clinical Lectures.

I must, therefore, refer to observers of the last few years for a description of that special disease which all at once assumes a dangerous aspect, and is almost always mortal. It is only this special condition of disease which I wish to bring under your notice in this lecture, intentionally omitting to notice grave conditions and symptoms which complicate attacks of jaundice of variable duration, and indicative of the existence of biliary obstruction.

Among the many contemporary authors who have studied sudden *typhoid jaundice*, may particularly be mentioned Rokitansky, Henoch, Budd, Dusch, Griesinger, and Frerichs.* But side by side with these English and German authors, it is only fair to cite some French physicians who have specially based their works on clinical observation and general pathology. Let me particularly refer to the treatise of Dr. Monneret, in which the diagnosis and nature of the malady are studied with great sagacity, and in a manner which shows a profound acquaintance with all diseases in which the liver plays a part. Dr. Genouville, in his inaugural thesis, expressed concurrence in the views of Dr. Monneret. Finally, Dr. Blachez has expounded a theory of Gubler, and published interesting details regarding that species of malignant jaundice called *canis.*†

If you read the reports of cases of idiopathic malignant jaundice, or if you observe one well-marked case of that malady, it will be impossible for you not to be struck with its similarity to the symptoms presented in cases of the pyrexiæ. It may be remarked, in fact, that from the beginning of the attack, the whole organism is affected by the disease, as in dothinenteria, small-pox, bilious fevers of tropical climates, and the pernicious fevers of certain countries.

Suddenly, or after some days or weeks of feverish discomfort, the patients complain of general prostration: in vain, they struggle against this state, the malady augments in severity: exhausted in strength, they take to their beds, and soon afterwards, may be detected jaundice, often accompanied by frequent hemorrhages from the skin and mucous membranes.

When the progress of the disease is rapid, there occur, after the third or fourth day, various nervous accidents, such as excitement, convulsions, delirium, and at last profound coma: in which state death takes place. It is important to remember, that in many cases there is a total absence of reaction, scarcely any quickening of the pulse, even a diminution in the temperature of the skin, and no tendency to a critical discharge from the intestines. When the malady continues for several days, repeated hemorrhages take place from the stomach and intestines, after which the vomit and stools consist almost entirely of altered blood. The vomit is black, and identical in composition and aspect with the matter ejected in hæmatemesis from cancer of the stomach. There is sometimes bile in the matter vomited; but that is only observed at the beginning of the attack. The urinary secretion, which is abundant, deposits altered blood-globules; and (according to Frerichs) *leucin* and *tyrosin* could also be detected in the deposit. The same observer has remarked, that in these cases the urine contains a very small quantity of *urea;* and pathological anatomy reveals structural changes in the kidneys similar to those which exist in the liver.

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* **Frerichs**: Practical Treatise on the Diseases of the Liver and Biliary Passages, 2d edition, 1866, p. 164.

† **Blachez** (P.): De l'Ictère Grave: thèse de concours pour l'agrégation: Paris, 1860.

There may be a recurrence of the epistaxis; and the gums are frequently covered by black sordes, and exude blood from their free margins.

Jaundice may either be general, or limited to the upper half of the body, as in a case recently described by Dr. Hecker. The shade of yellow is more or less deep: and the jaundice may either be green or bright yellow, but these variations in color are of no importance—of no more importance than the greenish-brown sometimes observed in the conjunctivæ. Dr. Monneret, and other observers, have remarked, that the appearance of the countenance contrasts with the state of general prostration: the features, so far from presenting a Hippocratic appearance, appear on the contrary quite expanded.

The two leading symptoms then are the hemorrhages and the yellow color: when the patients are roused from their apathy to answer questions, they sometimes complain (as did our patient in St. Bernard Ward) of muscular and articular pains, which are probably nothing more than the febricula so common at the commencement of pyrexiæ and toxæmic diseases. They seldom complain of headache: sometimes they have attacks of suffocation, sighing, and irregular respiration.

On examining the different organs, a variety of phenomena are generally observed, all of which are important. The jaundice solicits first of all an examination of the liver. According to Dr. Frerichs, in the majority of cases, there is pain at the pit of the stomach and in the right hypochondrium, as you see in our patient in St. Agnes Ward (whose history I shall forthwith relate to you). Palpation and percussion increase the pain; while at the same time they generally, but not always, enable us to ascertain that there is considerable *diminution* in the volume of the organ. This assertion of Frerichs is all the more remarkable, that a great many cases occurred in women at the sixth, seventh, and eighth month of pregnancy, a stage of gestation at which we know that there is a notable augmentation in the size of the liver, irrespective altogether of any morbid condition. There is, however, nothing doubtful in the statement of Frerichs, for the majority of the patients died from the disease, and on posthumous examination of their bodies, the liver was found to be atrophied.* But many of the cases of Budd,† Hanlon, and Monneret, as well as the anatomo-pathological history of our patients in St. Bernard and St. Agnes Wards, weaken the generalization of the assertion of the Berlin professor. In these cases, the liver had retained its normal volume; while on the other hand, in the case related by Dr. J. Worms, it had lost nearly one-third both in size and weight.

From all these facts it may be concluded that diminution in the volume of the liver has only a relative importance, inasmuch as it is a symptom which is wanting in numerous cases. But pain in the epigastric and hypogastric regions has a greater degree of importance, as it is almost always met with, and is the consequence of the morbid action which is progressing in the liver, and sometimes in the mucous coat of the stomach, so frequently the seat of profuse hemorrhage. It is surprising that observers have not more often noted the presence of pain in the spleen, pathological anatomy having demonstrated that that organ has often in this disease been the seat of organic changes, or at least of passive congestion, as is observed in septicæmia.

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* **Frerichs**: Traité Pratique des Maladies du Foie: traduit de l'Allemand par les docteurs Louis Duménil et J. Pellagot. 2me édition, Paris, 1866.

† **Budd**: On Diseases of the Liver. Third edition, London.

Notwithstanding the existence of dyspnœa and irregular respiration, nothing morbid was discovered in the lungs or heart; and the autopsy confirmed the conclusions formerly established by clinical experience, viz., the general absence of lesions in these organs. In this way, we become reduced to the necessity of referring these functional disturbances to an affection of the splanchnic nervous system.

It is well, however, to bear in mind that Hecker, following other observers, has explained the sluggishness of the circulation by the existence of fatty degeneration of the muscular fibres of the heart. As to bronchial or pulmonary hemorrhages, they are not the result of a special pathological change in the lungs, but of passive congestion of all the organs.

I have often compared malignant jaundice with the pyrexiæ: yet every pyrexia has as one of its chief elements acceleration of the pulse; while on the contrary, in many cases of malignant jaundice, there is only a slight elevation of the pulse, or sometimes even a fall below the usual standard. I do not include in this remark the quickening of the pulse which occurs just before death, for that is a phenomenon which belongs to the last agony. The inconsiderable frequency of pulse in typhoid jaundice depends upon the general prostration of functional and organic life: the whole economy being prostrate, there is no febrile movement, because there is no tendency to reaction. The original change in the blood, and the disorganization of the liver (when it exists), are of such a nature, that it is impossible for the reflex and sympathetic actions to be produced, on which depend acceleration of pulse, increase of temperature, and in lieu of them, we have sweating or copious diuresis. It appears that the action of the morbid principle, whatever its origin, whether in or external to, the individual, is such that it does not impart any power of reaction to the organism. But notwithstanding this absence of fever, properly so called, malignant jaundice is not the less appropriately grouped with the pyrexiæ, with typhoid fever, and with yellow fever, which are only special septicæmias, with or without special febrile reaction, according to the intensity of the cause, or the resistance of the individual patient.

Likewise, the hemorrhages are passive in typhoid jaundice, and similar in character to those observed in malignant small-pox and malignant scarlatina, the hemorrhage in these pyrexiæ being frequently one of the signs of their malignity.

Many authors believe that in malignant jaundice there are two stages: to one of them, belong jaundice, hemorrhages, and the almost total absence of febrile reaction: to the second stage, belong nervous symptoms, convulsions, delirium, and coma.

When we study the theories of these authors, we find that they regard the nervous symptoms as consequent upon secondary poisoning, or on biliary poisoning of a simple or complex kind. We shall afterwards discuss this interpretation, which I believe to be too absolute: at present, I only wish to remark that, if there exist a first stage, the epiphenomena constituting the second stage seem to me to be the consequences of the progress of the malady. I shall then briefly analyze the different nervous symptoms which supervene—sometimes, on the third or fourth day of the disease, and at other times, not till one or two weeks from the commencement of the jaundice and hemorrhages.

The delirium, though generally of a quiet character, may be accompanied by excitement, according to the habits or idiosyncrasy of the patient: generally, it begins during the night, and continues with only slight intermissions: soon, as in all grave diseases, it is succeeded by coma. I have already said that the patients are seldom attacked by convulsions: general convulsions, when they do occur, have a tendency to assume the eclampsic form. Dr. Monneret observed a case in which they occurred only on one side, but in that case, at the autopsy, it was found, that there was hemorrhagic meningitis.

Death is the usual termination of malignant jaundice; but cases terminating in recovery have been observed by Hanlon and Griffin. In these cases the nervous symptoms were mild, and of short duration. It is not stated whether any critical phenomena occurred in these cases. There are also other recorded cases which demonstrate the possibility of a propitious issue in this disease: Professor Monneret relates the case of a medical student whom he attended, who completely recovered from an attack of idiopathic hemorrhagic jaundice. Baudon relates a case of typhoid jaundice: he says that in this exceptional case, "there was inflammation and enormous enlargement of the parotid gland, which extended from the right temple to below the inferior maxilla. Probably, the parotitis was critical, for from the time of its appearance, there was marked amendment." Dr. Carville's memoir, just published, on an epidemic of malignant jaundice, observed by him in the summer of 1859, would seem to show that typhoid jaundice is a less formidable disease than has been supposed.* Of 47 patients who had idiopathic hemorrhagic jaundice, only 9 died. This was a very much less mortality than that hitherto met with in sporadic malignant jaundice. The mere announcement of this result is so entirely opposed to existing opinions in general pathology, that one is inclined to believe that some important data relating to the cases escaped observation.

Before treating of the nature of the disease, it is necessary to review the principal anatomical lesions of malignant jaundice. So great has been the part assigned in this disease to lesion of the liver, that it has been called "acute yellow atrophy of the liver," and "diffuse hepatitis."

The liver, in truth, is often the seat of a more or less generalized alteration of the hepatic cellules: according to Rokitansky and Frerichs, the walls of the cellules are destroyed: they say, that it is hardly possible to recognize even a few isolated nuclei in the affected parts, a fact which may depend on their being lost amid the amorphous and fatty matter. There must be both destruction of the structure of the cellules and an excess of fat. When the lesion is less advanced, some hepatic cellules remain; but they are infiltrated with fatty matter and biliary pigment. The structural alteration of the liver is never uniform throughout the entire parenchyma, there being healthy portions amid those which are diseased. The alteration of structure advances with the greatest rapidity in the left lobe; and this lobe also often presents a yellow, ochrous appearance on the surface and where a section is made; whereas, in the right lobe, it is only in isolated portions that the altered appearance is seen.

Sometimes, however, the destruction of the cellules is so generalized, that the weight and volume of the organ have decreased to the extent of one-third or even two-thirds: the liver is then diffluent, and of a yellowish color, having no longer the usual granitic appearance: moreover, its fibrous envelope, the capsule of Glisson, appears too large, and exhibits numerous wrinkles.

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* CARVILLE: Archives Générales de Médecine for August, 1864.

The German school of pathologists has erred in applying the term "atrophy" to this alteration of the cellules; and M. Ch. Robin has done well to point out that there exists *destruction*, and not atrophy of the hepatic cellules, with or without change in the volume or consistence of the liver.* The actual state of scientific knowledge does not enable us to make any positive statement as to the condition of the hepatic vessels or parenchyma, except that there is softening and fatty infiltration: sometimes, the softening must to a certain extent be attributed to putrefaction. The biliary ducts are not engorged with bile; and it is only in exceptional cases that the existence in the gall-bladder of a large quantity of bile, has been ascertained.

The facts which I have now stated in relation to the *destruction of the hepatic cellules* are quite in accord with the microscopic examination of the liver of our patients in the St. Bernard and St. Agnes Wards, and of the patient of Dr. J. Worms, whose case was so kindly communicated to me by that gentleman. But let me add, that Hanlon, Griffin, Budd, Monneret, and Robin, have not only ascertained that the physical characters of the liver may be normal, but likewise, that histological examination gives, in many cases, only negative results. It is unnecessary to insist at length upon the importance of such unquestionable scientific facts as those now mentioned; if malignant jaundice can exist without any lesion of the liver, such lesion is not necessary for the production of the disease, and, *à fortiori*, cannot be the cause of the change in the blood.

Lesion of the liver, then, has only an importance which is secondary, and may be compared to the alteration observed in the kidneys, the spleen, and sometimes in the muscular fibres of the heart. Frerichs, and before him Budd and Spaeth, noted a fatty state of the renal parenchyma: the straight and flexuous tubuli, when examined under the microscope, presented a more or less extensive desquamation, and their epithelium was either destroyed or charged with amorphous and fatty matter. Observe that in malignant jaundice, the urine has been found deficient in urea, and the blood to contain it in excess; while also, there existed albuminuria apart altogether from renal hemorrhage. These clinical and anatomical facts show, that sometimes the renal function is at fault, as so often happens likewise in malignant diseases, fevers, and many toxæmic affections.

The spleen is often increased in size, soft, and very friable. Lesions met with in other organs, I regard as of secondary importance: the majority of them depend upon stasis of the blood and hemorrhages into the parenchyma or mucous membrane, phenomena most frequently observed at the base of the lungs or in the mucous membrane of the stomach or intestinal canal. No important lesion of the nervous centres has been observed; and the meningeal hemorrhage, to which I have already referred, was merely an occurrence consequent upon the general hemorrhagic tendency. But the blood which is found in considerable quantity in the venous system, particularly in the venæ cavæ and right side of the heart, has always presented the characters which it exhibits in septæmic affections: it has been of a pitchy, of a dull violet color, diffluent, and has (according to Frerichs) contained leucin and urea in appreciable quantities. The heart was often very flaccid; and Dr. Hecker, as well as Dr. Peter (in a recent autopsy), detected granular and fatty degeneration of the primary muscular fasciculi.

I have just been saying, that atrophy of the liver is a structural change which is likewise met with in numerous cases of blood-poisoning. There are indeed some cases of toxæmia, accompanied by jaundice and numerous hemorrhages from the mucous membranes and into the parenchymata, which in respect of symptoms, not less than lesions, resemble malignant jaundice, and leave, even at the anatomical table, the mind in a hesitating state. The following case, which has just come under our notice in St. Agnes Ward, is one of those calculated to leave the diagnosis in suspense.

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* ROBIN (Charles): Note sur l'état anatomo-pathologique des éléments du foie dans l'ictère grave. [*Mémoires de la Société de Biologie*, 1857, p. 9.]

On July 26th, a man, aged thirty-two, was admitted to our hospital service with slight cyanosis of the face and contraction of the superior extremities. He had been suddenly seized, twenty-four hours previously, with painful cramps in the calves. A little later, very profuse bilious vomiting supervened, which continued all night. Next morning, the cramps had ceased in the legs, and the painful contractions were experienced in the superior extremities.

On the morning of the 27th, you saw the man of whom I am speaking: he then had slight cyanosis of the face, an exceedingly feeble, whispering voice, a very remarkable state of contraction in the superior extremities, particularly in the right, which was the seat of great pain. There was no albumen in the urine.

On the 28th, forty-eight hours from the first manifestation of the symptoms, jaundice appeared: and there was persistent pain in the right hypochondrium.

On the 29th, I remarked to you that there was an increase in the intensity of the jaundice; and that during the morning, slight epistaxis from the right nostril had occurred—the nostril by which, according to Galen, epistaxis takes place in affections of the liver.* The liver was enlarged, so as to pass three finger-breadths beyond the ribs: it was hard, and pressure upon it caused pain. There was acute persistent pain in the epigastrium. The patient said that he felt worse; and his condition was evidently aggravated. The prostration of strength was absolute: the voice was very feeble and languid; and he complained of headache. Throughout the whole spinal column, from the neck to the loins, he experienced very acute pain. There was some contraction of the muscular masses of the neck and spine, which were painful on being pressed; an increase of pain was occasioned by raising the inferior extremities, previously extended, a circumstance which indicated irritation of the spinal cord. The pulse was 112: respiration was loud and quick. Some fine râles were heard at the base of the right lung; but on account of the patient's debility, auscultation was difficult. The remainder of the day was passed in a state of somnolence. The patient had the calm of stupor, but no delirium: he realized the gravity of his condition, and said that he was dying. The vomiting and contractions ceased. His slumber was easily broken by making such pressure on the arm as interrupted the venous circulation. Moreover, he gave indications of suffering acute pain when he was touched on the abdomen or chest, and still more, when touched on the neck. He swallowed slowly, and with difficulty. He died at one in the morning, without a struggle, without having lost consciousness, and without having had any recurrence of nasal or intestinal hemorrhage.

At the *autopsy*, made by Dr. Peter, then my *chef de clinique*, a sanguineous effusion was found at the lower part of the mediastinum: the blood was black, and encysted in the neighborhood of the pericardium. Blood was also infiltrated in the whole extent of the mediastinum. The subjacent cellular tissue of the parietal pleura was infiltrated with blood, so as to give to the entire extent of that membrane a blackish-red color: but there was no hemorrhagic transudation into the pleural cavity. At the summit of the left lung, there was hemorrhagic effusion, encysted by a false membrane: this sanguineous collection measured eight by four centimetres. There was no pleuritic effusion in the left side. Into the right pleura, there was slight sero-sanguinolent exudation. Under the diaphragmatic pleura, on both sides, there was an exudation of blood.

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* "Oportet autem per directum fluere sanguinem, ex dextra quidem nare hepate affecto . . . ex sinistra autem liene." [*De Crisibus*, lib. iii.]

In respect of size, the heart was normal; but it was flabby and very pale. The valves were healthy.

The spleen was twice its natural volume: it was friable, but not diffluent. There was hemorrhage from the hilum.

Submucous and interstitial hemorrhage occupied the whole of the great cul-de-sac of the stomach, without there being any rupture of the mucous membrane or effusion into the interior of the organ, which was in other parts quite healthy. The small intestine was absolutely healthy, except that in its lower third, where psorenteria existed, the solitary glands appeared on the surface like so many millet-seeds. Peyer's glands were healthy.

The liver, which was of augmented volume (particularly in the right lobe), was yellow, anæmic, and fatty.

The kidneys, particularly their cortical portions, were anæmic; in size, they were normal: the suprarenal capsules were healthy.

The pancreas was everywhere injected and friable; there was no hemorrhage into its peripheric cellular tissue.

The parietal peritoneum was much injected, but was not the seat of hemorrhage.

The brain and spinal cord were healthy. Neither the cerebral nor medullary meninges were injected.

Microscopic examination disclosed fatty degeneration of the liver; the hepatic cellules were irregular in shape, being swollen out by little drops of fat, which were soluble in ether. Others were granular, but changed though the cellules were in form, they had not disappeared, as in acute atrophy of the liver. Granular alteration of the kidneys, particularly of the cortical substance, was observed: there was fatty infiltration of the cellules. Granular degeneration of the muscles of the heart, the normal striated arrangement of which was much less evident than usual, at certain points had quite disappeared. The great pectoral muscles had undergone a similar change.

Frequent hemorrhages occur in cases of poisoning with metallic poisons, such as the salts of antimony and arsenic: fatty degeneration is likewise met with in these cases, and in poisoning with phosphorus; but the hemorrhages and fatty degeneration of the liver and kidneys are, as we have seen, the consequences or the characteristic lesions of malignant jaundice. Now, we have been told that this man had been working for a long time in a manufactory of arsenious acid, but had discontinued that employment for a year. It could not, therefore, be supposed that the metallic poison had remained all that time in the system without manifesting its effects. On the other hand, this man was addicted to alcoholic excesses: these habits rendered him subject to a profuse diarrhœa, which explained the psorenteria found at the autopsy; and it is likewise possible that the alcoholism was the cause of a consecutive change in the structure of the liver. Finally, this alteration may itself have been the starting-point of all the symptoms which in the aggregate receive the name of "malignant jaundice."

We now see the category of suppositions to which we can appeal in respect of the symptoms, the etiology, and the lesions of this disease. To elucidate the question of poisoning, I caused the liver and the kidneys to be carefully subjected to a chemical examination, when, as I expected, no trace of arsenic was detected. Nor was there any phosphorus found. It appears that this man succumbed under an affection which commenced with choleraic symptoms and cramps, after which the jaundice supervened. Under those circumstances, my diagnosis was—malignant jaundice. At the autopsy, we found lesions which explained the jaundice and the death, but threw no light upon the genesis of the symptoms.

Malignant jaundice may occur *at all ages;* but the subjects of most of the published cases are adults. I do not know of any case occurring in infants of what can properly be called malignant jaundice. This is a point regarding which I must make some remarks, so that I may not seem to be opposed to a statement made in a contrary sense to the Société Médicale des Hôpitaux. There is, it is true, a kind of jaundice in infants depending on retention of bile, regarding which Dr. Porchat has written an excellent work; and which had before been the topic of very important clinical remarks by Burns, Gardien, Underwood, and Rosen. According to all of these authors, the jaundice of very young infants terminates in death, when there is a continuance of constipation for more than three or four days: they consequently recommend the administration of purgatives with a view to cause the flow of bile into the intestine. But none of the authors whom I have quoted, have described the occurrence of typhoid symptoms in these cases, and most of them are silent on the etiology of the retention of the bile.

Dr. Porchat, in his thesis (1859), after remarking that the etiology of jaundice in very young infants has been little studied, proceeds carefully to investigate the causes of this malady. In a first category of cases, anatomical examination demonstrated to him that the biliary ducts were free, but that the bile, thick and abundant in the gall-bladder, had been unable to reach the duodenum, as was proved by the whitish appearance and chemical analysis of its contents. In a second group of cases, the biliary ducts were absent, or the ductus choledochus was represented by a fibrous cord. In all the cases, the bile was secreted, but not excreted; and consequently, it must have been absorbed by the veins and lymphatics, as was proved by an examination of the blood, urine, and parenchymata, which were tinged by the coloring matter of the bile.

Retention of the bile, not dangerously poisonous to the adult, is sufficient to cause serious consequences to very young infants. Observe, gentlemen, that this is not a matter of mere hypothesis: comparative pathology and the experiments of Claude Bernard* have proved, that retention of bile proves fatal to puppies after some days, while adult dogs do not succumb from complete obstruction of the ductus choledochus. In 1844, Dr. Campbell† published three cases of jaundice complicated with hemorrhage from the cord, and which terminated in death. In these cases, the retention of bile was the consequence either of arrest of development, or of contraction of the excretory apparatus, or possibly of obstruction of the choledoch duct by a biliary concretion. In all the cases, the jaundice was of a very decided character: in two of them, there was umbilical hemorrhage: and in the third case, although there was no hemorrhage from the umbilicus, the infant died in a state of coma after vomiting a fluid resembling coffee-grounds.

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* BERNARD (Claude): Leçons de Physiologie Expérimentale appliquée à la Médecine: Leçons sur les propriétés physiologies et les altérations pathologiques des liquides de l'organisme. 1859.

† CAMPBELL: British and Foreign Medical Review, t. xx, p. 553.

It appears, then, that very serious symptoms and even a fatal termination may occur in biliary retention in very young infants or in infants under one year of age. Cases of this nature may be ranged side by side with the very unusual cases in which the retention of bile in an adult has brought on fatal consequences; but again I say, that malignant jaundice of the adult may exist without lesion of the liver, and is never caused by obstruction of the biliary ducts.

Such being the fact in relation to the fatal jaundice of very young infants, let me remind you that malignant typhoid jaundice is chiefly met with in adults. Mental depression seems to have a large share in producing it; wretchedness and excesses hardly act otherwise than as debilitating causes. Cases of this disease, collected by Hanlon and Griffin, which occurred in persons apparently quite healthy and not seemingly suffering from destitution, appeared to demonstrate to me the influence of middle courses. Two patients were pointed out by M. Hérard, both of whom inhabited the same furnished lodgings, were attacked at an interval of only a few hours, and both died at the Hôpital Lariboisière. I propose to make use of these facts to demonstrate the nature of the disease. As for depression of spirits—and to this point I revert intentionally—there can hardly be any doubt as to the reality of its consequences, particularly if we remember the importance which the older authors attached to low spirits produced by diseases of the stomach and liver, recalling to mind their action on the nervous system. Finally, let me add that the cases of Frerichs,* in opposition to those of Spaeth, seem to show that pregnancy may be a cause of malignant jaundice; for in twenty-two women attacked by it, eleven were with child.

I now come to consider the nature of malignant jaundice; I shall with brevity examine critically some of the different theories which have been advanced. Rokitansky and many others are satisfied to ascertain the anatomical lesion, but Henoch and Dusch have endeavored to explain the destruction of the hepatic cellules by enunciating the double hypothesis of the existence of paralysis of the biliary radicles and of the hepatic vessels. It is not necessary to discuss fancies of this description; let me merely remark that Dusch believes that the elements of the cellule being dissolved, are absorbed, and so produce secondary toxæmia, giving rise to the nervous symptoms which characterize the second stage of malignant jaundice.

Bright was the first to raise the question whether the hepatic lesion was the primitive phenomenon or only a consequence; but he proceeded no farther, and regarded the alteration in the structure of the liver as an inflammation of the organ. Budd, who had observed many cases of different kinds of malignant jaundice, derived from his clinical experience numerous objections to the different theories which had been previously emitted. He stated that malignant jaundice could not be merely an inflammatory affection, because hepatitis was not generally followed by the characteristic symptoms of typhoid jaundice. The general symptoms could not be the consequence of mere retention of bile, because protracted jaundice is constantly being met with unaccompanied by the slightest nervous disturbance. Moreover, as I have already remarked, retention of bile is inadequate to lead to disorganization of the hepatic cellules.

Therefore we may conclude with Budd that typhoid malignant jaundice is not the immediate result of biliary retention, and that the alteration of the hepatic cellules, when it exists, is dependent on a different cause. But, admitting that the retention of bile does not lead to serious consequences in respect either of the liver or of the general system, we must look elsewhere than to the liver (which need not be the seat of any lesion) for the cause of typhoid jaundice.

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* FRERICHS: Op. cit., pp. 261, 262.

The symptoms of typhoid jaundice, their sudden appearance, particularly the signs of moral and physical prostration resembling the symptoms which usher in fevers and toxæmic affections, lead to the belief that a poison or morbific germ which has either entered the organism from without, or been generated within it, is the cause of all these disturbances, which first appear in the nervous system, then in the liver, spleen, kidneys, and heart. Nearly similar phenomena are met with in dothinenteria: a feeling of general discomfort, and a prostration of strength, mark the beginning of the malady, the moment that is to say, when the morbid poison begins to take effect: subsequently, the disease manifests itself with the usual train of symptoms according to the form it assumes; and the special intestinal alterations are secondary results.

The morbific poison may enter the system from without, that is to say, may have its origin in vicious hygienical conditions. The cases of Hanlon and Griffin (quoted by Graves and Budd), and those observed by Dr. Hérard in the Hôpital Lariboisière, justify the conclusion, that the insalubrity of certain habitations, particularly during very hot weather, may originate a morbific element analogous to that which engenders typhoid fever through overcrowding, and to that which causes yellow fever and bilious fever in tropical climates. Here, let me remind you that Dr. Carville's memoir seems to prove that malignant jaundice may occur in an epidemic form.

The source of the morbific poison may be in the individual himself, when he has been long subjected to physical fatigue and moral depression. The equilibrium of the functions may be so modified, that as a sequel of some determining cause of variable nature, the liver may become the seat of a functional lesion, and typhoid jaundice may declare itself. Recall to mind the case of the patient sent in by Dr. Firmin, and who occupied bed 30 of St. Bernard Ward. This woman, aged fifty-three, had become reduced to great misery, through pecuniary losses and great mental distress. For a fortnight preceding her admission to hospital, she had no longer spirit left to work: she felt tired, and had no appetite. Her medical attendant, at his first visit, found that she had both typhoid fever and jaundice. She died in a comatose state some days after admission to hospital. It is probable that unfavorable hygienical conditions and profound sorrow had brought about an alteration in the whole organism; and that from this change proceeded the jaundice and the typhoid symptoms. There was no organic lesion found in the liver or intestinal canal.

In similar cases, the presence of jaundice is sufficient to prove that the hepatic functions are disturbed; and in some patients, the functional disturbance might in itself lead to serious consequences. The passage of the bile into the blood, which does not generally produce much inconvenience, may, in delicate subjects, be the starting-point of fatal consequences. This was observed by Graves, who, to prevent the nervous symptoms liable to occur in such cases, lost no time, as I have already told you, in favoring elimination of the bile by administering diuretics and sudorifics.

There is more reason to dread nervous complications when percussion reveals diminution in the volume of the liver, that is to say, acute yellow atrophy of the organ. Budd, indeed, while he admitted that the poison which engendered the nervous symptoms at the beginning of the icteric attack might suffice to produce secondary nervous symptoms, thought that some share must be attributed to the lesion of the liver, because the disorganized anatomical elements by being absorbed, augment the action of the original poison.

To all the other morbific causes, primary or secondary, must be added disturbance of the function of hepatic hæmatosis, as has been judiciously observed by Professor Monneret. Bile is not the only secretion of the liver. Into the liver opens the entire system of the vena porta: in it, the portal blood demands a special elaboration indicated at its exit from the liver, by increase of its temperature, and its containing hepatic sugar. This process does not arrest the blood-forming function of the liver, as is shown by comparing the blood of the vena porta with that of the subhepatic veins. To form bile, the liver removes from the blood all elements which if not eliminated must prove injurious to the system.

Thus we see that the liver, which Galen regards as an organ of hæmatosis (for the same reason that the lungs are so regarded), cannot be suddenly and permanently annihilated by the disease, without the suppression of function thereby induced acting injuriously upon the composition of the blood, and producing a corresponding influence upon the nervous system.

Now, if the chronic and slowly progressive alterations in the structure of the liver lead to no serious modifications in the composition of the blood and functions of the nervous system, neither can any such modifications arise in the cases in which hepatic hæmatosis is suddenly and permanently suppressed.

It follows, therefore, that malignant or typhoid jaundice (also called *fièvre jaune nostras*) is a general disease, similar in its nature to the pyrexiæ, characterized by disturbance of the nervous system, and by structural changes of the liver, spleen, and heart, which changes are special, though not invariably present. It also follows, that this disease is nearly always fatal; and that it appears to be the consequence of a poisoning originating sometimes in the surrounding médium, and at other times in the organism itself.

Gentlemen, I must enter an emphatic protest against the doctrine that there exists a similarity between malignant jaundice and yellow fever. I had, as you know, an opportunity, in the early part of my career, of seeing a great many cases of yellow fever; and when I afterwards met with cases of malignant jaundice, I had no difficulty whatever in recognizing the difference between the two diseases.

There is one palpable point on which it is easy to agree; that is, the absence of jaundice in yellow fever: in upwards of a thousand yellow fever patients who came under my observation, not one had jaundice. I recollect two soldiers affected by jaundice being admitted to the military hospitals which I visited daily during the epidemic: the attendants in the wards, though they had never been instructed how to discriminate the one disease from the other, were perfectly satisfied that the two classes of patients were not suffering from the then epidemic yellow fever, and the medical officers without the least hesitation adopted the same view: the tint of the skin was itself sufficient to show the existence of an entirely different disease. When a more minute scrutiny had been made into the distinctive phenomena of the two diseases, there remained no longer room for any one to entertain the slightest doubt on the subject.

When we proceed to compare malignant jaundice with yellow fever, looking to the symptoms and anatomical lesions irrespective of the jaundice, it seems surprising that cautious and experienced physicians should regard as similar two affections which present such different characteristics; but the astonishment ceases when it is borne in mind, that those who have instituted this comparison never saw the epidemic, and that those who had studied yellow fever, only knew by books the malignant jaundice with which they compared yellow fever.

In yellow fever, the extreme violence with which the fever sets in, the great severity of the pains in the loins, the indescribable discomfort of which the patients complain, can only be compared to the similar symptoms which usher in an attack of confluent small-pox; while in malignant jaundice, the initial period is rarely invested with such violent characteristics. Hemorrhages from the stomach and intestinal canal are sometimes observed in malignant jaundice, but they are not profuse; whereas, in yellow fever, black vomit, and dejections of a similar kind are seen in nearly all the fatal cases, a circumstance to which the disease owes its names "vomito negro" and "vomito prieto." Black vomit, which in one of the diseases is of rare occurrence and small importance, is a principal characteristic in the other.

It is now held that real jaundice [*jaunisse réelle*]—the affection properly called jaundice—has an almost invariable character in malignant jaundice [*ictère malin*]; and that in it the urine always contains a large quantity of biliverdin, and acquires a still deeper color on the addition of tincture of iodine and nitric acid, whilst icterus [*ictère*] properly so called, is never seen in yellow fever, in which disease the urine is as red as in acute rheumatism, is often suppressed, and never contains the coloring matter of the bile. It is my conviction then, that the similarity which certain authors have endeavored to establish between the two diseases is strained; and can only be maintained by physicians who have not made them a subject of comparative study.

Malignant jaundice can hardly be confounded with any other disease, particularly under the climatic conditions in which I have observed it. Typhoid fever complicated with jaundice, a complication, however, which is very unusual, can hardly lead to a mistake, except during the first days of the attack: but the intensity of the icteric tint, and the various hemorrhages, particularly those from the gastric and intestinal mucous membrane, which take place during the early days of jaundice, do not long allow the nature of the case to remain in doubt.

I should be going beyond the limits of my subject, were I to dwell at length upon the differential diagnosis of malignant jaundice and the malignant bilious fever of tropical countries. It is necessary, however, that I should here mention that the two principal conditions which constitute malignant jaundice, the yellowness and the hemorrhages, are also met with in the bilious fever of the tropics, which is distinctively characterized by its more or less marked remittent type and repeated rigors.

Though in the hepatitis of our climate, as in malignant jaundice, there is a yellow color of the skin, and frequent hemorrhages, we learn, from clinical observation, that in hepatitis the yellow color of the skin is less intense, and shows itself more slowly; and that the hemorrhages are less profuse. On the other hand, the fever is more violent than in typhoid jaundice.

The treatment of idiopathic hemorrhagic jaundice has not generally been successful. It appears that observers have almost always treated the symptoms: hemorrhages have been combated by the mineral and vegetable acids, and the vomiting by iced drinks and gas-charged beverages. Preparations of cinchona have seemed to sustain the strength, and retard the death of the patient. Dr. Hérard prescribed, with temporary benefit, emetic doses of ipecacuan to one patient, who, however, died on the eighth day of the disease.

Perhaps purgatives, and in particular saline purgatives, which have a special action on the liver, may be prescribed with advantage. In pursuing this plan, we should be adopting the treatment of yellow fever successfully followed in America, and should also be responding to the indication supplied by constipation, imitating nature, moreover, which often selects the intestinal mucous membrane as the medium by which to eliminate poisons.

LECTURE LXXXI.

SYPHILIS IN INFANTS.

SYPHILIS IN THE FŒTUS.—*Abortion: Pemphigus: Suppuration of the Thymus Gland and Lungs.*

SYPHILIS IN THE INFANT.—*Pox rarely shows itself before the second week, or after the eighth month.—Slow Form: Subacute Form: Symptoms: Coryza: Fissures: Ulcerations and Mucous Crusts at the mouth, anus, and folds of the skin: Cutaneous Eruptions, Roseola, &c.—Peculiar Tint of the Face: Characteristic Physiognomy of the Syphilitic Infant.—Cachexia.—Visceral Lesions.—Pathogenic Conditions of Syphilis in the Recently Born Infant.*

HEREDITARY SYPHILIS.—*Transmission by the Mother: by the Father.*

ACQUIRED SYPHILIS.—*Syphilis may be transmitted to Nurse by Syphilitic Nursling.—Has the Nurse been infected* in coitu, *or by her Nursling?—Transmission of Syphilis by Vaccination.—Transmission of Syphilis from the Fœtus to the Mother.—Treatment of Congenital Syphilis.*

GENTLEMEN: To-day, I enter upon the discussion of one of the most delicate and most controverted questions in pathology; and athough it is my intention to confine myself within its narrowest limits, I neither wish to conceal from myself nor from you that it is a subject beset on every side with difficulties.

Syphilis, in whatever manner it may be engendered in the system, holds the first rank among those affections the study of which belongs exclusively to clinical science, and does not admit of assistance from any other science. We are shut out from experiments on the lower animals; and experiment limited to the human species is, as you know, liable to a thousand sources of fallacy. It is perhaps by taking syphilis as an example, that one would arrive more certainly at the way to give an account of curative methods and proceedings, and of the scientific value of medicine when left to its own resources.

Impressed though I am with the importance of this study, convinced though I am of its profitable nature, even when problems are discussed for the solution of which the elements do not exist, I have shrunk from pursuing it, possibly from a sense of the magnitude of the task. I shall not, therefore, discourse to you here regarding syphilis in recently born infants: you have had very frequent opportunities of observing it in a state of full development. Nevertheless, while I thus restrict myself, I cannot but look back regretfully upon the field which I have abandoned.

The special hospitals, both those for men and for women, present you with the most ample materials, and your teachers second your inquiring zeal; but in addition to these precious opportunities, there are others, among which are our clinical services, where in place of *the rule* you will find *the exception*—a system of instruction not less necessary.

Observe how the science of syphilis is constituted, and how the doctrinal revolutions accomplished under your eyes are organized. In the hospitals for syphilitic males, laws are laid down with an authority which facts do not oppose. The dogmatism of observers is sincere, because their conclusions are derived from cases occurring under similar conditions. Science is conducted upon this basis, till the time comes when the physicians placed in another sphere, being brought into contact with doubtful cases, raise objections, and at last—as always happens—pass from hesitation to formal opposition.

Were examples required to illustrate a subject so familiar to you, infantile syphilis would itself supply them of the most conclusive kind. In that domain of science, opinions resting upon foundations of the least possible stability have held their ground, because there was an unwillingness to break the unity of a theory; and Hunter himself, despite his great talents, resolutely formulated principles strikingly contradicted by the very facts upon which he sought to establish them.

It would be difficult for you to make a complete study of pulmonary tuberculization in an hospital devoted to phthisis; and in the same way, lock hospitals, in which only confirmed cases of syphilis are received, do not exhaust the category of observable venereal cases.

In these wards, gentlemen, you are placed in the most favorable circumstances for studying syphilis as it occurs in early infancy. In juxtaposition with recently born infants presenting the most characteristic symptoms of the disease, you find others affected with uncertain eruptions, and others, again, in whom there are exanthematous and ulcerous lesions of great gravity, regarding which there cannot exist even a suspicion of syphilitic infection.

It is well that you should profit by this instructive aggregation of cases: and the remarks which I am going to make to you will originate from our surroundings—from the cases which we observe together—for I attach great importance to keep you in a field of observation which is real life. Far from confining myself to the exposition of established opinions, I shall enter upon unsettled questions, upon risks of wrong conclusions, and upon unsolved problems, because there are circumstances in which it is worse than a mistake—it is a fault—for a physician to arrive prematurely at a conclusion.

To begin, let us consider the recently born syphilitic infant, reserving the more obscure questions of pathogeny.

Syphilis may attack the infant *during intra-uterine life;* or it may not manifest itself till after birth, in which latter case, there are no signs at birth of the disease, the germ of which exists, and will develop itself sooner or later. In the second case, the entire process of evolution takes place under our eyes: we see the disease in its very beginning, and we follow it through all its phases. In the first case, on the other hand, the commencement escapes our observation, the progress is uncertain, the diagnosis more dubious, and the description less precise.

The accoucheurs of former times stated, and modern practitioners have confirmed the fact, that syphilis of parents, at least syphilis of the mother, is a frequent cause of *abortion*. It has been said that this predisposition which causes syphilis has been exaggerated. I do not know the conclusions which unattainable statistics might justify, but I do know, and hesitate not to affirm, that when you are called in by a woman in whom premature labor has become habitual, you would do wrong were you not to regard venereal contamination as among the supposable causes of miscarriage, causes of which you ought to make a list on which to adjudicate, before you form your opinion.

But it is not enough, unknown to the family, to have inscribed syphilis as one of the probable causes to be successively eliminated one after another; it behooves you to inquire, whether this cause was uncertain and incomplete, or whether it authorized such a supposition.

To say positively that repeated abortion is often of syphilitic origin is to say too much and too little. Generally, labor takes place very nearly at the full term, and terminates in the birth of a dead child. When the fœtus is born alive, and viable in proportion to its age, when the too early accouchement is entirely attributable to the mother, there is no ground for including syphilis among the conditions, in point of fact so obscure, which have curtailed the duration of pregnancy. Maternal syphilis does not appear, according to the most complete information which we possess, to extend its influence to the vitality of the placenta; and I am not acquainted with any lesion of the placenta possessed of an undoubted specific character.

And yet, we may ask, which we could not have done some years ago, up to what point is this immunity absolute? Syphilis was formerly regarded as comprised within a circle formed by a small number of symptomatic indications localized in the skin, or in the mucous membranes conterminous with the skin, and extending, but slowly, to the osseous tissue: it was supposed not to invade the splanchnic organs. The placenta did not seem to be more susceptible than the liver, spleen, or lungs of a venereal degeneration which had not been directly observed, and was in contradiction to the ordinary laws of the disease.

In the present day, a new direction has been given to scientific inquiry, and the impossible has ceased to exist: to the alterations in the integuments and bones, we have now to add parenchymatous alterations discovered by the aid of the microscope, and the existence of which had been foreseen by clinical observers. In this department, nearly everything yet remains to be done. I call your attention to paths hitherto scarcely explored, being convinced that you will not think, like some persons, that it is necessary to wait until the truth has verified research.

Some problems remain for future elucidation: but it is a well-established fact, that the cause of syphilitic abortion is the death of the fœtus *in utero*.

Does there exist any known symptom characteristic of the affection which has deprived the fœtus of life?—For my part, I admit that I cannot point out to you any really significant lesion; and I incline to the belief, that those authors who have been most explicit in a contrary sense would have done better had they imitated my reserve. You will be told about the general appearance of the still-born child, the color of its integuments, the maceration of its epidermis, the ulcers on its body, and the hideous deformities which it presents. The more graphic the picture which is drawn, the more necessary is it to be distrustful of its representations. Once on a time, physicians participated with men of the world in regarding all rebellious and obstinate ulcers as venereal: it is from that period that we date descriptions undoubtedly destined to impart disastrous consequences to syphilis.

An infant is born at or before the full term; it lives, but has contracted, during fœtal life, a malady which at birth was already in process of evolution, and destined to prove fatal. It is to this syphilis, developed in the infant before its birth, and continuing during the first days of extra-uterine life, that some authors have assigned characters so precise as to enable them to base a diagnosis on them: I refer to pemphigus, to alterations of the thymus gland, and to pulmonary lesions.

For two reasons, I shall be brief on this subject; first, because you have very few opportunities in this hospital of seeing infants at birth, and it is my plan to lecture on clinical cases which you have facilities for observing: and in the second place, because the discussions regarding these specific manifestations are of a too recent date for me to require to bring them before you at great length.

None of these lesions are met with at a more advanced age, when syphilis shows itself by many varied and indisputable signs: they thus possess the twofold specificity of being of a venereal character, and peculiar to the fœtus.

Pemphigus makes its appearance within so few hours after birth, that the preparatory stage has evidently been proceeding during intra-uterine life. The bullæ, which are chiefly situated on the palms of the hands and soles of the feet, form rapidly, become filled with semi-purulent liquid, burst, and then give place to ill-conditioned ulcerations. The surrounding parts have a bluish color, like most of the cutaneous inflammations of recently born infants. The general health is radically impaired; and there appear the usual signs of infantile cachexia, which as you know, almost invariably terminate in death, whatever may have been their origin.

It cannot be disputed, that pemphigus is met with in very young infants: it is an equally well-established fact, that in them, as in adults, this affection is the expression of a deepseated and radical disturbance of the system: the only question open to discussion is, whether this pemphigus is syphilitic. On the one side, it is objected, that the bullæ have no specific character, neither in themselves individually, nor in the manner in which they are grouped—that pemphigus is one of the rarest complications of confirmed syphilis—and that all the causes of pemphigus find their legitimate, and so to speak, classical place in puny recently born infants. These objections have a value which cannot be ignored: they are met by an argument which, though indirect in its nature, is not the less important. "In most of the cases in which pemphigus exists," says Professor Paul Dubois, "I have been able to verify the signs of former syphilis in the parents, or to obtain from them convincing evidence that they had the disease."* Other observers declare that they have been less successful in their search for evidence of this kind, although they have pursued similar inquiries. The settlement of the question, therefore, must depend upon the vigor of the statistical investigation.

I have sometimes mentioned to you a fact, which I had the opportunity of observing along with one of my professional colleagues. He called me in to see a child, about fifteen days old, in which the most precise signs of syphilis existed. The father had had a Hunterian chancre, and secondary symptoms, of which he believed that he was perfectly cured. I told him plainly that his child had congenital syphilis: I asked him whether he himself had not still some traces of syphilis. He replied in the negative; but nevertheless, I proceeded to investigate the case minutely, and discovered, without difficulty, exostoses of the tibia, which left no doubt as to the disease. He then told me, that fifteen months previously, his wife had been delivered, at the seventh month, of a still-born child, which he had preserved in spirits of wine. He showed me the little dead body; and on its skin, I distinctly perceived numerous traces of pemphigus.

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* Dubois (Paul): Syphilis Congénitale. [*Bulletin de l'Académie de Médecine*, 1851; t. xvi, p. 980.]

So far as I was concerned, this demonstration did not amount to more than the establishing of a probability; and several physicians who participated in this indecision finally accepted a compromise. They considered that maternal syphilis had determined a sort of cachexia in the fœtus, which had led to an eruption of bullæ which was not specific. By accepting this too facile hypothesis, you will imprudently open a door which you will with difficulty be able to close. Support is thereby given to those who see in the cachectic diseases of early infancy, certain derivations and metamorphoses of—to use the fashionable phrase—ancestral syphilis [*syphilis des ascendants*]. This is a dangerous direction for theory to take—one which leads, and has led, to rash generalizations, in which imagination is substituted for observation, and all morbid manifestations are merged in one arbitrary pathogenesis.

Suppuration of the thymus, and *suppuration of the lungs*, have furnished two observers, whose sagacity is known to you, with the materials for interesting monographs. These lesions, which sometimes exist separately, and sometimes together, are of rare occurrence, and their relation to the health of the father or mother is still matter of uncertainty.

That is the limit of our knowledge of syphilis in the fœtus. I pass over, without remark, alterations of the liver, of which I shall afterwards have to speak to you, and syphilitic peritonitis, regarding which Simpson has said a few words.* The only manifestations attributed to intra-uterine syphilitic taint are first of all abortion, then pemphigus, suppuration of the lungs, and still more, suppuration of the thymus.

When the last vestiges of intra-uterine life have disappeared, when the infant in virtue of respiration, and particularly by its changed mode of alimentation, has passed into a new life, syphilis, till then absolutely latent, makes its existence known by signs which easily escape detection. I propose, therefore, to direct your attention to a somewhat more detailed account of symptoms.

It is a law which holds good in the whole domain of medicine, that little circumstances have often a leading significance: but this fact is specially true in respect of syphilis, if any difference of degree as to the truth of the law can be admitted. In infantile syphilis, the diagnosis can be established only by patient inquiry into minute circumstances; and in cases of this kind, descriptions are good only when they are long.

In the infant, which in coming into the world bore no certain traces of venereal infection, *pox rarely develops itself before the second week;* and it is very exceptional for the disease to make its first appearance after the eighth month. Usually, it appears about the fourteenth or fifteenth day after birth. These dates, which I gave so far back as 1847, in a memoir which I published conjointly with my friend Dr. Lasègue,† have been confirmed by all subsequent observers, and are in harmony with those indicated by our predecessors; if we except cases of doubtful authenticity.

The manifestation of the symptoms, therefore, is preceded by a more or less prolonged incubation, during which the physician cannot discover the slightest indication of the impending malady. I am well aware that the physicians of the Hôpital des Enfants Malades (to whom the science of infantile syphilis owes so much useful information) believe in a sort of premonitory cachexia. There is no such condition. The doomed infant either has or has not (as the case may be) all the attributes of robust health up to the day on which the first symptoms declare themselves. I go still farther, and maintain that vigorous health does not always exercise the influence attributed to it, upon the progress of syphilis. We see infants, to all appearance in vigorous health, rapidly decline under the stroke of syphilis, while others, more puny, bear up under a similar shock. In the infant, as in the adult, two influences are in operation, viz., the activity of the disease, and the resistance of the patient; but it is difficult to estimate the power of resistance, till we know the extent of the proofs to which it is subjected.

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* Simpson (James Y.): On Peritonitis in the Fœtus.

† Trousseau et Lasègue: Archives Générales de Médecine, 1847.

But to make this possible, it would be necessary for the infants to be of the same age, identical in apparent health, and affected with syphilis in exactly the same degree of intensity. In some, irrespective of treatment, evolution is slow, passive, essentially chronic from the first: in others, it is active, subacute, and semi-febrile: the appearance of the patient is greatly changed, complications increase, which ultimately induce a secondary derangement of health more dangerous than the original malady.

Bear in mind, gentlemen, that this entirely clinical diversity of evolution is capable of supplying in respect of treatment important counter-indications; that it renders necessary certain therapeutic reservations; and that it explains why the treatment of syphilis in infants is not so commonplace as in adults.

The signs by which the constitutional affections are manifested are numerous, and do not occur in an order sufficiently precise to authorize a chronological classification. Many symptoms are wanting, and their chain of sequence is full of chance occurrences and contradictions.

Another mode of classification, however, is available. Some of the symptoms have an unambiguous meaning, while the nature of others is open to considerable uncertainty. It is particularly to the first of these classes that I wish to direct your attention.

Affections of mucous membranes conterminous with the skin are not uncommon in new-born infants. In most of the eruptions classified together under the name, more convenient than scientific, of glanders [*gourmes*], there are often lesions of the mucous surfaces which are accessible to sight: but this can only occur when there is a very confluent state of the exanthematous eruption. It begins in the skin, whence it spreads. In syphilitic infants, the mucous membranes may be, and really are, affected, although the eruption be little apparent, not seated in their vicinity, nor even externally manifested.

Coryza is one of the signs which appear earliest, and also one of those which have been best studied. The infant breathes with increasing difficulty by the nostrils: through the insufficiency of nasal respiration, it is embarrassed in sucking. Up to this point, there is nothing special to distinguish the specific from the other forms of coryza.

Soon, there is a running from the nose, and a few drops of blood exude, but there is no true epistaxis. The secretion becomes more and more sanguinolent, without being profuse: it irritates the alæ of the nose and the upper lip, causing ulcerations which become covered with crusts where dried by the external air. On making a more attentive examination, there will often be found at the angles of the alæ of the nose small ulcerated fissures, already characteristic, inasmuch as they exactly reproduce the special aspect of the fissures seen in the commissures of the lips.

At a more advanced stage of the disease, the bones lose their support, the cartilages become eroded, without being perforated, the nose flattens, and gets a squashed appearance. The upper part, little prominent in infants, spreads out, giving a strange effect to the face. The lesions, however, do not generally proceed to this extreme degree: the progress of the structural change generally stops at its second stage: sometimes, it progresses by fits and starts, just as chronic eruptions alternately augment and diminish. The opportunities after death are only too frequent of ascertaining the true nature, and different degrees of the lesions of the nasal mucous membrane. Coryza is, almost in every case, the earliest sign of infantile syphilis.

The mucous membrane of the lips and mouth is, perhaps, less frequently attacked than that of the nose; but to compensate for this the symptoms are more obvious. We find at the orifice of the mouth, fissures, in more or less proximity to one another, radiating in the course of the natural folds of the integuments; and also rounded ulcerations, true *mucous crusts*, having the same seat, though not exactly the same aspect as in the adult. The striæ have a characteristic appearance, and are such as I have never seen except in syphilis: in proportion as their situation is distant from the labial mucous membrane, so is their size smaller. At their bottom they have an appearance which is more or less bright red, bleeding, and gristly: their edges are finely fringed, and blackened by adhering coagulated blood. Tenacious, like all fissures which occupy constantly elastic parts, they often leave indelible cicatrices after recovery. I have seen both young men and young women at the age of puberty who still had these cicatrices, stigmata, the nature of which they did not suspect.

The mucous crusts are hardly ever met with except at the commissure of the lips. They are small, thick, protruding, whitish, and have, at first sight, a diphtheritic appearance. They seldom invade the cheek. Originating within a fissure, and becoming developed consequent upon an irritation irrespective of syphilis, there do not exist the same reasons for their occurring within the mouth as in adults.

The *mucous membrane of the pharynx* ought to be carefully examined, though, very often, it is not affected. By never omitting in any case to inspect the back part of the pharynx, you will find, more frequently than the statements of authors would lead you to suppose, mucous plates occupying the anterior or posterior pillars, but never the posterior wall of the pharynx. On the pharynx their appearance is not the same as at the angle of the lips: they protrude little, are very superficial, and having no exudation on the surface, are not liable to be mistaken for diphtheritic patches.

In every case in which it is possible to suppose that the infection was received from the nipple of the nurse, importance has naturally been attached to the state of the nursling's mouth and lips. An impression has existed that the localization of the symptoms, or at least their predominance at the points of contagion, must furnish valuable information. I cannot too strongly warn you against the danger of being swayed by that notion; for although you may derive advantageous indications, you may also be led into the most regrettable mistakes by giving way to the belief in question. By an exactly similar tendency it has often been concluded, from the concentration of lesions around the genital organs, without any other evidence, that young children have received venéreal inoculation in shameful assaults. The infection of the nursling by the nurse may take place by a single erosion; and it is not maintained that the disease has numerous centres of origin because the inoculated lesions belong to the secondary period. It is only too certain that a single chancre is sufficient to admit pox into the system, just as a labial ulceration may suffice to do the same. Many children affected with hereditary syphilis have had one or more mucous patches on the lips; but what conclusion are we to draw from the fact that one or several mucous tubercles are seated at the buccal orifice?

Particularly bear in mind—and the fact is one of which I should not speak were it not often forgotten—bear in mind, that in the case of the infant, no more than in the case of the adult, does the number of secondary lesions in any particular part imply that the infection has entered by that part. The anatomical disposition of the anal orifice is similar to that of the mouth; and, consequently, you will find the same lesions as in the mouth—fissures, rhagades, oozings, and consecutive ulcerations; but I ought to mention that the affections are generally less extensive and less severe around the anus.

There are other parts where the infant's skin seems to resemble, in its structure, the mucous surfaces, and where, when under a pathological influence, it nearly assumes their characters. I refer to those folds in the integument, so deep in fat children, which become ulcerated, or at least irritated by rubbing, and yet more by the infiltration of excrementitial fluids, and which so specially demand precautions as to cleanliness: in these situations, fissures and ulcerations are often produced. It is prudent to beware of being misled by the deceitful appearance of simple ulcers; and it is on the hands and feet that these lesions are characteristic. I shall consider them in relation to cutaneous eruptions.

I have passed in review the venereal alterations of the mucous membranes: I have assigned them the first place, because they are more expressive, and particularly because they clear up the diagnosis by their frequency, their specialties, and their importance in relation to inoculation. The constitution of women has often been compared to that of children: I cannot venture to say to what extent this comparison is valid, but here it is justifiable to some small extent. In women, as in recently born infants, the mucous membranes are, much more frequently than in men, the chosen seat of syphilitic lesions. Suffice it to remind you of the remarkable frequency of syphilitic sore throat in women.

Among the *cutaneous eruptions* properly so called, *roseola* is generally the first to show itself: it is also very frequently the first to appear in recently born children. More or less generalized, occupying by preference the inferior extremities, not seen frequently on the face, it manifests itself by spots varied in form, extent, and color. The exanthem appears and disappears rapidly; so rapid indeed is the disappearance, that before the physician arrives, it often happens that the eruption is gone.

Subsequently, *different eruptions* appear, among which are the many forms, to describe which dermatologists have exhausted the systems of classification. Take a syphilitic infant: examine in it each kind of exanthem, the well-defined pustule, papule, vesicle, &c., consider the aggregate of the eruption, and you will be struck with the very special aspect of some of the eruptive lesions which solicit your attention, and will decide the diagnosis.

That which makes the description given in books of venereal exanthemata so delicate and sometimes so subtle, is the desire of authors to find in each form a distinctive character. Clinically, we have a right to substitute the real fact for this dogmatism: given a syphilitic eruption, let us put aside the non-distinctive lesions, and restrict our inquiry to the best-marked lesions.

The cutaneous affections which may almost be entitled pathognomonic are those upon which alone it seems to me of any use to insist: of these, the *mucous patches* occupy the first rank, after which come *squamous affec-*

tions and *ulcerations*, which represent the second phase of the evolution of different elementary alterations, and the color of the skin.

The mucous patch, as you know, is one of the manifestations of syphilis which is most frequently observed; and yet it is one regarding the value of which fixed views are still far from having been attained. So common is it in women, that in them, there is scarcely a case of constitutional syphilis exempt from it; and in infantile syphilis, it is not less frequent. I have shown you mucous patches upon the cutaneous margins of mucous membranes: you will likewise find them on the skin, in the vicinity of the anus, in the inguinal folds, on the haunches, and even on the trunk. Their form of development varies with their situation. They are quickly curable, when situated where they are free from being chafed; but they accumulate and thrive (if I may be allowed the expression) wherever there exists both rubbing and exudation.

It is as difficult to describe the mucous patch in the infant as in the adult; and I really cannot depict it without recalling its appearance to your recollection. Let me simply say that in early infancy mucous patches have a more spongy and less indurated base, that the bottom of the lesion is more generally moist, and the oozing more abundant: and since, from want of an exact definition, I invoke your memory, I cannot do better than compare them to those met with in the labia minora and on the internal surface of the labia majora.

The mucous tubercle would of itself be sufficient in the recently born infant to decide the question of syphilis: but then how numerous are the causes of uncertainty and error! How many non-specific eruptions affect a very similar form under the influence of rubbing, moisture, and contact with irritating substances! The diversity of names given to this lesion tells very clearly the diversity of appearances which it presents.

I am anxious to try to describe to you the special ulcerations of the skin, buttocks, and thighs, which you have had so good an opportunity of observing during the last few days in a little child in bed 17 of our nursery ward.

I pointed out to you these *serpiginous ulcerations of the skin*, exactly resembling the traces left in wood by xylophagous insects. These ulcerations, which frequently do not exceed two millimetres in breadth, have so decidedly special an aspect, that I regard their presence as one of the most pathognomonic signs. After they are healed, moreover, they leave linear cicatrices, at first red, then white, which by their form distinctly remind us of their origin.

In the infant, *false psoriasis* occupies the palm of the hand and the sole of the foot. The skin, at first wrinkled, seems to grow thicker; the epidermis, less elastic than natural, cracks at the digital intersections, and wherever movements subject it to extension. Soon, some patches of epidermis are detached, and the surfaces which they leave denuded become covered with new epidermis, which is so thin that it is no stretch of language to compare it to the outer skin of an onion. The feet and hands thus denuded, assume a livid, occasionally copper color, but present nothing else which is characteristic. The inexperienced physician might easily be led into error, were he to rely on descriptions necessarily insufficient, as he might confound lesions of syphilitic orgin with stripping of the epidermis in the recently born infant, an occurrence depending upon wholly different causes.

As the mucous patch originates and develops itself upon various cutaneous eruptions, after the manner of a parasitical growth, vesicular and pustular eruptions may produce specific ulcerations. Whether the eruption does or does not assist in the transformation, it certainly has to do with it: every venereal ulceration in children has an eruption as the basis of its development. The pustule, in place of cicatrizing, spreads out, and burrows: its diameter increases, its edges become elevated, and phenomena are observed similar to those seen in certain cases of small-pox at a stage when the pustules ulcerate, in place of becoming more or less covered with crusts. Engendered in this way, the syphilitic ulcers of children are met with in every situation in which products of the eruption can exist: they have, however, a preference for the buttocks, the lower part of the abdomen, and the cutaneous folds in the vicinity of the genital parts.

I have striven, gentlemen, to indicate salient features to you, without dwelling upon them. I wish you to realize the imperfection of my sketch, and to feel the necessity of your completing it by your own personal observation: this necessity becomes specially apparent to me when I proceed to direct your attention to the *peculiar hue of the face.*

It not unfrequently happens that the physician, taught by long familiarity with this appearance, will almost at once diagnose syphilis after having simply seen the child's face, although the peculiar hue can be described but vaguely in words. The visage presents a special shade of bistre: it looks as if it had been lightly smeared with coffee-grounds, or a very dilute aqueous solution of soot. There is neither the pallor, the icteric hue, nor the straw-yellow tinge of skin seen in other cachectic affections; the tinge is not nearly so deep, but is almost like that of the countenance of a recently delivered woman, and either does not extend at all, or only partially, to the rest of the body. I know no disease except syphilis in which a child's skin has this peculiar color: and consequently, when it is well marked, it has more diagnostic value than any other symptom.

The child's little suffering face presents some characteristics besides the bistre color.

The eyebrows have either not been developed, or have fallen out: the eyelashes are often everted: at the external angle of the eye, we sometimes find fissures like those seen on the lips and at the opening of the nares.

In place of eyebrows, from which the hair has fallen, there are seen two yellow, bistre-colored stains, and a considerable amount of desquamation; and these same bistre-colored stains, which in fact are patches of psoriasis, are most abundant on the chin and round the mouth.

I have been obliged in my description to decompose the syphilitic eruption, taking separately its surest manifestations; and it would be useless in me to attempt to re-establish it completely. The forms of infantile syphilis are grouped together so differently, they vary so much individually, in extent, in progress, and in tendency to transformation, that it is necessary to be guarded in forming an opinion, necessary to watch events, and to distrust rules applicable only to particular cases.

Were syphilis in the adult the subject before us, I might now proceed to speak of treatment, and so complete the history of the disease. But we have at present to do with syphilis in early infancy; and to describe the cutaneous manifestations of syphilis is to indicate a part only of its manifestations, those which are the most important in respect of diagnosis, and which enable us to give a name to the affection; but the least important perhaps for the clinical physician, whose desire is not only to give a name to the disease, but also to be able to foresee its ulterior evolution.

Syphilitic infants are from the very first under the influence of a *cachexia*, which adults do not always escape, but which in them is far from occurring so constantly or presenting so much gravity. In proportion to the greater or less severity of the symptoms will be the greater or less chances of life or death; and all the signs which intimate the imminence or danger of a disturbance of the general health will acquire the greatest possible value. When called in to an infant said to be affected by syphilis, hesitate to indorse the opinion and be physicians; watch the most insignificant disorders, the slightest functional disturbances, and do not look upon the state of the skin as the sole means of estimating amelioration or aggravation in the state of the patient, as you ought to be able almost authoritatively to do in older subjects.

The cachectic physiognomy of the syphilitic infant has been described in a very exaggerated manner; but the disorder is not the less deepseated that it is the less visible. When a robust, well-formed infant has brought with it into the world a sufficient reserve stock of vigor to traverse this period of severe trial, it becomes weak and dejected, loses flesh a little, and is rather puffy, and presents a pallor of seemingly œdematous character, while at the same time the integrity of the functions is preserved. The infant is in the same condition as the adult under the influence of the same cachexia; in proportion to the operation of the treatment the amelioration of the general state becomes apparent. The little patient being no longer irritated by its sores or by the contact of the excreta with the ulcerated surfaces, sleeps better, and is forthwith benefited by the tranquillity of its slumber. The complexion loses the bistre hue; the physiognomy becomes more lively and cheerful. Should this happy change take place during the early weeks of treatment, an entirely favorable result may be hoped for.

Unfortunately, however, the course of events is not always so propitious. The syphilitic infant is seen to grow thin, and to suck with less avidity, symptoms arising from diminished appetite and embarrassment caused by persistence of the coryza. The sleep is short and disturbed. Digestion is imperfectly performed; vomiting is not a usual symptom; diarrhœa is of frequent occurrence, and is of an inveterate, often sanguinolent character, the large intestine specially participating in the infection. Respiration is inadequate; and the more important functions being thus implicated, no longer assist in accomplishing the urgently required reparation.

The cachectic condition sometimes exists to such an extreme degree that the termination of the case is more disastrous than there seemed reason to anticipate; an excessively weak infant left in a state seemingly serious rather than alarming has died simply from syncope.

Infantile syphilitic cachexia presents a twofold study; on the one hand, we have to consider the degree of severity in which the syphilis exists, and on the other, the presence in subjects so young of all the causes of exhaustion, which are called inanition, diarrhœa, intermittent fever, or pox.

The autopsy may often disclose nothing to which death can be directly attributed, but may reveal lesions on which depend slow, profound disturbances of the economy. To this category belong the alterations of the liver studied by Gubler; peritonitis in the fœtus described by J. Y. Simpson (an affection from which young infants are not exempt); certain pulmonary lesions, as yet imperfectly known; and organic alterations of different organs presenting no appreciable specific character.

Gentlemen, having spoken at some length upon the symptoms of infantile syphilis and their subordination, I now come to consider the origin of the manifestations of which I have given you a sketch. It is nearly twenty years since I first tried to explain the *pathogenesis* of infantile syphilis. I was guided then, as I now am, solely by experience, uninfluenced by any doctrinal bias. Among the facts which I have observed, and specially among the conclusions which these cases seemed to warrant, some were looked upon as rash hypotheses and others as enormities. These views, which excited so much opposition when originally announced in clinical lectures at the Hôpital Necker, have now become classical, so that in place of having to defend, it is sufficient for me to state them.

At the period to which I refer, a generalization, captivating from its exclusiveness, had reduced the transmission of syphilis to the simplest possible formula. Chancre produced chancre, in virtue of a law so absolute, that from the first moment that syphilis appeared its presence might be affirmed, although its origin was shrouded in obscurity similar to that which envelops the beginning of everything. Original inoculation was alleged to be the only means of infection; and you know how many ingenious combinations, how many learned essays on human morality, how many clever anecdotes, served to fill up the gaps, and give reasonableness to the theory.

The infant might escape the depravity of an inventive libertinism, but neither relations nor nurses enjoyed a like immunity. The infant being in contact with infected individuals, the object of caresses more imprudent than blameworthy, it became the innocent victim of the most unforeseen inoculations. We see the adult elude investigations undertaken in his own interest; but how much more reason is there for even the best-conducted inquiries to fail to disclose the mysteries of the transmission of syphilis in the recently born infant. The intelligent skepticism which reaches incredulity through a course of sagacious observation, which destroys belief by ridiculing credulity, always possesses witty aspects of which experimental truth is deprived. Where is the physician who has not regretted having been too credulous, and who among us has not experienced a feeling of honest self-approval in having detected fraudulent representations? To show—what was only too certain—that syphilis was largely used as the means of deceit and lying was a service for which we ought to be grateful.

Facts, at last, became so numerous and so decisive, that criticism was crushed by demonstration. The progress of the truth was gradual; and the movement has not yet attained its full extent. Rules, at first rejected as erroneous, did not afterwards admit of being discussed; and in relation to certain points, it may be said, that in respect of the generation of infantile syphilis, scientific knowledge is complete.

Had we only succeeded in establishing on a solid basis the pathology of syphilis in early infancy, the gain to science would have been very precious. The study of the venereal disease in children had the advantage of suggesting doubts as to the strict accuracy of prevailing theories, and of inducing physicians to subject them to clinical revision. Then began researches into the transmission of secondary symptoms, which opened up an entirely new path of inquiry, which made it seem less impossible for their transmission to take place from adult to adult, by proving that it did take place from infants to adults, and from adults to infants. As, however, I have restricted myself for the present to the consideration of syphilis in infants, I must now return within the limits of my subject.

A syphilitic mother may give birth to an infant carrying the germ of her disease: that is the first, and the least disputable fact. It is a second, and not less positive law, that a syphilitic mother may produce a child free from syphilis. In respect of both laws, syphilis follows the law applicable to all hereditary affections.

Is maternal syphilis transmissible by the mother, only when she had the disease prior to conception? Or, may pox, contracted by the mother during pregnancy, be transmitted by her to the fœtus?

This is an important and difficult question: do not attempt to solve it by the simple rules of common sense, which all agree cannot solve any medical problem. It has been thought most probable, that when syphilis is derived from the mother, it must have existed in her before conception, it being no doubt, more natural to suppose infection of the ovule than of the fœtus. Cases, too, in which syphilis contracted by the mother during the latter months of pregnancy have not contaminated the infant have also seemed to lessen the probability of infection after fecundation. Finally, there has been an unwillingness, through fear of consequences, to admit that the blood of the fœtus can be vitiated by its mother's blood. Were that admitted, how could it be denied that syphilis may be communicated to the infant by lactation, and in other ways still more hypothetical?

I am never afraid of the consequences of a positively ascertained fact. It is quite true that a mother infected before conception may give birth to a syphilitic infant: it is also true, that a mother infected during pregnancy may infect the fœtus which she carries in her womb; and of this latter truth you had an example in bed 24 of our nursery ward. Between the two classes of cases, however, important distinctions have been rightly established, and ought to be maintained.

The more thoroughly we examine the simplest laws of the pathogenesis of syphilis, the more do we find that possibilities multiply, and that *casuistry*, if I may use the word, becomes increasingly subtle.

For how long a period is maternal syphilis susceptible of transmission? Does it exist during the primitive, or during the secondary symptoms? Does it exist in the tertiary period? May it still be present after an indefinite period has elapsed since the last manifestations of the disease? Again, supposing that the infection is possible at any date, which period is the most favorable for its transmission?

Unfortunately, I cannot answer all these questions. I do know, however, that the mother may conceive a syphilitic infant, at a time when she herself seemed exempt from the disease, which had left no traces. I believe that the period most favorable to transmission is that which succeeds the first phase of the secondary symptoms. I also know that the mercurial treatment, against which they begin to speak, when properly carried out, nullifies syphilis in the woman, even though, as is asserted, it do not cure it; so that she, after having conceived a succession of syphilitic children, is treated by mercury, and then produces uncontaminated offspring.

It may perhaps appear strange to you that I thus circumstantially announce a self-evident proposition: but I insist upon it, because syphilographers have not sufficiently taken into account a fact of which they were not ignorant: being engrossed with the treatment, they have almost forgotten the subject of pathogenesis. I leave for your own reflections this very elementary idea, which you will see is not devoid of importance.

At what period of pregnancy is syphilis, contracted after conception, transmissible? This we do not know: but there is rightly a disposition to believe, that the nearer the date of infection is to the commencement of pregnancy, the greater is the probability of the fœtus becoming contaminated. Does this depend upon the disease of the mother having a duration relatively longer when she is infected at an earlier state of gestation? This question, I cannot answer; and indeed I do not feel myself sufficiently certain as to the fact itself to offer an explanation.

We have been supposing a case in which the mother only is syphilitic; but let us suppose a parallel case, in which the father alone is infected. This is a less complicated problem, inasmuch as the paternal influence must be contemporaneous with fecundation; but then again, it is obscure in respect of the evidence of paternity.

For my part, I do not hesitate to declare (and I have long held this opinion), that *syphilis is transmitted from father to child* when the mother is not infected: I also recognize as fully as any one the difficulties attending a decisive investigation, and would remark that the practice of medicine does not encourage obstinate illusions. But some reservations which a knowledge of the world exacts, certain cases impose, of which I am convinced by having seen enough of such cases: and you, gentlemen, will meet with them in sufficient number to share with me this conviction.

Here, again, the question presents itself in terms similar to those in which we asked it in respect of the mother: At what stage of its evolution is paternal syphilis transmissible? The answer is the same—with this difference, however, that the opportunities being more numerous, and it being more easy to be well informed as to the syphilitic symptoms of the man, in respect of their progress, date, and phenomena, we may perhaps find more precise elements for arriving at a decision.

An infected woman has a thousand reasons for concealing the nature of her malady: besides, she often does not know whether or how she has contracted it; and as she has ignorance for an excuse, she generally escapes continuous observation. In the man, there can hardly be offered any pretext for ignorance. He has no reason for concealment; and, thank God, you will meet with more men who are anxious than men careless as to the future. Observe, I make this remark only in respect of venereal affections.

You will find that you are often consulted on this subject by men, but never by women, about to be married. You will be told the exact date of infection, the symptoms which supervened, the treatment which was prescribed and followed: every facility will be afforded you for verifying the statements made, and you will be solicited to do so: no information will be withheld from you. Yet, how many uncertainties, and legitimate grounds of hesitation in forming your opinion!

It is unnecessary to say that a man who is syphilitic ought to abstain from procreation. But the question is: To what extent is there absolute security, when a long period has elapsed since the disappearance of the disease?

I have often mentioned to you the case of a physician who consulted me: he had been cured of syphilis, married, and became the father of a syphilitic child. I have cited the case, because it presented every condition required to constitute such a demonstration as science demands; and because it came under my notice at a time when professional opinion was still undecided. Since I met with that case, how often have I seen similar conditions lead to similar consequences! How often, also, let me add, have I seen fathers properly cured of syphilis by the classical medication, engender children exempt from any trace of the disease.

The hereditary character of syphilis, as of all other diseases, is liable to so many exceptions, that it is necessary to guard ourselves against the undue influence of preconceived opinions; and to bear in mind, that while, in respect of hereditary transmission, there is everything to fear, there may be, occasionally, everything to hope for. It sometimes happens, that under the most unfavorable conditions, both father and mother being affected with pox in the most palpable manner, everything consequently conspiring against the health of the fœtus, it nevertheless comes into the world free from the disease. But on the other hand, the symptoms of syphilis in one parent having yielded to rational treatment, we conclude, after mature deliberation, that all is safe; but nevertheless, the child is born infected, and dies of syphilis.

Hereditary infection is not the only risk which infants are exposed to from syphilis. The infant, in its *constant contact with the nurse*, or with other women who bestow on it those little services without which it could not live, often incur the hazard of contracting syphilis in a very easy manner by *direct inoculation*. I am not now speaking of the risk of inoculation in the genital passages, that being a matter on which it is quite unnecessary to insist, though some ability has been shown in using it as an argument in favor of certain theories. A child *directly inoculated* by the nurse or otherwise, becomes of course a cause of danger to all those about it, being as able to transmit, as it has been to receive infection.

Gentlemen, if, going back in thought some years, I fancy myself explaining to my students, at the Hôpital Necker, the laws which preside over the post-partum infection of the recently born infant, I am able to estimate without any difficulty the advance which science has accomplished since that time. Then, I had to discuss denials, contend against pressing objections, accumulate proofs, collect cases, and give to my hearers a review of these cases with the fullest details, or submit the patients themselves to the skeptical criticism of my class. Now, facts having spoken, principles are sufficiently firmly established to require merely to be stated. I shall, therefore, be brief, as I ought to be, when dealing with unassailable doctrines.

The nurse may transmit to the infant the primary taint by which she was affected. This has never been denied. *She may also inoculate it with secondary symptoms;* and although the possibility of transmission in this way was long contested, this mode of transmission is much more frequent and not less satisfactorily established than the former. Here, too, there is reciprocity, as in the former case,—the nurse may be the victim of the nursling affected with hereditary syphilis.

It is a great matter to affirm this law: but this affirmation is not enough to dispel all the doubts which will present themselves to you in practice. The nursling, like the nurse, is liable to a double inoculation; it may be infected after birth, or it may bear the insidious germ of a disease destined to break out at the end of some months. *The nurse may be inoculated in coitu, or by the contagium of the recently born infant.* Have we the means of recognizing each of these occurrences with certainty, or of estimating their relative probability?

It would be superfluous in me to impress upon you the importance of this inquiry, the momentous gravity of which you can understand, even when it does not assume the form of a judicial inquiry. In cases of this kind, the physician exercises a judicial duty which is paramount to every other: his responsibility is enormous in the eyes of the world; but, for himself, it will be enough to realize that responsibility in his own conscience.

The more I feel the magnitude of the responsibility of giving a decision in cases involving these questions, the more do I desire to be able to fortify your judgments by giving you precise data.

Unfortunately, I cannot furnish you with absolute signs; but obedient to the demands of a duty which I believe to be imperative, I shall endeavor to prevent your being misled by perilous assertions. Every case will come before you surrounded by complex circumstances, and you will have to disentangle each particular truth, without generalizing the results of your examination. Do not be astonished at this seeming impotence of science: accept it as a necessity with which your daily practice will make you familiar. Medical laws are to the physician what the legislative code is to the magistrate: without them, deviations from the right road would be incessant: but guided only by them, individual problems cannot be solved; for being a lawyer never sufficed to make a man an able acute sifter of evidence and examiner of witnesses.

The nurse who has transmitted syphilis to or received it from the nursling, may be in similar, if not in identical conditions. It seldom happens that you are consulted at the first occurrence of the untoward symptoms; more or less time has elapsed since inoculation, so that you have to gather the history of past events from recitals in which ignorance contends with deceit. It has been said that the infection is communicated more frequently by the mouth than by the womb: but how numerous are the exceptions to this pretended rule. Supposing that the original centre of contagion was in the situation where the infant most frequently came in contact with the nurse, and supposing also that the infant was suckled, how incalculable the opportunities of its disseminating the evil! You see vaccinated infants inoculate themselves with vaccine matter from the arm on all parts of the body much better withdrawn from their attempts: you also see the pus of a primary ulceration carried to the genital organs, to the belly, and to every situation to which the nurse herself carries her incessant intervention.

If the question be as to primary symptoms, which are almost limitable in duration, limits may be indicated; but we do not know at what period secondary symptoms have ceased to be inoculable. Even a nurse, like all other women, escapes from inquiries which in the case of a man would yield valuable information. In women, the ulcerations consecutive to chancres cicatrize without leaving visible traces, the induration of the edges is not so prominent, and the glands are less affected: the chancre may have its seat on the womb, or may be concealed in some situation unsuspected by the physician, however well acquainted he may be with the divagations of debauchery.

Theoretically considered, the solution of the question is beset with doubts: in actual practice, however, it is simplified; for by considering very subordinate circumstances in connection with each other, by analyzing statements, and discussing their contradictions, we are enabled to base conclusions upon plain solid reasons.

In cases of this description, as in all medico-legal consultations, the possession of knowledge is the important element. Acquire a profound acquaintance with infantile syphilis: study thoroughly the evolution of syphilis in the woman, and having thus made yourselves strong by the possession of knowledge, you will be in a position to grapple with the difficulties of each inquiry. To express the fact in better terms—you will be able to use difficulties themselves as means of discovering truth.

There is still another mode of inoculation, which, while it is very much like that of which I have been speaking, is in some respects distinct from it: I refer to the *transmission of the syphilitic virus by vaccination.* This possible means of transmission, at first denied by deservedly esteemed syphilographers, seems to me to be firmly established by conclusive experiments. In 1861, you saw in our service a very sad example of this mode of transmission; and the facts, so testing, of what may be called the epidemic of Rivalta, will satisfy any physician as to the correctness of this view provided his mind be not previously influenced by an opposite bias.

It would be out of place here to refer to the extended discussions which arose out of the law-case of Dr. Hubner of Bambey, which, since 1854, has originated so many contradictory allegations. Since that date, other cases of the same kind have been cited; but it would lead me far beyond the limits of my teaching in this place, to discuss or even describe rare cases, which I know to be exceptional and the subjects of criticism; while at the same time, it would be absurd in me to consign them to the limbo of apocryphal pathogeny. I have already, moreover, sufficiently noticed them in my lectures on vaccination.*

There still remains for consideration another question relative to the transmission of congenital syphilis. Suppose a child engendered by a father who had had the pox, but who no longer showed symptoms transmissible by inoculation: *Could this syphilitic child when in utero infect its mother?* You can estimate, gentlemen, the number of difficulties which surround such a problem: you can see how many elements will be wanting for its solution, because it may be asked, whether the woman supposed to be infected by her fœtus had not been previously the subject of syphilis which had passed without recognition. Be that as it may, the transmission of syphilis from the father to the mother, through the medium of the fœtus, is now admitted to occur.

The fact admits of an easy physiological explanation. It is indeed certain that the mother by mingling her blood with that of her infected fœtus becomes infected with syphilis. Is it at all improbable that a fœtus the blood of which is syphilitic should infect the blood of its mother? It is about the third month that the circulation of the fœtus becomes active. By the umbilical vein, it receives the blood of its mother, and it returns to her by the umbilical arteries that which has traversed its organs, and which is a mixture of its own with the maternal blood. You know, without my impressing upon you the fact, that in the fœtus the blood and the blood-vessels are formed almost simultaneously. The fœtus, therefore, has blood which is peculiarly its own, and if the fœtus is syphilitic by its father, its blood is syphilitic in virtue of the same title as the other parts of its organism. Consequently, it can infect its mother through the medium of its blood, just as the syphilitic mother can infect the fœtus in her womb.

A reliable observer has communicated to me the following case, which is in accordance with physiological facts: A young lady of unquestionable morality became pregnant within a few days after her marriage. Her husband, a physician, had had syphilis three years previously, and had no remaining trace of the disease, except slight engorgement of the cervical glands. The lady, at the third month of her pregnancy, felt an itching in the labia majora; afterwards, it was found that there existed pretty extensive ulcerations in process of transformation into mucous crusts. Some days later, they were perfectly formed: there was also sore thoat and engorgement of the cervical glands. At the eighth month, the lady was delivered of a miserable child, which became affected with syphilitic coryza and ophthalmia on the tenth day after birth; and which died, when six weeks old, with fatty liver, ascites, and œdematous extremities. The day before its death, epistaxis occurred.

The most disputable fact in this case is the appearance of ulcerations on the external genital organs, ulcerations which may be attributed to direct contagion. But it must be remembered that they were mucous crusts, and not true chancres; and the development of mucous crusts in these parts, which are during pregnancy the seat of great erethism, appears to me to be a phenomenon in all respects analogous to the formation of vegetations and crista galli on the surface of the vulva in some non-pregnant women. The exuberant vitality of these organs during pregnancy is the starting-point of the morbid manifestations.

Perhaps I have dilated too much upon the manifestations of syphilis in infants, upon the possible and probable forms of inoculation and upon its hereditary genesis. I have omitted all historical notices of the subject, feeling that as I could not do justice to all the observers who have so powerfully contributed to the elucidation of the disease, I ought to abstain from quoting any of them.

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* Volume I, p. 113.

I have still to speak of the *treatment* of syphilis in infants. Though the subject is most important, it is both possible and useful, I think, to discuss it with brevity.

Whatever may be the age of the patient, syphilis must be treated in accordance with the same principles. The remedies to be employed are the same; and the end to be attained, as well as the means of attaining it, are also the same. Keep steadily in view this simple and fundamental truth. Were it allowable to employ in infants the classical preparations so easily administered to adults, the problem would be solved, or rather there would be no problem to solve. The difficulty does not arise from indications, but from counter-indications and obstacles presented by the infantile constitution to the tolerance of medicines.

I have spoken to you of the influence which syphilis, when left to itself, exercises upon the general health of the infant. I have shown you the increasing cachexia exhibiting itself in functional disturbances, and particularly in an altered state of the digestive function. These are the points to which your attention ought to be unceasingly directed: it is by the state of the stomach and intestines that you are guided as to the increase or diminution of the doses of medicines, and the suspension of a remedy.

Mercury ought to be the basis of your treatment. I neither ignore the renewed objections to the mercurial treatment, nor the inconveniences which attach to its employment: I am aware that by a sort of periodical reaction, there have been on different occasions attempts to combat and dethrone it, but I also know that these attempts have only had their day, and that mercury after being strongly denounced, has always been reinstated in favor by the force of circumstances. In the recently born infant, the employment of succedanea is impracticable: depuratives of the best repute are out of court simply because it would be utterly impossible to make use of them.

Of the preparations of mercury used internally, after numerous trials by myself and pupils, I continue to prefer the solution of corrosive sublimate so well known as the *liqueur de Van Swieten.* I give it to the extent of one or at most two grammes a day in milk: so administered, the infant takes it without repugnance. Nevertheless, though the administration of the perchloride of mercury be easy, it very often happens that one is obliged to renounce its use on account of the diarrhœa which it keeps up or causes. In such cases, it is best temporarily to desist from all mercurials, because, under the circumstances, not one of them can be used with impunity, all of them favoring the troublesome tendency which it is essential to combat.

It was proposed to give calomel in very small doses, particularly at the time when it was hoped that by combining it with chlorate of potash, we should be able to avert salivation in the adult and diarrhœa in the infant. I am not sufficiently well informed in relation to the advantages of this method of treatment to recommend you to adopt it; but I may say, that I am inclined to believe that the addition of the chlorate of potash would, at least for adults, lessen the antisyphilitic action of the perchloride of mercury.

The protoioduret of mercury does not seem to me to possess any advantages; and I do not think that at present it possesses many advocates.

With the laudable purpose of averting threatened disturbances of the digestive function, or preventing their imminence, it has been recommended not to give any medicines internally, and trust entirely to mercurial frictions. Although this method reckons among its supporters many respectable physicians, I reject it, as one which (besides other inconveniences) leads to gastro-intestinal symptoms. The skin of the infant receives mercurial applications badly, for they are always irritating, a preventive to their being absorbed. The external use of mercurials, still much in vogue in England, Germany, and in all the north of Europe, is only exceptionally employed in France, even in the treatment of syphilis in adults.

As a general rule, we ought not to break the skin of the young infant. Even when the infant's skin is healthy, it has not a sufficiently active vitality to furnish a therapeutical leverage of much value: when diseased, it acquires a very baneful influence, for which reason you ought always to endeavor to cure local lesions of the skin: to eradicate them is to render a signal service to the little patient. Is every ulcerative centre which you allow to become developed, or of which you follow the evolution, a source of general infection? I would not dare to say it, but I know from experience that the general health of the child resents injury of its skin. Whether it be that the little wounds of the skin cause an irritation which agitates the new-born infant, whether it be the greater or less exhaustion caused by every pathological process of ulceration, whether it be that the contact of irritating matters becomes a source of pain, it is a fact, that the syphilitic child always improves, and that the diseased state of the skin is often ameliorated.

You will have occasion, according to circumstances, to have recourse to the most varied topical applications to the skin, to caustics more or less diluted, and to emollients, the use of which, however, is very limited; but of all remedies I know none comparable to baths and lotions of corrosive sublimate. Perchloride of mercury dissolved in water by the aid of alcohol or chlorohydrate of ammonia has the very great advantage of being easily used in whatever doses circumstances demand, ranging from the slightly caustic lotion to the bath so weak as not to cause any appreciable sensation. For a child's bath, I never use more than a gramme of sublimate.

The infant has almost as much tolerance as the adult for this medicine: it would, therefore, be bad practice to reduce the strength of the solution to that which the formularies almost invariably prescribe.

In curing the morbid condition of the skin—which is possible even when we do not cure the syphilis itself—you have the advantage of leaving it available as the medium of a medication which may yield you great success. The debilitated cachectic infant may at any given moment be unable to repair its lost strength: tonic baths, sulphurous baths, afford resources which tonics given internally do not confer; but then, unless the skin be sound, you cannot resort to this kind of treatment.

In treating infantile syphilis, *diet* takes the first place as an adjuvant, if not as a medicament. Lay it down as a rule that the infant which is not suckled, or is badly suckled, has a hundred chances against one as compared to the infant fully nourished from the breast: unfortunately, the limits within which you can interfere in this matter are very narrow. Should the mother be unable to supply a sufficiency of milk from her breast, it is a serious responsibility to confide so dangerous a nursling to a nurse. You are, in fact, compelled to be satisfied with a nurse who is nearly suitable, with whose insufficiency you are acquainted. In the matter of infantile nourishment, there is nothing more compromising than half-measures.

It is even more essential that the infant be suckled by its mother, as internal treatment applied to her will sometimes have so powerful an influence as to cure both. In the infant, the iodide of potassium is almost never applicable, but it is often useful to the nurse and through her to the nursling; but in her also, the liquor of Van Swieten ought to be specially recommended.

I cannot too urgently impress upon you the necessity of adopting the most diligent hygienical precautions. Spare the infant from every cause of discomfort which can be known or pointed out in advance: carefully avoid giving the child a chill, than which nothing could be worse for it. The recently born infant must be kept in a temperature which is not only equal but is also high. You are aware of the importance which physicians of northern countries attach to the elevation of temperature in the treatment of obstinate syphilis, the patients affected with which are kept in veritable stoves. Use their example and experience for the benefit of infants affected with syphilis.

However numerous your precautions may be, with whatever solicitude you may surround the little sufferer, whatever devotion you may have found or awakened in the mother, you must be prepared to meet with many failures. In the recently born infant, syphilis is always formidable, and a disease which is apt to prove mortal. Left without treatment, it exhausts the patient by internal lesions, anæmia, and functional disturbances. It often opposes a long resistance to remedial measures: the economy has not the power to sustain the prolonged struggle, and frequently, the remedy injures on the one hand, when on the other, it proves beneficial. It may be said unhesitatingly, that congenital syphilis is nearly always mortal, if it show itself within the first fortnight after birth. The danger diminishes in proportion as it is long in manifesting itself.

In conclusion, gentlemen, allow me to recall your attention to the reservations which I thought it necessary to make at the beginning of this lecture. The syphilis which attacks the infant raises the most delicate problems: it is one of those diseases in which experience does not enable us to jump at conclusions. We can seldom proceed with our diagnosis aided by the security of pathognomonic signs, and are obliged to rely upon the attentive discussion and comparative examination of minute circumstances. The pathogenesis encounters difficulties in practice, which belong at once to the insidious nature of the evil, and to urgent interests which bring into the field dissimulation and lying. I endeavored to give you a sketch of the principal elements of the march, evolution, and symptoms of the disease; but my chief anxiety was to tell you again and again, that you must equally avoid an insufficiency of "*savoir*," and that excess of "*science*" which leads to the premature glorification of theories.

LECTURE LXXXII.

GOUT.

Preliminary Considerations.—The word "Gout" is much to be preferred to any of the other names which have been proposed in place of it.—Gout, Acute and Regular.—Premonitory Phenomena.—Disturbance of Digestion: Disturbance of the Nervous System: Disturbance of the Urinary Organs.—Catarrhal, Urethral, and Ocular Affections.—Arthritis, its Progress and Appearances.—Acute Gout in the form of short Paroxysms which either succeed to, or run into one another.—The paroxysm may supervene under the influence of an immediate appreciable cause.

GENTLEMEN: When, in compliance with the request of many of you, it became my intention to devote some clinical lectures to the history of gout, I believed that I was sufficiently acquainted with the disease to treat it in a proper manner. But when, with a view to put you in possession of the subject, I set myself to think over it—when I tried to arrange in proper order the numerous facts which had come before me in the course of my practice, and to complete the results of my own experience by the perusal of the writings of others, I perceived how far I was from being in a position to respond to your expectations. I will not, however, shrink from the task you have imposed upon me. I shall do my best to state to you my views regarding the nature and different phases of the disease; and I shall likewise point out to you what I think ought to be the general management of gouty persons.

When one has meditated upon Sydenham's Treatise on Gout, a marvellous monograph, at once concise and complete; when one has gone through the cases described by Musgrave in his work on anomalous gout—a work far too much lauded, I think, and in which gout and rheumatism are very often mistaken for one another; when one has read Scudamore's treatise on the nature of gout and rheumatism; when, finally (and to refer only to important works of our predecessors), one is acquainted with the Commentaries of Van Swieten and the Aphorisms of Boerhaave—one is almost able, with the aid of his personal recollections, to form some opinions regarding the disease of which I am now about to speak to you. And at present, if to put our ideas more in harmony with the existing state of knowledge, we read contemporary works—if we cast our eyes over Dr. Garrod's work, which, by the way, appears to me far inferior to its reputation—if we rummage the innumerable essays published on the subject, and among others, the excellent thesis defended before the Faculty of Paris by Dr. Galtier Boissière, we feel convinced, notwithstanding the pretensions of modern medicine, that we have made no advances, since the time of Sydenham, in our knowledge of the treatment, phenomena, and special nature of gout.

Translate the work of the English Hippocrates into what is called more scientific language, and you will not only admire the description which that great man gave of the disease, but you will be surprised to find how little he left to be said regarding it by those who came after him. Being himself tormented for many years by acute or chronic normal gout, his description is only applicable to normal gout; but that he has described in so masterly a manner as to leave almost nothing unsaid in respect of it.

With regard to anomalous gout, you will not find the materials for its history in special treatises. These materials are scattered through books which treat of gout only in a very incidental manner; and generally you will find them concealed under names widely different from those which unmistakably pertain to it. Thus, for example, there are many so-called metastases of rheumatism, which are nothing else than metastases of gout.

Anomalous gout, more common than is generally believed, assumes such diversified aspects, normal gout itself is so often divested of the type created for it by nosologists, that one would be deceiving himself were he to believe that he was familiar with it.

In thus pointing out to you the difficulties of the question, I confess beforehand my incompetence, and perceive the deficiencies for which I may be blamed.

To prevent any mistake as to the meaning which I attach to certain words, and to certain theoretical views which will frequently recur in the course of these lectures—to enable you to understand what I myself understand by normal and anomalous gout—you must have clear ideas of specificity and diathesis.

Here, then, we are once more brought back to the great question of specificity, upon which I every moment insist, because every moment we see it plays its part at the bedside of the patient. This part, which the school of Tours, through Bretonneau, its most illustrious representative, has placed in a strong light, is no longer disputed by any one. Will any one deny that specificity intervenes in almost all affections to such an extent, that those apparently the most similar differ in reality from each other in a very marked manner—when you see it, in acute or chronic diseases, imposing on the attentive observer by constantly showing itself in the form of anatomical lesions, by invariably manifesting functional disturbance, and by the nature of its concomitant symptoms? Along with the characters possessed in common by several species of diseases, there always exist others which belong exclusively to each individual disease, and which serve to distinguish it.

The subject now before us presents a remarkable example of this. There is certainly a great analogy between gouty arthritis and rheumatismal arthritis, irrespective of the general characteristics of inflammation which they possess in common. But even in this inflammation we can detect notable differences, if we consider its preferential seat respectively in the two diseases, its appearing accidentally in rheumatism and periodically in gout, the production of tophus as a sequel of an attack of gout, and never following an attack of rheumatism; and in particular, if we consider the manner of evolution. These differences are still more evident, when we study the general symptoms which precede, accompany, or follow the local articular manifestations. In gout, these manifestations are nervous disturbances, which, if I may use the expression, are obligatory phenomena of the attack, but which are absent in rheumatism. They are of such a nature, that, with only a single articulation involved, and involved to a less degree than if several joints were invaded by rheumatism, the gouty patient does not bear up so well, and is more weighed down by his disease than the rheumatic.

In the complications, or rather in the local non-arthritic manifestations, these specific differences also exist. Thus, in gout, affections of the urinary organs supervene which are not seen in rheumatism; and, again, the heart is very often implicated in rheumatism, and seldom in gout.

In a word, gentlemen, whether you have to do with the local or general manifestations of the disease, you will find everywhere engraven the indelible characters of specificity. When, at the first glance, we do not observe the stamp of specificity imprinted upon the more external phenomena, by careful looking, it will be found. I admit, that if I receive no explanation of the case, if all except the affected part be concealed from me—if, for example, I am only shown a joint, or the instep of a person affected with arthritis, it will often, no doubt, be difficult for me, by merely looking at it, to say, whether I have to do with gout or rheumatism. But I ask you, whether any reasons exist why more should be exacted of me than of the naturalist? Among the ablest botanists, is there one, who by merely looking at two leaves, each belonging to different plants of closely allied species, will name the species of plant from which each leaf has been taken? Before giving his opinion, he must see the fruit. Well then to continue the comparison, I also require to see the fruit of the gout. If I find that the articular affection has been followed by tophaceous products—if the patient tell me that he has experienced symptoms characteristic of gravel—if I make myself perfectly acquainted with the mode in with the local affection was evolved—my diagnosis is made certain. It is not different in gout from other diseases. The first view is often insufficient to discover the specificity, and it then becomes necessary to examine the phenomena in detail.

A patient is suddenly seized with symptoms of a more or less serious character, and implicating the lungs, intestines, or brain. These symptoms have suddenly supervened, and progressed in a peculiar manner, so that at first you do not know to what to attribute them; but you learn the patient is the child of gouty parents, or of parents subject to attacks of asthma or megrim; and then you suspect the existence of the diathesis of which the visceral affections are the manifestations. Should the patient tell you that he has had attacks of gout, and that the disease has been abruptly suppressed, your suspicion becomes complete conviction.

I shall not now expatiate at greater length upon the subject of specificity, as I have already very fully discussed it with you. I have now to speak of *diathesis*, a topic not less capital in relation to gout, and one intimately linked with the subject which I have just been recalling to your recollection.

The word "diathesis" has been employed in very different significations. I have already told you the meaning which I attach to it. Without now troubling myself by endeavoring to find a new definition, I shall take that given in the Dictionnaire de Médecine of MM. Littré and Ch. Robin, the most complete vocabulary which we possess:* "Diathesis [*διαθεσις*] is a general tendency, in virtue of which an individual becomes the subject of several local affections similar in their nature." This definition, recommended by its brevity, perfectly expresses the idea which I wish to express.

An individual, under the influence of a special cause, in consequence of an accidental wound, or as the result of a surgical operation, forms pus, which accumulates in abscesses in different parts of the body. He is, we say, under the influence of the *purulent diathesis*, meaning thereby, that he has a special tendency in virtue of which he becomes the subject of numerous purulent affections.

Another individual contracts syphilis: the special tendency engendered by the introduction of the syphilitic virus into the economy shows itself by very different lesions affecting different tissues; but these lesions, however different they may be in appearance, all arise from the same cause, and are, in reality, all of the same nature. In scrofula, cancer, and all affections depending upon a diathesis, be the diathesis acute or chronic, what takes place is exactly similar. The morbid localizations are nothing more than manifestations of one general dominant tendency. But a difficulty seems to suggest itself! The general tendency is very far from constantly showing itself in the same organs, the same tissues, and the same anatomical elements. The peculiar character of the organs, tissues, and anatomical elements imparts very different appearances to the affections with which they are attacked.

To those physicians in whose eyes localization constitutes the particular disease, the differences in appearances are so many different diseases, while to those who consider that the disease consists much more in the aggregate of the general phenomena, in their evolution, in their progress (and that, thank heaven! is the direction in which sound observation leads), these affections, differing in appearance, are only multiplied expressions of the same species of morbid action. To the real physician, exostosis, alopecia, psoriasis, roseola, bubo, and chancre are always syphilis—syphilis in different garbs.

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* Twelfth Edition (Paris, 1865), p. 444.

I have taken syphilis as an example, because no one will venture to raise his voice in contradiction of a fact which is now so well established—because no one could mistake pox for anything else, infinitely varied though the forms be under which it clothes itself. I have spoken of the most common manifestations of this disease; but under how many other forms, of which we cannot always at once appreciate the nature, is it not concealed! How many nervous symptoms which appear as its sole phenomenal expressions are dependent upon it, symptoms which remain inexplicable till the more decided characters of the diathesis which has produced them afford the key to the diagnosis!

The statements which I have made regarding syphilis hold good in respect of many other diseases, and particularly of gout, the manifestations of which are infinitely varied; and which you will have to recognize under the different disguises by which it is so often covered.

But there is still another point upon which I wish to insist—that though the diathetic tendency generally shows elective affinities for a certain number of organs, rheumatism for the large and gout for the small joints, and for certain small joints in particular, there are many circumstances in which these elective affinities seem to disappear, the diathesis presenting unexpected localizations.

It has seemed useful to call your attention to these great facts in general pathology before commencing the history of the disease which is to form the subject of these lectures.

Gout is an admirable name, because in whatever sense it may have been originally employed by those by whom it was invented, it is not now given to anything else than that to which it is applied. How often have I pointed out to you the value of names, which from being independent of all scientific pretension, are appropriate *uni et toti definito!* The names *goutte*, *vérole*, *variole*, and *coqueluche* are all the better that they have but little nosological meaning. They are excellent names, precisely because they imply nothing doctrinal, because they find a place in every system of nomenclature without expressing any article of pathological belief; they are irrespective of all theories; every one is satisfied with them, and understands them much better than the barbarous Greek and Latin terms by which it has been attempted to supersede them. "Gout," therefore, is the best name which we can make use of; it is much to be preferred to "podagra" (*podagre*), in favor with those authors who have written in Latin, and which has the disadvantage of signifying pain in the foot, and consequently of only describing a part of the condition which it designates, and therefore requiring the complementary terms "chiragra" (*chiragre*), "ischiagra" (*ischiagre*), &c., signifying pain in the hand, in the lumbar articulations, &c. For the same reasons it is preferable to "arthritis," which, although a term more general in its signification, has the inconvenience of only expressing the local affection of the joints, and not embracing those others so frequently present.

I now enter upon my subject.

Gout is said to be "regular" or "irregular," according as its character is frankly inflammatory, acute or chronic—according as it reveals itself by other affections involving certain viscera, or as its seat is difficult to determine.

Regular gout is the form which authors have had specially in view in that which they have described under the name of "podagra;" because in it the articulations of the foot are those which are usually implicated, particularly in first attacks.

It is podagra, or to use a better term, gouty arthritis, which we have to study in the first instance. Always remember that although local inflammatory action of the joints is more particularly characteristic of gout, it is not the less necessary to take great account of the totality of the general precursory or concomitant symptoms which stamp the disease with the seal of specificity.

The *premonitory symptoms* of an attack of gout indicated by Van Swieten, Scudamore, and other scrupulously careful observers, have engaged the special attention of Dr. Galtier Boissière, who in his inaugural thesis (to which I referred at the beginning of the lecture), has largely developed their picture.

In the *organs of digestion*, the dyspeptic symptoms, consisting in gastric disturbances, though not alarming, are generally, at least, very conspicuous. The appetite is diminished and irregular, presenting caprices unusual to the patient. He will, for example, select stimulating articles of diet, meats strongly spiced and acids, as if he felt the necessity of stimulating the lazy functions of his stomach. After the repast, he will complain of gastralgic pains, of weight, and a feeling of fulness in the region of the stomach; he will have flatulence accompanied by vomiting of pituitous matter, and nidorous eructations, at times tasting of rotten eggs.

In some cases, there will be a complaint of aching in the right hypochondrium: and the physician will detect slight tumefaction of the liver. This symptom, noted by Scudamore, who believed that the hepatic affection might proceed to a material change in the structure of the organ, noted also by Portal and Galtier Boissière, is perhaps caused partly by the dyspeptic disturbances of which I have been speaking.

But the most prominent premonitory symptoms of an attack of gout are *disturbances of the nervous system*. The gouty subject, at this period of his attack, complains of weight in the head, and inaptitude for every kind of intellectual labor: the altered cerebral condition is indicated principally by a nervous excitability which is often excessive both in regular and irregular gout, but particularly in the latter. This nervous excitability shows itself by phenomena varying much in the character according to the individual. There is an indefinable character of discomfort, and mental uneasiness; and curious changes of disposition. Though some persons show an exaltation of their brilliant qualities, this is far from being always the case. Gouty persons generally acquire a morose, susceptible, and irascible temper, formerly foreign to them. This is so usual that it has passed into a proverb with authors on gout. So great, sometimes, is this perversion of disposition, and so constant is it in some persons, that not only the individuals themselves know that an attack is coming on, by feeling causelessly cross for some days, but also those about them can foretell the attack by these moral phenomena; just as the catamenial period is announced in some women by a manifestation of changes in the mental condition.

In the urinary system, something peculiar sometimes declares itself. The urine assumes an unusually red color; and deposits a sediment resembling bright pink sand, or pounded brick. The passage of this urine through the urethra causes pain, a sensation of heat, sometimes smart burning; and it is even not unusual for this irritation of the urinary passage to produce blennorrhagia.

Gouty blennorrhagia is particularly observed in anomalous gout, and is then unconnected with the emission of urine charged with sand; but, I repeat, that it is not unusual to meet with it in the frankest possible attacks. Though this blennorrhagia is attended by more or less acute pain, it ceases spontaneously, and soon. The physician ought to be aware of this fact, so that when the discharge shows itself, he may be able to calm the anxiety of the patient, and state the real nature of the affection.

Blennorrhagia is not the only catarrhal affection which complicates an attack of regular gout. The catarrhal predisposition was clearly indicated by Barthez. Scudamore has mentioned *ophthalmia*, which in some subjects supervenes a day or two before the attack. Dr. Galtier Boissière has also noticed it. In some cases, it assumes a very intense form.

Gnashing the teeth is a premonitory symptom which I have only found mentioned by Graves.* The celebrated clinical professor of Dublin says that the patients have an irrepressible desire to grind the teeth, and that this is excited by painful sensations in the urinary organs which they fancy cannot be otherwise appeased. The urgency of the desire to grind the teeth is so great in some gouty persons, that at last their teeth become worn down to the sockets.

Such, then, are the premonitory phenomena of an attack of gout. Afterwards, the disease declaring itself more and more, the *arthritis* becoming more imminent, it is found upon examining the regions which are about to be affected, that they exhibit a peculiar swelling of the veins. Sydenham says: "*Quod in omnibus podagricorum paroxysmis solemne est, insignior intumescentia venerum membro vexato intertextarum se in conspectu dat.*"

The nervous symptoms of which I have been speaking generally disappear with the appearance of the more characteristic local phenomena of gout: sometimes, however, they remain, and complicate the articular pains. They may even assume so great a degree of intensity, that the bodily discomfort and mental anxiety torment and weary the unfortunate sufferers at least as much as the pains in the joints. "*Ut haud facile sit dictu utro horum æger calamitosius doleat:*" they sometimes proceed to such excess, that, to continue the quotation from Sydenham—every attack is an attack of rage as well as of gout—"*non rectius podagræ quam iracundiæ paroxysmus omnis dici potest.*" Independent of these phenomena—whether they exist or do not exist—the nervous excitability shows itself by spasms of the members which are affected by gout. The patients complain of tremors, shiverings, cramps, and very painful convulsive seizures.

Gentlemen, throughout all the morbid conditions which we have now been rapidly reviewing, have we not already seen that the diathesis is in action before there is time for the local affection to show itself in a precise form? Have we not seen that gout explodes on all sides before it is definitively installed in its chosen seat? Long before any pain has been felt in the articulation destined to be the seat of suffering, the whole economy is a prey to the diathesis by which it is impregnated: "*totum corpus est podagra.*"

At last, the time comes when the malady explodes—when the real attack begins. For some hours, sometimes for a whole day, the precursory symptoms had ceased. The patient feels better, but this improvement, when he has had experience of previous attacks, does not deceive him: he knows that it is but a truce, the prelude to a more formidable assault which he will have to sustain. He goes to bed at night apparently quite well, or at least more hearty (*alacrior*) than on the previous day; and sleeps quietly (*sanus lecto somnoque committur*). Then, suddenly, generally between midnight and three in the morning, according to the time which has elapsed since he went to bed, he is awakened by pain, which is generally localized in one of the great toes.

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* GRAVES: Leçons de Clinique Médicale, trad. par Jaccoud, 2me édit. Paris, 1863, t. i, p. 598.

It is a remarkable circumstance, one quite inexplicable, nevertheless noted by all good observers, that attacks of acute gout almost always set in during the early hours of the night: Sydenham says about two o'clock. It is also a fact confirmed by observation, that the part attacked is usually the metatarso-phalangeal joint of one of the great toes. A tabular statement drawn up by Scudamore shows that this occurred sixty times in one hundred cases.

The pain at first resembles that of a dislocation (*ossium dislocatio*). To ease it, the patient rests the foot on its outer side, every moment changing its position in trying to find an easy position, but he never finds one which he can bear even for a few minutes. Should he try to sleep, the pain gives him no truce: it becomes more and more intense, and ere two or three hours have elapsed, has become quite intolerable. Those who have endured this suffering compare it to the fancied sensation of a nail being driven into the joints—to tearing of the flesh by powerful pliers—to the teeth of a dog crushing the bones—to squeezing in a vice—to the penal torture of the boot-screw, when the tormentor is constricting the limbs of the victim between planks of oak, and forcing with his mallet the corners into intervening spaces. In a word, the victim of gout describes in the most terrible language the infernal pains which he endures: *nunc tensionem violentam vel ligamentorum dilacerationem, nunc morsum canis rodentis quandoque pressuram et coarctationem exprimens.* His tortures are all the more cruel that the startings of the limb prevent his keeping the foot at rest. His pains soon attain such intensity that he can bear nothing upon the affected part. The contact of the bedclothes is intolerable; and to obtain protection from it, he lifts them up with the free foot. Should he unfortunately live in a stone-paved street, and his lodging be situated in the upper stories, vibration of his bed caused by the vibration of the building—felt much more than in the lower stories—makes the wretched sufferer mad with rage when a heavy vehicle passes. He is distressed by the least movement, so that no one dare walk across his room with a heavy step, far less touch the bed on which he is lying.

To have a right conception of the distressing state induced by an acute attack of gout, one must have been present at the painful spectacle: when once we know the extent of the suffering, we will be indulgent to patients who in their despair invoke the aid of all who promise them speedy relief from pain. Although your conscience prevents you from prescribing energetic remedies, which cut short attacks of gout, but produce future evil consequences, you can make allowance for the too legitimate impatience of the sufferers: you can understand why many prefer to incur the future evils with which you threaten them, than to continue to endure the dreadful present pain.

The excruciating pains, however, cease spontaneously. In a frank attack of gout, they diminish towards morning, *sub galli cantu*, as Sydenham says, while at the same time, there is a remission in the local increase of temperature and the shiverings by which the pains are accompanied. There is slight perspiration; and the patient is at last able to sleep. When he awakes, his pains are much less acute; and he observes that the part in which they are situated is red and swollen. After this, he generally gets through the day without much acute suffering; but towards evening, the pains acquire renewed intensity: at night, they are as severe as they were on the previous night: again, towards morning, they abate, and during the day, become as much lulled as on the previous day. This cycle of remission and reaccession of severe pain lasts for from four to eight nycthemera.

There is at last an end of the crisis. The pain gradually becomes less and less; and is ere long nothing more than an uncomfortable numbness (in first attacks) which continues for eight or ten days, in persons of fifty years and upwards, but for a shorter period in those who are not so old.

In addition to the articular pain, of which I have tried to give you an idea, some patients complain of its being often accompanied by strange sensations. Some say that they feel as if a jet of slightly tepid water [*aquæ tantum non frigidæ*] was trickling along the limb corresponding to the foot attacked by gout; while a greater number state, that the seeming trickling is like that of boiling hot water or molten lead. There are others again who complain of an icy cold sensation. "Some patients," observes Ambrose Paré, "say that they burn, while others complain of icy coldness."

Let me now describe the aspect of the affected parts. Suppose that the part attacked is the great toe, which, I repeat, is by predilection the seat of the attack. The subcutaneous veins of the part, as well as of the neighboring regions, are much swollen, as in some cases of articular rheumatism. Though there be this point of resemblance between the two diseases, there is also this difference, that in gout, the swelling of the veins, which not only occupies the foot but likewise the neighboring parts, extending to the leg, precedes the other symptoms of articular inflammation: and although in rheumatism as in gout, there is more or less swelling of the parts accompanied by bright redness of the skin, this redness has a peculiar appearance in gout, which is very different from the bright redness of rheumatism. It is a redness resembling that of the peony rose: the skin has a shining gloss like the peel of an onion: it bears some resemblance to the appearance of an abscess about to perforate the external integument by its walls becoming thinner.

If you slightly touch the toe, or even pass the finger lightly over it, you excite excruciating pain, which will extend beyond the affected joint, and have a response in the instep. The redness is not confined to the seat of pain: it spreads gradually over a certain surface: both where it is visible and beyond that limit, an œdematous swelling is perceived, and the skin retains for a considerable time the marks made on it by pressure.

The redness, after attaining, in twenty-four or thirty hours, its maximum intensity, diminishes, or at least is replaced by a violet hue, in proportion to the degree in which the pain decreases. The œdema, on the contrary, still goes on increasing for from four to six days: when at last it has disappeared, and the attack is at end, the joint remains stiff. There is difficulty in walking, which difficulty is enhanced by weakness, and a diminution in the sensibility of the skin in addition to the stiffness. From the beginning of the attack, the gouty patient declares that his foot is soft, and to adopt his favorite expression, it is cotton, which means, that with wide shoes, and upon very even ground, he walks with hesitating step, unable to feel the ground on which he treads. Ten, fifteen, twenty days, or more, elapse before the joint regains its normal movements.

Gentlemen, in the rapid sketch of acute gout which I have now given you, I have represented a first attack occurring in a robust young man.

At the end of the attack, and when the pain has yielded, the parts which have been affected perspire spontaneously. Some days later, the skin of the same parts slightly desquamates; and generally—I might almost say invariably—also becomes the seat of itching, a phenomenon peculiar to gout, and not observed after attacks of rheumatism.

It is unusual for more than one joint to be affected in a first attack of gout: nevertheless, in persons who may be said to be of gouty breed, the two great toes may suffer at an interval of a few days, or even simultaneously. The attack has, in general, been announced by prolonged precursory phenomena: in general, also, the foot last seized is the least affected, and the soonest to get right again. The consecutive œdema, likewise, is of shorter duration.

Sometimes, but less frequently—speaking always of persons hereditarily gouty—the joint of the great toe and other articulations of the foot, the great toe and the *tendo Achillis*, the foot and the knee, the articulations of the foot or hand, are implicated in a first attack; in subsequent attacks, the progress of the disease is different. The symptoms of the disease are very different in respect of the duration of the paroxysm, the form of the manifestations, and the concatenation of the symptoms.

Here, gentlemen, we are able to see clearly the nature of the analogy between gout and rheumatism—we see that the symptoms do not progress *uno tenore*, that they consist in a series of little attacks—*series et catena paroxysmulorum*—to use Sydenham's expression. For five or six days, the pain goes on increasing: it then becomes less severe; and the fever subsides. This ameliorated condition continues for from seven to fifteen days; the patient is under the belief that he has got rid of the enemy, when, quite suddenly, the fever lights up again, and a new attack sets in, which, however, does not last so long as the attack which preceded it. Convalescence seems again to have commenced, when, in their turn, other joints are seized, and the attack lasts for from six weeks to three months. Am I not entitled to tell you, that there is an analogy between gout, characterized by a series of paroxysms, and acute rheumatism, which, at intervals of longer or shorter duration, involves all the joints, not previously implicated?

In this form of gout, the symptoms set in after the same fashion as in a frank attack, which may be considered typical, with this exception, that the season of appearance is not the same.

I have not yet mentioned that a first attack of acute gout usually occurs in winter, towards the end of January or beginning of February. Is this remarkable fact to be explained by winter feeding being more succulent, by animal food being the chief aliment, and there being a dearth during winter of fresh vegetables and fruits, which constitute so suitable a regimen for gouty persons? Is it explained by the circumstance, that it is usually in the winter months that festive meetings take place, when the most sober are apt to be led more or less to renounce the habitual regularity of their accustomed mode of life? The explanation is admissible, but it is also very disputable. However it be accounted for, the fact is indisputable, and has been noticed by the best observers. *Gout with successive paroxysms* shows itself early or late in the year, that is to say, at the beginning of spring or end of autumn. The wherefore, I know not.

This form of gout with its series of attacks always sets in in the same manner as the other: it is announced by similar precursory phenomena, but it gives a longer notice of its coming: its prodromata are more decided, and the person who has once experienced them is never afterwards mistaken as to what is going to happen when he feels them.

Although this form of gout is generally the sequel of a first attack of regular gout, it may also declare itself all at once in its specially characteristic form.

It is not, I repeat, one joint only which is the seat of the gouty manifestations—it is not the foot only which is attacked, as in frank gout, although that does occasionally happen: sometimes, it is the knee, sometimes the elbow, sometimes also, though not often, the hands. After having continued for seven, eight, ten, twelve, or fifteen days, in one or more situations, it leaves them, to take possession of others: and every time, the inflammatory affection (which invades several joints at the same time) is accompanied by the same general phenomena, by fever, horripilation, and spasmodic symptoms.

I have already told you that the attack consisting of little paroxysms may last for some weeks, or even for three months. Should it extend beyond that period, it is no longer acute gout: it is chronic gout, which one must be careful not to confound with anomalous gout, even when accompanied by anomalous symptoms.

The older the patient, the more prolonged is the attack: and the attack is also more prolonged in proportion to the age the patient has attained without having had an attack, and the length of time which has elapsed since the last attack. An individual, for example, has, for the first time, been seized with gout at the age of twenty; although it is not generally till forty that we meet with attacks made up of a chain of paroxysms, unless brought on by some immediately exciting cause, such as bad fare, or injudicious medical treatment, which by arousing the diathesis, causes the attack to occur sooner than would otherwise have been the case. The attack may be brought on by an injurious effect produced upon a joint by a blow, by overwalking, by the pressure of new shoes, or in fact, by any other mechanical violence; and it is not unusual for the first attack of gout even when of the frankest description, to have been induced by a cause of this nature. The attack may likewise be the sequel of a course of mineral waters, or of any other unsuitable medication: it may also be determined by the fever which ushers in a pretty severe cutaneous eruption, an occurrence of which I have lately seen an example. But an attack which has not been preceded, as usual, by the general phenomena which announce an attack occurring without appreciable cause, is also of much shorter duration. Besides, when ended, it leaves behind it fewer consequences. There is less articular deformity, and what there is, is less persistent. The patient sooner regains freedom in his movements. To use the language of our predecessors, it would appear, as if the morbific matter is not at that early age sufficiently prepared to produce at once all its effects.

This rule, however, gentlemen, is far from being absolute: to it the exceptions are numerous, and a first attack, even when it comes on accidentally, may not only last a long time, but may also leave marks behind it, as serious effects as those which remain after gout characterized by often-repeated paroxysms. I know a physician, the child of gouty parents, whose first attack was occasioned by dislocation of the knee. The joint never got quite right; and the sequel of the accident was lameness, which still remains. Such cases, though exceptional, are sufficiently common to make it very necessary for you to be aware of their occurrence.

REGULAR CHRONIC GOUT.—*Consecutive Deformities of Joints.—Tophus a manifestation only met with in Gout.—The Visceral Complications are very different from those which constitute Anomalous Gout and Paludal Gout.*

GENTLEMEN: The remarks which I have made on acute gout having successive paroxysms, leads me to speak of chronic gout, into which it frequently degenerates. Chronic gout may be *regular*, *irregular*, or *anomalous*.

Regular chronic gout is generally met with in men in the decline of life. It is also the form which the disease assumes in women, who, as you know, are much less subject to gout than men. In the male subject, it does not generally appear till after fifty years of age; but it is nevertheless not very uncommon to meet with it in men of thirty or forty: in these cases, the patients have been sufferers from frequent attacks of the disease in its acute form, in early life, at about twenty or twenty-five years of age, or sometimes earlier. This acute gout, almost always, if not always, hereditary, is the more likely to become chronic, the more it has been meddled with; the more that time has not been given the disease to develop itself before it has been vigorously assailed, the more that in its first paroxysms an attempt has been made to abort its crisis; and finally, the more that after the imprudence of interfering at the wrong time, the patient has not been subjected to a regimen calculated to compensate for the evil caused by the perturbatory treatment.

Regular chronic gout, in respect of the frequency of the recurrence of the paroxysms, resembles acute gout with successive paroxysms, there being this capital difference, however, that its attacks are longer, and during the intervals, the symptoms are not entirely absent. The attacks, in place of lasting for four, five, or six days, continue for fifteen, twenty, or thirty. Again, four, five, or six joints are always either simultaneously attacked, or attacked in such rapid succession, that no sooner does one joint get free, than another is seized; and so on successively with other joints. The inflammatory manifestations lead to engorgements of the affected parts, which continue with inveterate obstinacy. The feet, tibio-tarsal articulations, wrists, and elbows remain swollen: and the œdematous sort of tumefaction, which often extends beyond the joints, simulates white-swelling [*tumorem subalbum concitantes*]. The comparison is the more applicable, that the extremities of the bones which enter into the joints are affected, and their periosteum implicated—that there exists a veritable *dry arthritis*—that the swelling which results from this ostitis and periostitis is complicated with the production of tophaceous deposits, of which I shall afterwards have to speak.

The articular affections never altogether disappear, and the joints never regain their original suppleness of movement. Although in acute gout, once the attack is over, the patients get back perfect freedom of motion, in chronic gout, the movements are more or less interfered with. False anchylosis, to a greater or less extent, takes place; which is the result both of the inflammation of the parts, and of their having remained for a long time in an unnatural position. Walking is difficult, and sometimes impossible; and this impossibility depends not only on the lesions of which the limbs are the seat, but likewise on the general debility of the system: for there is a sensible change in the state of health, even when there are none of those well-marked visceral disorders, which at a given moment frequently become epiphenomena of the disease.

The *visceral disturbances* which supervene, coming on more or less quickly according to the individuals, consist in palpitations of the heart, and difficulty of breathing, phenomena which are sometimes purely nervous, but also, at other times, dependent upon organic lesions of the heart and great vessels: visceral disturbances also consist in pulmonary and intestinal catarrhal affections, the latter manifesting themselves in diarrhœa, and in some cases in dysentery. You can understand, gentlemen, that under the influence of the perturbation experienced in the digestive and respiratory functions, disturbance of the plastic functions leading to debility and loss of flesh are not long in appearing: these conditions under the influence of atmospheric changes increase the severity of the usual neuralgic pains. The victims of this cruel form of gout rapidly pass into a state of premature senility.

Deformities are the most common *bad consequences to the joints* of chronic gout. The articulations of the foot usually suffer from various forms of club-foot, particularly *pes equinus*. It is easy to understand the mechanism by which this deformity is produced. It is the result of the continuous pressure, for weeks, of the affected foot, exercised by the weight of the blankets upon its extremity placed in a vertical position. But the circumstance which contributes perhaps most of all to produce them is painful contraction of the posterior muscles of the leg, by which the heel is bent backwards, and the foot pushed forward. When the attack is over, the patient finds that he limps exceedingly, and can only walk on the tips of his toes. It is necessary, therefore, both for physician and patient to do all that in them lies to prevent this deplorable deformity, by supporting the foot in a proper position during the whole of the attack by pillows and a trough, and by protecting the foot by a cradle from pressure by the weight of the blankets.

Club-foot, however, is not the only deformity of the same kind chronic gout may leave behind as a legacy. Other articulations may be seized in the same way. For example, it is not unusual for the knees to remain bent, from the inferior extremity having been long kept in a bad position, and from painful contractions of the muscles which flex the leg upon the thigh, contractions which may be repeated at very short intervals during an attack of from six to eight months. At last, the leg remains irrevocably bent upon the thigh, which is flexed on the pelvis.

I was acquainted with one of the great noblemen of England, who from his youth had suffered horribly from gout, who for many years was totally deprived of the use of his legs, which were semiflexed and anchylosed, consequent upon a prolonged attack of chronic gout. This unfortunate sufferer was a cripple reduced to the necessity of sitting squat on a little platform, and making powerful use of his arms when he wished to move from one place to another.

It sometimes happens that there supervene acute attacks, attacks as acute as in frankly inflammatory regular gout. These attacks are the more readily induced that, during the interval between them, the patients have had a voracious appetite, which they have given free scope to; and that the impossibility of moving has prevented them from taking any exercise to aid digestion which is performed with great difficulty.

The acute excessive pains are essentially of a transitory character, and affect sometimes the articulations which were formerly the seat of chronic gout, and sometimes others not previously affected. Resembling the pains of acute regular gout, like them, they declare themselves during the night, awaking the patient with a start, which they compare to the grasp of an iron hand, or a blow with a club [*ictus quasi clavæ*]. The sufferings are still farther increased by the pains declaring themselves in the course of the nerves supplying the affected parts, and by cramps in the corresponding limb. The cramps make the patients cry out with pain, and would be insupportable if they lasted a little longer: "*Si vel tantisper durarent humanam patientiam dejicerent vincerentque.*"

To fill up the measure of the misery of the patients, they have, in addition to the articular, neuralgic, and muscular pains, to endure nephritic colic, which is perhaps a still more cruel kind of suffering. This symptom, in itself horribly painful, exasperates the tortures endured by the patient, whilst the vomiting, which accompanies it shakes violently his body.

In speaking to you, gentlemen, of larvaceous gout [*la goutte larvée*], let me say that the gravel on which depend attacks of nephritic colic and gout are sisters, to adopt the expression of Erasmus: "I have nephritis," he wrote to one of his friends, "and you have gout: we have married two sisters." To adopt a more medical phraseology, gravel and gouty arthritis are manifestations of the same disease. The former belongs to the visceral affections to which I have just been directing your attention. It is the result of the disturbed secretory functions of the organs whose special function it is to eliminate the urates and the uric acid generated within the economy, and carried along in the current of the blood, while at the same time, the sweat is modified, both in quantity and quality, consequent upon a deranged state of the cutaneous functions. The most remarkable result of the disturbance in the functions of the skin is the production of the cretaceous concretions described under the name of tophus, and regarding which I have intentionally refrained from speaking till now, that I might do so at greater length.

Gout is the only disease in which *tophus* is met with. What then is tophus?

Sometimes, after an attack of gout, more or less acute, more or less prolonged—sometimes, also, when the patient has not had a very severe attack—we see formed under the skin around the joints, tumors more or less projecting, hard, polygonal, and not round, but having a mossy beard: they consist of accumulations of calcareous salts which chemical analysis shows to be a mixture of soda and urate of the phosphate of lime, the phosphate being always proportionately in smaller quantity than the urates. These calcareous deposits sometimes form in the interior of joints, and when they are very large, the surfaces of the bones lose their normal relations; whence arise deformities, which increase those already produced by faulty positions and contractions of the limbs.

Deformities produced by tophus present a peculiar appearance. The fingers, when their joints are the seat of the concretions, become warped, shortened, and irregularly knotted. Nothing of this sort is observed in rheumatism, which often leaves swellings behind it, but they are pretty regular in form, the affected joints presenting a spindle shape. I do not refer to that kind of rheumatism called *knotty*, remarkable examples of which you have seen in a patient in St. Bernard Ward, and in a man who was for a long period attendant in St. Agnes Ward.

The occurrence of tophus in gout is something far too special to escape even the most superficial observation; every author mentions it as one of the most characteristic features of the disease.

When an opportunity occurs of making an autopsy of a gouty subject—such as we have twice had, and in particular in a case which presented itself two years ago in a person who died of gravelly gout in our clinical service—we find on opening the affected joints, the articular surfaces covered with patches consisting of layers of various sizes more or less uniformly distributed, composed of a whitish chalky substance, penetrating sometimes into the substance of the cartilages. The affected articulations are generally dry; and this absence of synovia explains their stiffness during life. There are cases, however, in which instead of a diminished, there is an increased secretion of synovial fluid; and this increase may proceed so far as to be true hydrarthrosis.

I have already stated the chemical composition of the calcareous deposits. They are largest when external to the joints. Generally, tophitic formations are small; but it is by no means unusual for them to be of considerable size. They may be as large as a hazelnut, or a pigeon's egg, and sometimes they attain the size of a small hen's egg. They may remain quite independent of the skin, which glides freely over these tumors, which form, and then become detached. It also often happens that their presence ultimately produces irritation of the integument by which they are covered. The skin then assumes a purple hue, becomes thin, and ulcerates; at the bottom of the ulcer which is formed—generally an indolent fungous ulcer—calcareous deposits may be perceived, which can be easily detached by a pointed instrument. As they are forthwith reproduced when removed, large quantities of them are collected in some cases. Without accepting as verities the poetic exaggerated statements, to the effect that a certain Baylas and a certain Acragas were incased when alive in chalk, which they secreted in such frightful quantities that their tombs might have been constructed from the material they supplied, it is a fact, that gouty subjects sometimes produce one hundred, two hundred, or three hundred grammes of calcareous matter.

The time comes when the ulceration which furnished these tophaceous products dries up without causing any great amount of suppuration: and this occurs whether the concretions do or do not become detached: the wound then closes, leaving a small cicatrix, which at a later date, on the occurrence of a new attack of gout, will reopen, again to close and again to reopen. When the attacks follow one another at very short intervals, the tophaceous deposit accumulates round the joints, invading all of them, as happened to Gordius, who composed upon himself the following jocular epitaph:

"Nomine reque duplex ut nodus Gordius essem."

These calcareous deposits form elsewhere as well as around the articulations. I knew an individual, the tips of whose fingers were garnished by them: in another person, the entire skin of the palm of the hands and the plantar surface of the feet were covered with chalky patches resembling the atheromatous concretions sometimes met with on the internal coat of arteries. A lady of sixty had the cutaneous palmar folds of both hands marked with white radiating lines such as are seen in those who have long been employed in tempering plaster.

It is rather remarkable, that tophus very often occupies edges of the ear: indeed, in some persons, they are even met with in that situation before the gout has distinctly shown itself. They then constitute a diagnostic sign distinguishing gout from rheumatism. I have often observed them on the lobule of the ear, as was noted by Plater, who mentions that he had a patient whose whole body, even the eyelids, was studded with them: "*Ex toto corpore, per poros, adeo ut etiam palpebræ oculorum non exemptæ fuerint, ejus modi materia gypsæa, circa poros cutis mox in tophos mutata, prodisset.*" Léger, author of a treatise on gout, published at Paris in 1753, states that he found these concretions in the lungs.

Although I cannot adduce any necroscopic observation of my own in support of the opinion, I think the question may be asked—whether certain formidable vascular lesions, aneurisms for example, may not have as their primary cause similar concretions on the internal coats of arteries? May we not attribute certain cerebral symptoms met with in gouty persons, such as vertigo and symptoms indicating softening of the brain, to the formation on the arteries of the encephalon of these gouty concretions?

Physicians who have watched the progress of the evolution of tophus believe that it is formed during the paroxysm of gout. They are mistaken: the deposit appears during the interval between attacks, or, at least, when the attacks have not been of long duration, and when they do not recur in such rapid succession as to run into one another, in which cases, their secretion has commenced during the preceding, and continued during the succeeding attack.

While, however, tophaceous concretions generally show themselves after attacks of articular gout, cases occur, as I have already told you, in which the secretion of calcareous matter takes place irrespective of any arthritic attack. This sort of *cutaneous gravel*, if I may employ a comparison based on the great analogy between the composition of urinary gravel and tophaceous concretions, gravel of the skin, constitutes the sole manifestation of the diathesis, and is accompanied merely by a slight feeling of pain, of pricking, unattended by any disturbance of the general health.

I have just been speaking to you of the elimination of tophus from cutaneous ulcerations. This elimination only takes place when the tophaceous concretions have obtained a pretty considerable volume. When the deposit is small in quantity, it is pretty easily absorbed; and nothing is so well calculated to promote this absorption as regular exercise combined with suitable regimen, two points of capital importance, upon which I shall have to insist when I come to speak of the treatment of gout. It is generally in the first formation of tophus that absorption is observed. The subcutaneous tumors which are formed completely disappear: the joints, the movements of which were impeded by their presence, and by diminution in the synovial secretion, regain their free mobility, and move without occasioning crepitation to indicate extreme dryness of their surfaces.

Regular gout may *supervene suddenly, and be chronic:* that is to say, its outbreak need not have been preceded by paroxysms in any way characteristic of acute gout.

In attacks subsequent to the first, the gouty person is subject to less urgent symptoms: the articular inflammation has little violence, is not extensive, and the pain is much more obtuse than in acute gout. It does not interfere with sleep: sometimes, the patient is even able to walk, and the œdematous swelling of the parts is transient. The paroxysms, however, have a longer duration than those of acute gout, and recur, at longer or shorter intervals, during several months, or even years: they soon approximate, last longer, and leave the patient during his short respite, a valetudinarian, sensitive to atmospheric variations, subject to general disturbance of health which I have pointed out to you, and which is often connected with appreciable organic affections.

The combination, in some cases to a very great degree, of organic lesions of important viscera with frank articular manifestations of gout, may cause one to believe in metastasis of gout to the viscera, although in reality there is only an exaggeration of the morbid phenomena which precede and accompany both chronic and acute gouty arthritis.

Till now, we have not had under consideration anomalous gout properly so called: in it, the visceral predominate over the articular manifestations. I now proceed to speak of larvaceous gout, which as yet I have not discussed.

LARVACEOUS GOUT.—*Comparison of it with Palustral Larvaceous Fevers.—Megrim: Asthma: Neuralgia in various forms: Gravel: Hæmorrhoids: Cutaneous Affections: Anomalous or Visceral Gout.—Bright's Disease.—Pulmonary Catarrh.—Suppressed Gout.*

GENTLEMEN: The questions which arise in relation to larvaceous gout, called *arthritis larvata* by Stoll, are assuredly among the most difficult in pathology; for, to discover the disease under the mask which it assumes, there are required not only great experience, but likewise a most scrupulous attention. Even with the most consummate experience, and the most scrupulous attention, mistakes in diagnosis are frequent. You know what is meant by "a larvaceous fever." Under the influence by which it is produced, the palustral diathesis, predominating in the economy, reveals its existence by morbid phenomena essentially different from those which characterize the paroxysm of a normal intermittent fever. The phenomena to which I refer are neuralgic affections, disturbance of the cutaneous or intestinal secretions, thoracic, or cerebral symptoms: they are, in a word, various affections, which when they appropriate an unknown something, become malignant, constituting what are called pernicious fevers, which must not be confounded with simple larvaceous fevers.

There is a larvaceous form of gout as well as of fever: the gouty diathesis may declare itself by affections essentially different from those which characterize ordinary gout. As they may constitute its primary manifestations, the difficulty of detecting the nature of the disease can be easily understood.

To give you a striking example, let me recall to your recollection a case which I related on a former occasion.

I had as an intimate friend an English major, who had been long subject to megrim, recurring with a periodicity so well marked every second Wednesday, that almost the exact hour of the seizure was known. So regular were the paroxysms in their progress and duration, that, strange to say, the time of their termination could also be foretold. They lasted some hours, and then left the patient in perfect health. His first seizures occurred during a sojourn in the Antilles: from that date the attacks never failed to recur on precise days. Matters were in this state when I became acquainted with this gentleman in Paris. He was so worn out with his sufferings that he asked me, at any price, to deliver him from them. This occurred in 1825, when I was just commencing the practice of medicine, and did not know what megrim was. Acting under the advice of some of my professional brethren, I put the patient upon a course of strong doses of aloetic pills (*pilules Ecossaises*). Under the influence of this repeated purging the attacks lost their periodicity, and occurred at longer intervals; but these changes did not prove very beneficial to the general health. Previously, the paroxysms had been succeeded by a condition of well-being, which contrasted in a remarkable manner with the feelings of discomfort which gave notice of their return. There also occurred in this case, that which occurs in persons under the dominion of diathesic periodicity, in those who are gouty, in those who are hæmorrhoidal, viz., a state of indescribable discomfort preceding the attacks, which, when they set in, are assuaged by these necessary preliminary sufferings.

My major was settled for the summer at Fontainebleau, whither he invited me to come from time to time on a few days' visit. One morning he caused me to be awoke, that he might show me his foot, which occasioned extreme suffering. Swelling and great redness of the parts plainly told me, that I had to do with a paroxysm of acute gout of a very frank character. I then suspected that it was a case of regular gout, but I did not know to what extent its manifestations ought to be respected; moreover, I did not know then that gout and megrim are sisters. Notwithstanding the principles in which I was reared in my early medical education, I was subject, like many others, to the influence of the doctrines of Broussais, at that time in full vigor; and consequently I judged that it was necessary to subdue the inflammation by antiphlogistic treatment. Leeches were applied to the affected parts, which were forthwith enveloped in poultices sprinkled with laudanum. The inflammation subsided, to the great joy of the patient, and to the great satisfaction of the physician. Only too soon had I to regret my imprudent intervention. From that moment, my unlucky friend lost his former good health. He had a second attack, which was an attack of chronic gout—irregular, moderate, and atonic. Not only was the general health altered, but there was likewise a deplorable corresponding effect produced upon the spirits and mental powers. The major lost his mental acumen and habitual gayety of manner; he became heavy, cross, and tiresome. Ere long, he had a first attack of apoplexy; and two years later, he was carried off by a second attack.

Such then, gentlemen, is the nature of larvaceous gout, megrim, periodic megrim, preceded by general discomfort accompanied by vomiting; which latter symptom, with the headache, are characteristic, and generally last for some hours. Récamier always called the attention of his audience to it; and, before him, many others had pointed out the nature of this singular neurosis. So evidently is it in many cases a manifestation of the gouty diathesis, that articular gout and megrim are observed in the same person, the one subsiding on the appearance of the other; and that it is often, also, the only expression of the hereditary tendency in subjects who are the children of decidedly gouty parents.

We may connect with these periodic megrims certain transient cerebral symptoms occurring at longer or shorter intervals, symptoms which Musgrave, Wepfer, Van Swieten, and all who have studied the question, have correctly classified with the phenomena produced by irregular larvaceous gout.

There is sometimes *vertigo*, as in that man of whom Boerhaave's commentator relates, that during two years he was always seized with vertiginous symptoms when he attempted to stand up. In vain had the ablest practitioners endeavored to cure him. Quite suddenly, he had an attack of gout, of which disease up to that date he had had no indication: from that time, he found himself free from the distressing vertigo to which he had formerly been liable.

There is sometimes disturbance of the sensorium. A gouty man was complaining of his vision: his eyes, he said, seemed as if covered with a flake of snow. These sensations disappeared after an attack of gout in the foot.

Hippocrates, speaking of epilepsy, said: "*Magni morbi in vehementia existentis solutes coxarum dolor.*" Van Swieten mentions the case of a man, his patient, who had had violent abdominal pains, accompanied by delirium and general trembling: at a later period, this individual had a severe attack of epilepsy. From the date of that seizure, he had twice a year attacks of regular gout, and was no longer tormented by the nervous symptoms he had previously experienced.

In my lectures on *angina pectoris*, I took great pains to point out to you that that neurosis might be a manifestation of the gouty diathesis. As a case in point, I related the case of a patient whom I had recently seen in my consulting-room.* Similar cases could easily be collected from the writings of authors, where you will also find cases similar to those described under the name of diaphragmatic gout by W. Butter, an English physician.

Musgrave and Stoll have spoken of gouty cardialgia. Hoffmann has mentioned *spasmodic vomiting* of a similar nature. Certain delusions, called *vapors* confounded with symptoms of a hypochondriacal or hysterical character, are sometimes explained by the occurrence of attacks of regular gout. *Nervous asthma* is assuredly the most common of all these strange neuroses which are manifestations of larvaceous gout. This is a point on which I insisted in a lecture specially devoted to this disease.* I shall recur to it.

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* See p. 146 of this volume.

Here, to confine my illustrations to my own practice, I shall describe to you the following case. I was acquainted with the brother of an apothecary, an individual celebrated in the annals of the Ecole de Droit of Paris, in whom attacks of asthma periodically alternated with attacks of articular gout. The thoracic symptoms recurred during two or three months without the supervention of any affection of the joints: they then occurred, when the asthmatic attacks ceased.

The same patient also suffered from paroxysms of nephritic colic, and passed with the urine either notable quantities of fine sand, or gravel of pretty considerable volume: he then had neither gout nor asthma.

In fact, *gravel*, like asthma, megrim, and other neuroses, to which I would add *hæmorrhoids*, is a form of larvaceous gout.

Certain *cutaneous affections*, particularly some forms of eczema and chronic lichen, belong to the same category. The gouty nature of these diseases of the skin, long since recognized by our predecessors, is likewise admitted in the present day by the most intelligent practitioners, among whom it is sufficient to mention one of your teachers, Dr. Bazin of the Hôpital Saint-Louis. As for myself, I unhesitatingly accept as a fact this transformation of gout, of which I have seen numerous examples. Among others, I may mention the case of a friend, a distinguished literary critic, who subject for many years to attacks of regular gout, is free from them when he has the cutaneous eruption.

Gentlemen, these irregular manifestations of gout in the progeny of gouty parents, may constitute the sole expression of the gouty diathesis hereditarily transmitted to them: they may precede any regular manifestation: they may alternate with them or follow them; and in the latter case constitute a form of anomalous gout of which I have yet to speak.

In *anomalous gout*—also called *visceral gout*—the symptoms which in regular gout, acute or chronic, occupy a secondary place, exceedingly predominate over the articular manifestations, and even frequently constitute the sole phenomena of the disease. The functional disorders are usually associated with organic affections of a more or less serious nature, though sometimes they exist independent of any appreciable anatomical lesion. In general, visceral gout is a transformation of regular gout, acute or chronic, the articular manifestations of which have been treated by violent and too long-continued disturbing measures.

Among the symptoms of anomalous gout, the most important are *albuminous nephritis*, and to be more precise, *Bright's* disease. This fact, which did not escape the observation of Bright himself, has been confirmed by Garrod, and, before him, by Rayer.† How often have I, when called in along with this last-named eminent physician to patients affected with albuminuria, found gout concealed behind the renal affection! How often have I discovered that albuminuria, whether nephritic colic or gravel existed or not, had no other starting-point than gout!

Pulmonary catarrh, by which a great many aged gouty persons terminate their existence, is also a common form of visceral gout. This catarrh causes habitual congestion of the respiratory apparatus, which, on auscultation is shown by fine subcrepitant râles, by signs of chronic bronchitis, frequently complicated by pleural effusions, latent in their origin.

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* See p. 95 of this volume.

† RAYER: Traité des Maladies des Reins, et des Altérations de la Secrétion Urinaire. Paris, 1839.

Although gout does not affect the heart, like acute articular rheumatism, cardiac affections, and diseases of the great vessels are very usual in gouty persons. In rheumatism, it is the endocardium which is affected, but in gout, it is the tissue itself of the heart which is implicated: there are some cases also in which chronic effusion into the pericardium takes place. Aneurismal dilatations of the great vessels have been observed; and when speaking of typhus, I told you how these concretions, by becoming deposited on the internal tunic of arteries, might up to a certain point account for the production of these serious vascular lesions.

The liver, so often attacked in regular, is still more frequently in anomalous gout. Gouty *chronic hepatitis*, mentioned by Baglivi, Stoll, Scudamore, and others, is characterized by pains in the right hypochondrium: by an increase or diminution in the volume of the liver, rendered appreciable by palpation and percussion; by jaundice, or at least by a subicteric tint of the skin. At the autopsy, the substance of the organ is often found exceedingly hard, granular, like cirrhosis; and (according to Lieutaud) charged with calcareous concretions. These concretions are also sometimes found in the *lungs*, where they become moulded in the bronchial tubes, forming cretaceous arborizations, of which I now show you a specimen.

When speaking of typhus, I alluded to a patient who died in our wards, ten years ago, with symptoms which evidently belonged to visceral gout. The history of this patient's case is here in its proper place, particularly because we seldom have opportunities of seeing gouty patients in our hospitals.

The patient to whom I refer was an employé, forty-nine years of age, admitted to the Hôtel-Dieu on the 7th October, 1858. I found him in a state of great debility and exhaustion. Although his mental faculties seemed unimpaired, his great prostration prevented him from bearing long questioning. The general pale tint of the skin suggested the impression that he had had great loss of blood. On questioning him, it was found that he had had hemorrhage from the anus some days previously; but this flux, which he estimated at 250 or 300 grammes, was not enough to account for the anæmic, pale-yellow appearance of the skin.

He stated that he had always enjoyed perfect health till 1855, when he had his first attack of articular gout, which seized almost simultaneously the feet and knees. The metatarsal phalangeal articulations of both great toes were first seized. This attack, of which the paroxysms were characterized by pain, swelling, and redness, never attained more than an average intensity, lasted fifteen months, and was not attended by any notable impression on the general health. It was announced by changes in the disposition of the patient, which by his own avowal had become irritable, disturbed, and excitable. This condition of mental disquiet, accompanied by dizziness, vertigo, complete insomnia, or disturbed sleep with disagreeable dreams, continued for two years, and ceased when gout exploded in the joints.

Everything seemed to return to a normal state, when the first attack of gout was over; but after a certain time, the patient again experienced a feeling of cerebral fatigue, accompanied by vague pains in the trunk and limbs: no frank paroxysm of articular gout declared itself, but the joints became painful. For these symptoms, sulphur baths were prescribed. In consequence of this medication, he fell into the serious condition which obliged him to enter our wards.

I have pointed out to you the mental and physical debility, and the great paleness of skin which we found in this patient. Our attention was equally arrested by the difficulty which he seemed to have in breathing.

On examining the chest, great effusion was detected in the left pleural cavity: fluid filled that cavity as far up as the middle of the infraspinous fossa of the scapula. Throughout this extent, there was complete dulness on percussion: and there was heard, on auscultation, an ægophonic resonance of the voice. The degree of fever was very moderate.

On the evening of the following day, nervous symptoms supervened which next day increased in severity. At the visit next morning, I found the patient in a state of low delirium, which cleared away when he was spoken to. He answered questions like one overwhelmed with fatigue. I observed slight muscular contractions, particularly in the hands and muscles of the eye, the globes being alternately drawn in a rapid manner from one angle of the orbit to the other. In the evening of the same day, there was a diminution in the low delirium and convulsive movements: the drowsiness continued: the patient, however, still answered the questions addressed to him. He died during the night, becoming extinct, so to speak, without the supervention of any new symptoms, and retaining unimpaired to the last his mental faculties.

The autopsy was made on the morning of 11th October, about thirty-six hours after death. The decomposition of the body was far advanced, which explains some anatomical peculiarities of which I am going to speak, particularly, softening of the tissues, and a coloring of the inner surface of the vessels. In the cavity of the cranium, there was a scarcely appreciable quantity of serosity. The encephalon presented on its entire surface, but particularly at the base of the brain, a marbled opaline hue, dependent upon infiltration of the meninges, which were adherent to the parenchyma, the infiltration being greatest at the fissure of Sylvius. The cerebral substance was generally softer than usual, so that on placing the encephalon on its inferior surface, the two hemispheres separated from one another, tearing the corpus callosum. However, when a jet of water was directed upon the softened substance, no fragment was detached. There was no fluid in the ventricles. Softening was found in other organs. The heart, which during life presented neither functional disturbance nor change of volume, was soft. The only other peculiarity which we had occasion to note was a vinous red color of the surface of the aórta, the internal surface of which was, perhaps, a little less smooth than is normal. The left pleura contained a litre of sanguinolent serosity. The left lung was squeezed back by this fluid upon the vertebral column: its tissue was red like the lees of wine, flaccid, and resembled muscle in the first stage of putrefaction. The kidneys were blackish. In one of them, I found a small calculus impacted in one of the calices: this calculus was about the size of a hempseed: others of smaller size had found their way into the bladder. On opening the tarso-metatarsal and metatarso-phalangeal articulations of the great toes, as well as on opening the left knee, we found the articular surfaces covered with a whitish substance resembling scaly white zinc. There was a similar deposit upon the interarticular cartilages of the femoro-tibial joint, upon the ligaments of all the joints examined, and along the sheaths of the tendons of the left foot there was a tophus as large as a pea. The concretions were pure uric acid.

The following case, which may to a certain extent be compared to that now detailed, will give you an idea of the perturbation into which the system may be thrown by anomalous gout.

A man, forty years old, of vigorous constitution, though the child of gouty parents, had from the age of twenty-five been subject to attacks of frankly regular, acute gout. Being a friend of pleasure, and incapable of submitting to any restraint which prevented him from giving himself up to it, he had had recourse to the pills of Lartigue and the syrup of Boubée whenever the paroxysms set in. The remedies never failed to produce the effect which the patient expected from them. As soon as he felt that he was going to have a paroxysm, he employed his anti-gout medicines; and as his attacks came on in the evening, when he was going to bed, his feet were sufficiently free next morning from the gouty condition to allow him to put on soft stockings, and go into society. Careless of my advice, laughing at my gloomy predictions, he continued to take his mischievous drugs. The attacks of gout, at first considerably separated from one another, and limited to the great toes, soon began to recur at shorter intervals: the hands and knees were seized in their turn. The joints became surrounded with tophus, which was at first absorbed, leaving the joints quite free: afterwards, the tophaceous concretions became larger and more permanent, and over some of them the skin ulcerated: the ulcers cicatrized, and then formed again. The attacks lost their acuteness, and yielded less promptly to the medicines which had at first so marvellously triumphed over them. To the acute, succeeded a subacute state; and at the end of some years, a chronic, soft, atonic gout had taken the place of the frank gout. The time came when the patient was obliged to keep his room for several months, and even to rest in his arm-chair. The pains with which he was tormented were much less localized, but as he could not and would not endure them, he had recourse to opium, the doses of which he rapidly augmented. During the latter years of his miserable existence, this unfortunate man became quite powerless. His temper, naturally headstrong, became still more acrimonious, rendering him insupportable to those around him. Without any apparent cause of provocation, he gave way to veritable paroxysms of rage. At a later date, he fell into a condition resembling dementia. Becoming unable to do anything for himself, it was necessary to lift him out of bed, dress him, and place him on a seat, where he remained for the day. Bent down, and very different from what he used to be in respect of care of his person, he reminded one of the helpless patients [*gâteux*] to be seen in lunatic asylums. But in conjunction with this state of brutishness, there was no definite phenomenon indicative of mental alienation. Such was the view taken by a physician who was consulted, one peculiarly qualified to give an opinion in such a case. There was nothing the least like delirium; and when the patient was roused from his state of torpor, he always replied with precision to the questions addressed to him. He had no symptoms of paralysis. The functions of organic life were performed without perceptible difficulty. The circulation never seemed to be embarrassed: respiration was regular: he retained his appetite: and his digestion continued to be perfect. At last, he was unable to leave his bed: day by day, his torpor increased: and he died in a state of coma.

Gentlemen, in the remarks now made, I have had in view the cases in which the organic or functional affections incident to anomalous gout slowly and gradually instal themselves, if I may use such an expression. There are other cases, in which the morbid symptoms supervene abruptly, constituting what the ancients knew under the name of *gouty metastasis*.

These metastases, the existence of which some physicians in vain attempt to deny, generally take place under the influence of a disturbing cause, which has inopportunely silenced the regular manifestations of normal gout. They sometimes occur in one organ, and sometimes in another, and their gravity in relation to the importance of the organ attacked, and the intensity of the affection which has been thereby determined, may be such as to occasion death in a more or less rapid manner: "*Ita incredibile quot morbos creat materia podagrica, sæpe subitò lethales,*" says Boerhaave.

These morbid affections are *thoracic*, viz., pneumonia, or rather peripneumonic catarrh, and intensely acute pleurisy with effusion—*gastro-intestinal*, viz., gastralgic pains, vomiting, cœliac flux, sometimes proceeding so far as to simulate real choleraic diarrhœa—*icteric* affections—*cerebral* symptoms, viz., vertiginous or lipothymic phenomena proceeding sometimes to fainting and mortal syncope—and *apoplectiform* phenomena, an example of which I recently saw with my honorable colleague Dr. Chaillou, and another example of which was mentioned to me a few days ago by my friend Dr. Demarquay, who had just met with it.

Dr. Demarquay's case occurred in a man who, being attacked in the foot by very acute regular gout, with a view to soothe his intolerable suffering, applied cold water compresses to the affected part. The pain was almost immediately relieved; but a few hours afterwards, Dr. Demarquay was sent for in great haste. When he arrived, he found the patient in a state of apoplectic semi-stupor. He spoke with embarrassed voice, and sputtered out the few words which he attempted to pronounce. Fortunately, sinapisms applied to the feet restored the articular inflammation, which ought not to have been interfered with, when, almost immediately, the cerebral symptoms disappeared.

Visceral gout seems to be the result of a sort of imperfect inflammation, analogous to that which manifests itself in the joints. The importance of the organs in which it occurs renders it much more serious than articular gout. The intensity of the phenomena by which it is characterized is, moreover, in general, proportionate to the intensity of the articular manifestations, which, having preceded it, are prematurely extinguished; and also to the rapidity with which the articular manifestations have disappeared under the influence of one cause or another.

Parallel between Gout and Rheumatism.—Articular Rheumatism: Chronic Rheumatism: Nodular Rheumatism.—Nature of Gout.

GENTLEMEN: We now come to one of the most difficult questions embraced in the subject we are now considering: Are gout and rheumatism the same disease.

Some physicians answer this question in the affirmative. Gout and rheumatism, they say, are only different forms of the same disease. This opinion, advocated by the ablest practitioners, was that of Professor Chomel, my venerable predecessor in this clinical chair. According to Dr. Pidoux,* rheumatism and gout have one common root, and form two branches of the same trunk, being the two great manifestations of that state which the old physicians called "arthritism," a word, which in spite of the efforts made to get quit of it, has remained in the vocabulary of science from remote times; and it is a mistake, he says, to study them as if they belonged to different species. Acute articular rheumatism is nothing more, he believes, than an expression of the arthritic diathesis. Still more, according to the views of my very dear colleague of the Hôpital de la Charité, it may be given as the nosological type of the disease; "for," to quote his words: "it combines in a picture almost synoptical, and in the most lively striking features all the symptoms and local determinations, and presents in sharp outline all the various isolated affections which may occur in the long course of an attack of chronic rheumatism, whilst all the powers of the disease are being evolved and brought into play."

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* PIDOUX: "Qu'est-ce que le Rheumatisme?" Annales de la Société d'hydrologie Médicale de Paris. Vol. vii, Paris, 1860-1861.

I consider that chronic rheumatism and gout have strong points of resemblance, as well as great points of difference; but between articular gout and podagra, and that which we call acute articular rheumatism, there exist only remote analogies, if we do not take into account the local inflammation, where certainly the features of resemblance are more striking.

In a first attack of gout, it is the small articulations which are seized; and, speaking generally, in seven cases in ten, it is the great toe only which is attacked. In subsequent attacks, and under certain conditions, which I have pointed out to you, other joints, including the large joints, are affected.

In the first and subsequent attacks of acute articular rheumatism, several joints are almost always invaded, either all at once, or in succession; and the large articulations are those which are first seized.

In a frankly declared attack of gout, the general symptoms which announce the seizure, in most cases completely subside. The fever which accompanies the articular inflammation, though becoming intensified towards evening, at the time of the quotidian paroxysm, does not appear for more than two or three, or at the most seven or eight days, and is never so great as in rheumatism.

Rheumatism is announced, and is likewise throughout accompanied, by a violent inflammatory fever, which continues for twenty or thirty days or sometimes for a longer period: its severity is proportionate to the intensity of the local symptoms, which present this difference from the local symptoms of gout, that while gout occasions exquisite pain even when the patient remains at absolute rest, articular rheumatism, generally, gives rise to pain only when the patient moves.

The fever, I repeat, in an attack of gout, does not continue for more than a few days: it lasts very much longer in an attack of rheumatism. The patient is fortunate if it cease within three or four weeks, though he should not have one moment's respite during that period. In rheumatism, however severe and protracted the paroxysms may be, you will never see the formation of the tophaceous concretions which constitute a pathognomonic character of gout with frequently repeated attacks.

Once the attack is over, the gouty patient is immediately restored to health, excepting that there remains a little weakness in the limbs which have been affected: but the rheumatic sufferer is far from being so quickly re-established even when the attack has been moderate. Even when not debilitated by a too energetic antiphlogistic treatment, the patient's convalescence is slow, and characterized by an anæmic condition which is long in disappearing.

While a first attack of gout is almost invariably followed by other attacks recurring at intervals more or less brief, an attack of acute articular rheumatism does not involve the certainty of another.

One of the most striking differential characters of gout and acute rheumatism is the remarkable and almost inevitable coincidence of cardiac affections with rheumatism, and the occurrence of cardiac affection at a later period, if at all, in gout. Again, when the heart becomes affected in gout, the nature of the affection is different from that which is met with in rheumatism. In the latter, from the first attack, the serous tissues of the heart are implicated—the endocardium much more frequently than the pericardium; whereas it is the muscular tissue of the organ which is directly affected in gout.

The pulmonary complications of gout are seated in the lungs themselves: of rheumatism, in the pleura.

In rheumatism, the urine is loaded with uric acid: gravel belongs exclusively to gout. But I do not mean to say, that an individual who has had an attack of acute articular rheumatism will be thereby forever exempted from gravel and nephritic colic.

The points of dissimilarity between the two diseases are also apparent, when, leaving out of view their symptoms, their modes of evolution, their duration, and their complications, we examine their respective etiology.

Articular rheumatism scarcely ever attacks persons who have passed the age of maturity; and it is most frequently met with in youth, adolescence, and mature years. It is not unusual to meet with it in childhood, irrespective of scarlatina, of which disease, as I have told you, it is one of the epiphenomena. Gout, on the contrary, although cases of its occurrence in young subjects are on record, although I have myself met with the disease in a patient six years old, the little Moldavian boy of whom I spoke in my lectures on asthma—gout, I say, is a disease of maturity and old age, and rarely declares itself before the age of thirty or forty.

Although articular rheumatism equally attacks men and women—reservation being made to this extent, that women being less exposed to the immediately exciting causes of the malady are a little less liable—gout appears to belong peculiarly to the male sex. The disease which in certain women passes for gout is *nodular rheumatism*, which is much more common in females than in males: it has numerous analogies with, but is nevertheless in many respects different from, gout.

Heredicity plays a prominent part in the history of gout: it occupies a very disputable place in the history of acute rheumatism. In rheumatism, immediately exciting causes, such as cold, particularly damp cold, have a very great influence. In gout, the diathesis, the organic predisposition, is everything: exciting causes occupy a secondary place, and are generally of no account in the first manifestations of the disease. It is not till the individual has had several attacks, that external violence, a fit of indigestion, or mental emotion, may become the starting-point of a new manifestation.

The statement which I have now made respecting the influence of exciting causes in the two diseases, to the effect that it is positive in the one, and almost non-existent in the other, explains why acute articular rheumatism is much more frequently met with in the poorer classes, at least in such of them as are exposed in their occupations to sudden changes of temperature and severities of weather, than to those who are able to live under favorable hygienical conditions.

Gout is peculiarly a disease of the rich, its manifestations being never so frequent as in those who lead an inactive life, who are addicted to excesses of the table, venereal excesses, or intellectual toil, all of which promote the development of the diathesis: "*Divites plures interemit quam pauperes, plures sapientes quam fatuos*," said Sydenham, a dreadful sufferer from gout, who gave himself this philosophical consolation.

To sum up: there is this capital difference between gout and acute articular rheumatism, that gout is a chronic diathesic disease, and rheumatism an accidental disease, a sort of fever proceeding *sua sponte*, which when once recovered from, leaves behind it, not the disease itself, but only consequences of the disease, organic affections of the heart resulting from the inflammation which has attacked the serous membranes of that organ. Should there be a return of the disease, the return is accidental, and not the result, as in gout, of a dominant diathesis. To the cases which have been adduced of the transformation of gout, I can oppose my long experience, in the course of which I have never seen any such transformation. I know that a gouty individual may like any other person have articular rheumatism, and that it is quite distinct in these cases from the symptoms experienced in the attacks of gout which previously occurred.

I have spoken of acute articular rheumatism, the diagnosis of which from articular gout seems to me as simple as possible. I have not been speaking of *chronic rheumatism*, an essentially diathesic disease, which is hereditarily transmitted, and may also, like gout, be acquired, and can manifest itself in a variety of ways.

Chronic rheumatism, when the type is precise and definite, presents sharply drawn distinctive characters which distinguish it from gout; but it must be admitted that there are many cases in which it exhibits so great a resemblance to chronic gout that it is almost impossible to establish an absolute distinction between the two diseases. I am not, at present, referring to cases in which the two diatheses coexist in the same individual; for these, though the patient may know how to tell you the symptoms which belong to his rheumatism and gout respectively, the physician will be unable to distinguish the phenomena which pertain to each.

The analogies to which I refer are all the greater when not only the two diatheses coexist, but when they are also both hereditary in the same individual: a gouty person may, therefore, procreate rheumatic progeny, and a rheumatic parent have gouty children. There evidently exists between the two diseases a strong bond of relationship: "*rheumatismus agnatus podagræ*," said the old physicians; but this relationship does not imply identity. They are probably sisters of the same mother: probably, according to the comparison of Dr. Pidoux, "they are sprung from the same root, and from two branches of the same trunk;" and it is not less probable, as has been said by my honorable colleague, that notwithstanding the features which they possess in common, and their frequent interlacement, they both have natures peculiarly their own. Each of them, therefore, deserves to be studied separately, though both must be as closely approximated in the nosological programme as in clinical practice.

I hope, gentlemen, to be able on a future occasion to give some such account of chronic rheumatism as I am now presenting of gout, imperfect though it be. Upon this occasion, I shall limit myself to sketching very rapidly the chief features which seem to establish the line of demarcation between the two diseases.

Mobility is the primordial character of rheumatism; this character at once presents itself. In gout, on the other hand, mobility does not show itself till the malady has become inveterate, till the attacks, which at first were strictly local, have frequently recurred, or the progress of the regular manifestations has been interfered with. Gout, it is true, is sometimes erratic in its nature, but primitive erratic gout is rare.

It is only in atonic chronic gout that you will see the patients very "barometrical" [*tres-barométriques*] to use a commonly employed expression, meaning very sensitive to atmospheric changes. In rheumatism, this singular sensibility is constant from the first; and is seen even before any other manifestation characteristic of the diathesis. So great is this sensitiveness in some rheumatic subjects, that they become aware that an atmospheric change is about to take place, one, two, or three days before the change occurs, the announcement being made to them by their experiencing pains and feelings of discomfort which no other exciting cause can account for. Some patients will tell you that they feel the coming rain or snow, when there appears every prospect of a continuance of fine weather.

Rheumatic pains take possession of the muscular masses; and no part of the surface of the body is exempt from their attacks. Sometimes, they are localized in particular situations, to which they habitually return; but generally, they move from one place to another place, which in turn they leave, to instal themselves elsewhere. They are as variable in their nature as in their seat: they are acute or dull, boring or aching, superficial or deepseated. Sometimes, the patient experiences a sensation of heat or even of burning, of pinching, or twinging. At other times, there is an indefinite, indescribable feeling of discomfort; or, on the contrary, there may exist neuralgia perfectly localized in the course of a particular nerve or system of nerves, hemicrania, facial, intercostal, brachial, or sciatic neuralgia, the latter being the most common of the neuralgic affections. Neuralgia may affect the different organs, constituting gastralgia and enteralgia, which are dreadfully painful, and sometimes accompanied by secretions from the stomach and intestines. The former occasions vomiting, which is sometimes very copious, and the second gives rise to diarrhœa. We also meet with hepatalgia and lumbar neuralgia, simulating hepatic and nephritic colic.

The manifestations of frank gout are of limited duration; and in chronic gout, up to a certain point, they are also of short continuance: the opposite statement holds good in respect of rheumatism. In acute gout, when the attack is over, the patient resumes his ordinary good health; and in chronic gout, there are intervals of respite between the paroxysms, however long the latter may be, except, be it observed, in those cases in which the disease leaves behind it the infirmities which I have pointed out; but chronic rheumatism never quite gives up its hold of those whom it has once made its prey, in this sense, that the exciting causes will awake the diathesis—that these exciting causes, which in frank gout (whether acute or chronic) have relatively little influence, present themselves very often in rheumatism.

Gentlemen, a few minutes ago, I spoke of nodular rheumatism [*rhumatisme noueux*] also known as *primary asthenic gout* and *gouty rheumatism*, names which, in my opinion, are improper, for the affection is rheumatic and not gouty. The terms *chronic-rheumatic arthritis*, and *chronic primary articular rheumatism* [*arthrite rhumatismale chronique*, *rhumatisme articulaire chronique primitif*] would be more appropriate, were it not that *nodular rheumatism* [*rhumatisme noueux*] has greater advantages by indicating at the same time the nature of the disease, and the special form of the articular lesion which characterizes it.

In my next lecture, I will make some observations on nodular rheumatism, fully entering upon the differences between it and gout.

At present, let us consider the *nature of gout.*

For the humoral and solidist theories of the old physicians, chemistry was obliged to substitute one of its own. It having been shown by chemical analysis, that in gouty persons the blood, and other parts of the body, contain uric acid, either in excess or diverted from its natural channels of elimination, it was hastily concluded, that the disease consisted in a defect of equilibrium between the acids and alkalies of the blood and the different fluids of the economy. From that time some, including Cajetan-Taconi and Marie de Saint-Ursin,* believed in an alkaline gout, while a greater number, with Forbes and Parkinson,† believed in an acid gout. According to the latter class of observers, uric acid constituted the morbific matter, the peculiar principle of gout, and they looked upon an attack of gout as simply the consequences of efforts of nature to eliminate this excess of acid.

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* SAINT-URSIN: Etiologie et Thérapeutique de l'Arthrite et du Calcul; ou Opinion Nouvelle sur la Cause, la Nature, et le Traitement de la Goutte et de la Pierre. Paris, 1816.

† PARKINSON: Observations on the Nature and Cure of the Gout, on the Nodes of the Joint; and on Diet in Gout, Rheumatism, and Gravel.

Modern science has developed this theory. The oxidation of the materials destined for the nutrition of the body, science said, is the fundamental act of life. It is accomplished by the absorption of oxygen, which, penetrating through the respiratory passages, circulates in the blood. The combustion of nitrogenous substances, the result of this absorption of oxygen, metamorphoses the nutritive materials so as to render them in part assimilable and in part not assimilable, the latter destined to be eliminated by the different emunctories. To enable nutrition to proceed regularly, combustion must be as complete as possible. The alimentary substances most difficult to oxidate or burn (which is the same), are the nitrogenous albuminoids, a fact explained by their small affinity for oxygen. Urea is the product resulting from the last stage of the oxidation of nitrogenous matters: it is soluble, and may, therefore, be thrown off in the urine and pulmonary exhalation. When the oxidation is imperfectly performed, there is a production of uric acid and urates. If the uric acid and urates are formed in such excess as to prevent their entire elimination by the normal ways, they accumulate in different parts of the organism, and by their presence occasion disturbance of the different organs to which they are carried.

This then is what constitutes the uric diathesis: and as this diathesis exists in gouty subjects, the uric diathesis and the gouty diathesis are the same thing to the iatrochemists.

It must be admitted, that the starting-point of their theory rests upon a fact which is incontestable, and which I shall explain to you by and by:—the fact to which I refer is the presence in excess in the blood and other parts of the organism of gouty persons of uric acid and the urates. But before the conclusion at which the chemists have arrived can be admitted, viz., that this excess of uric acid and urates is really the *materies morbi*, we must be sure that the uric acid diathesis is found exclusively in gouty persons.

Dr. Garrod has shown that uric acid may exist in the blood, in quantities varying with the time which has elapsed since the last meal, in persons in the enjoyment of perfect health, and who are not gouty. Again, in respect of the pathological state, uric acid and the urates are met with in other diseases besides gout. I do not cite articular rheumatism in support of this statement, because some physicians look on it as a form of gout, but I refer to intermittent fevers, in which the existence of the uric diathesis has been established, when care has been taken to analyze the blood in the first stage of the fever. It is also found in a high degree in persons who have been subject for a long time to low diet.

The results then of chemical analysis cannot be accepted as proving the identity of the gouty and the uric diathesis, because the latter is a diathesis common to different diseases which have nothing else in common. We have what may be called the diathesis of *fibrination* and *défibrination*. All the diseases termed inflammatory are characterized chemically by an excess in the quantity of fibrin found in the blood; while in other diseases, in the eruptive fevers, for example, there is a diminution in the proportion of fibrin. Though they present the one characteristic in common, they are not the less, on that account, essentially different diseases. It is neither the excess nor the deficiency in the proportion of fibrin, but the specific cause which dominates the alteration in the blood, as it dominates the other morbid phenomena, which makes them what they are.

So it is in gout; the production in excess of uric acid and urates is a pathological phenomenon inherent, like all others, in the disease; and like all the others, it is dominated by a specific cause, which we know only by its effects, and which we term the *gouty diathesis.*

The idea of this diathesis, of this organic predisposition, is so very necessary, that without it we should be unable to take one step in advance in the study of gout.

Let us grant for a moment that the presence of uric acid is the essential cause of the disease, how can we explain why it should happen that out of a hundred individuals placed in the same hygienical conditions, living in precisely the same manner, eating precisely similar food, one person only should have gout? How is it that the mode of life, the alimentation favorable to the excessive production of uric acid and the urates, and their accumulation in certain parts of the organism, should only produce the uric acid diathesis in ninety-nine cases in a hundred? How are we to explain the fact that in a number of individuals leading a life of indolence, addicted to the pleasures of the table, sinning against all the laws of hygiene, not one should be gouty, while we see others become martyrs to gout, although they have always led a most active and abstemious life? How are we to explain these differences unless it be, I repeat, by admitting the existence of an idiosyncrasy, an individual organic predisposition of an altogether peculiar character? It is this predisposition which we call the gouty diathesis.

The theory promulgated by my learned colleague, Professor Charles Robin, is also a chemical theory. He says:

"On considering the nature of the fibrous tissues of the economy, we see that in the act of nutrition they assimilate the albuminous substances, transforming them into *gelatin,* a constituent part of tissues; in the act of disassimilation, this gelatin is divided into various crystallizable principles, among which predominate uric acid and the urates. If from any cause the disassimilation should be in an excessive degree, the result is the more abundant production of the acid and its salts, which saturate the blood, and induce a pathological condition corresponding to that which Dr. Garrod describes under the name of the uric diathesis. The formation of tophus is explained by disassimilation taking place too rapidly, and by a consequent exosmotic transudation of the urates, whence results the deposit of these chalky masses in the cutaneous tissue, principally in the articulations, where the fibrous tissue predominates. These deposits take place in the different tissues, exactly as we see plastic deposits formed in certain diseases."

If the views of M. Robin be correct, how is it that we never meet with tophaceous deposits in the essentially fibrous tissues, such as the periosteum and dura mater? The preference, therefore, which tophus shows for the joints is a remarkable fact, which we cannot explain, and which presents a great subject of reflection to physicians.

These chemical theories, expressed in other terms, are elsewhere met with; for example, in the theory of Sydenham, when he speaks of the existence of a *morbific matter* resulting from the *coctions* being imperfectly performed in the *primæ viæ* and in secondary assimilating organs. Sydenham certainly did not give a name to the morbific element; with him there was no question of uric acid or urates, because he was not acquainted with them; but he made his *morbi seminium* play the part which modern chemistry attributes to the products which it has discovered. Take it all in all, the theory of the great English physician is much more medical than the theories of modern chemists.

TREATMENT OF GOUT.

GENTLEMEN: Ought we to treat gout? I mean, ought we to intervene actively during the paroxysms? Such a question would assuredly appear very singular, and possibly very impertinent, to the gouty patient; it would seem equally strange to a large number of physicians who cannot understand why there should be hesitation in interfering for the relief of the dreadful sufferings of which I have been sketching the picture. However great may be my desire to relieve my patients, I still ask myself the question: Ought we *to treat gout?*

Is it right to employ treatment to quell the cruelly painful articular manifestations? Ought we to treat chronic gout? Ought we to treat larvaceous gout, anomalous gout, retrocedent gout?

Sydenham, whose authority in such a matter is immense, inasmuch as independent of the experience he acquired as an eminent practitioner, he had had great opportunities of studying in his own person the disease by which he was tormented, and of judging of the good and bad results of the different medications which he employed, answers the question negatively, in respect at least of normal gout.

The conclusions at which he arrived as to the nature of the disease, concurred with the results of his own observation to show him the necessity of abstaining from interference. To him, the gouty subject was a sort of charged machine which had to be discharged externally, by some safety-valve, so as to prevent an internal explosion. The same sort of thing happens in gout as in the eruptive fevers, the cutaneous manifestations of which ought to be religiously respected. Should the morbific matter not find an issue by the external outlets, the excess of the excrementitious matters is driven-back upon the internal viscera, causing morbid symptoms of a much more serious character than those which occur in the natural and salutary course of the disease. Upon principle, Sydenham rejects the use of all topical means, which are often injurious and never useful. The medications which he recommends act upon the whole system, promote "coction," aid the elimination of morbific matter, and contribute to the defence of the organism in the contest which it has to sustain.

As to pretended specific remedies, the number of which was considerable in the time of the poet Lucian, who enumerated them in his pleasant poem the *Tragopodagra*, and had greatly augmented when Sydenham wrote, which goes on increasing in our day, when they have increased with incredible profusion in the fourth page of the political papers, and sadder still, even in our medical journals—against these mischievous drugs, Sydenham thus energetically raised his voice: "*Sane dolendum est, medicinam (artem nobilissimam) hujus modi nugis quæ sive ab inscitia, sive a pravitate scriptorum, credulis objiciuntur, usque adeo deturpari.*"

Without adopting the theoretical views of Sydenham, without even being able to form a satisfactory opinion as to the nature of gout, my personal experience leads me to pursue a course as reserved as that recommended by Sydenham. During the last thirty years, I have treated a large number of gouty patients. At the commencement of my practice, like many others, I attempted to fight with the disease: now, I cross my arms, and look on: I do nothing—absolutely nothing—to subdue attacks of acute gout, particularly when they occur in individuals in the prime of life. More than once I have had occasion to regret departing from this do-nothing system, and been led to realize how perilous the adoption of active treatment might become. When, strong in my conviction, I have left the malady to itself; when the patient has resigned himself to suffer, I have always seen him emerge from the crisis in the best conditions; and thus, at the cost of some suffering, I have purchased for my patients months of perfect health. When, on the contrary, I have interfered with the paroxysm, which unfortunately, is easily done, I may have escaped the dangers of shifted gout, but I ran the great risk of seeing the attacks recur at shorter intervals, and of transforming a frank and transitory gout into a cold, atonic, persistent gout. I have felt the entire truthfulness of Sydenham's aphorism: "*Hoc in morbo dolor amarissimum est naturæ pharmacum, qui quo vehementior est eo citius præterlabitur paroxysmus, atque insuper et longior erit intermissio et magis perfecta; et vice versa.*" I am now careful to use no means to smother the pain, which I look upon as very favorable to gouty patients: and in atonic gout, my desire is to see a return of the acute attacks, a return, however, which it is very difficult to induce by artificial means.

But in that form of acute gout, which I have called the *chain* form, in which, after four, five, or six days of suffering, new pains arise, the attack going on in this way for two, three, or four months, it is very difficult to refuse to give some relief to patients who implore it. That is especially difficult when the physician has not sufficient authority to convince the patient of the usefulness of this bitter remedy. Under such circumstances, one is obliged to yield, lest the patients in despair should fly to those gout-curing drugs which, when taken immoderately and without serious consideration, risk future evils by cutting short the paroxysm. In the case of such patients I interfere as a matter of duty, avert greater evils, and give protection from ignorant unscrupulous empirics. I act with a view to moderate the pain, to render it bearable, but not to extinguish it. I administer with the utmost possible prudence and method the medicines which I judge most useful, and involving least risk of proving injurious.

I have no doubt that the fears which I have now expressed of the dangers of the remedies applied to the treatment of attacks of gout will to some persons appear exaggerated. I know that there are a certain number of gouty persons who have arrested their paroxysms with impunity; and the knowledge of this induces others to repeat the experiment. But I ask, do these exceptional cases weaken the general rule?

Would we be justified in suppressing the menstrual flux because some one young wife or maid had had her courses abruptly stopped without much injury to her health? Supposing we had seen, in a young man, an habitual hæmorrhoidal flux suddenly interrupted, with no other detriment to the general health than slight giddiness and headache, should we be justified in concluding from that fact that the suppression of a flux which was right in a man in the full vigor of life would not prove prejudicial if practiced in the case of an old man? Certainly not: no physician would deliberately advise such experiments to be tried. Every physician knows that the supplementary functions which are constituted by habitual fluxes, serve useful purposes, and can only be interfered with when careful management is adopted.

Equally respect the external manifestations of gout; respect the articular pains, respect them especially in persons of advanced age. It is less dangerous to interfere with them in young subjects, provided great prudence be used, the means being proportionate to the strength of the patient, and the facility of being relieved by natural or temporary emunctories. There are persons in whom some maladies terminate favorably of themselves, by sweating, by diarrhœa, or by a urinary flow. I therefore advise you to pursue your medications in the same direction as indicated by nature.

When gout is visceral, non-intervention is not allowable; for there can be nothing worse than such an affection. In visceral gout, we must try to bring back the gouty manifestations to the joints, which as I have already said it is not easy to do: in such cases, it is justifiable to give the medicines supposed to possess anti-gout properties.

Colchicum is the most efficacious of all the anti-gout remedies which have been lauded. On this point, there can be no possible doubt: its virtues have been long ago recognized. It was called *theriaca articulorum* by Avicenna;[*] and Ætius said: "*Hermodactylon confestim minuit dolores.*" His "hermodactylon," as M. J. E. Planchon[†] shows, was *colchicum variegatum*, which does not differ in its properties from *colchicum autumnale*, the basis of most of the pretended anti-gout nostrums, so deplorably employed. It is the basis of the *eau médicinale de Husson*, the specific of Reynold, the specific of Want, &c., as well as of the too celebrated pills of Lartigue, which have been productive of so much mischief. It is probably to veratria, its active principle, that colchicum owes its sedative and contra-stimulant properties. Veratria, as well as such plants as cevadilla, and white hellebore (which contain veratria in larger proportion) takes its place in other nostrums, such as the remedy of Laville.

The most usually prescribed preparations of colchicum are the extract and the tincture of the seeds: the former is given in doses of from five to eight drops, and the latter in doses of from twenty to fifty centigrammes. The use of the medicine is continued during two, three, or four days. The same rule is observed in respect of the wine of colchicum, which is administered in doses of from five to twenty-five grammes. The tincture of the seeds is the preparation which enters into the composition of most of the anti-gout liquors which charlatanism has decorated with a variety of names.

When I prescribe colchicum I adopt Dr. Becquerel's formula for pills, which is as follows:

Sulphate of quinine, ...	150 centigrammes.
Extract of digitalis, ...	25 "
Extract of colchicum seeds, . . .	50 "

To be made *s. a.* into a pill-mass; and divided into ten pills.

Of these pills, the patient is ordered to take from two to three in the twenty-four hours, for from three to five consecutive days.

These pills differ very little from pills prepared according to an older formula of Dr. Debout, who employed them with good results in cases of gouty megrim. The following is his formula:

Extract of colchicum, ...	8 grammes.
Sulphate of quinine, ...	3 "
Powder of digitalis, ...	150 centigrammes.

To be made into a pill-mass, and divided into thirty pills.

One of these pills is taken every evening. Under the influence of these medicines, I have seen the pain of the gouty paroxysm cease within seven or eight hours.[‡]

Colchicum and veratria are not the only remedies which have the power of quieting the disease. This property is shared with them by all the great perturbatory remedies, such as bleeding, and purging, particularly purging with drastics, one of which, colocynth, enters into the composition of certain so-called specifics. However, while these perturbators give, to a certain extent, an account of their action, colchicum and its succedanea usually induce neither notable alvine flux, profuse sweating, nor augmented flow of urine, the perturbation caused by them seeming to exert a powerful impression on the nervous system.

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[*] Lib. ii, cap. ccclii, p. 247.

[†] PLANCHON: De hermaductes au point de vue botanique et pharmaceutique. Paris, 1855.

[‡] DEBOUT: Bulletin de Thérapeutique, for February, 1857.

The energy with which these medicines act makes it necessary for physicians to exercise the greatest prudence in their administration: to avoid producing retrocession of the gout upon the viscera, or transforming acute into chronic gout, it is requisite to begin with small doses, so as to moderate without entirely or abruptly removing the pain. I never use them at the beginning of the attack, but wait till it has lasted some days, and is drawing to an end. By acting in this way, we abate the present symptoms, without subjecting the patient to much danger; while we may likewise hope to modify the future symptoms.

It is often better, I repeat, to do nothing, and have present to the mind the fact, that the attacks of gout are separated at greater intervals from one another when the preceding attack has lasted for a certain time. Lucian makes the hero of his *Tragopodagra* say:

> Irritantibus me
> Soleo occurrere multo iracundior,
> His vero qui cogitant nihil adversum mihi
> Benignam adhibeo mentem, facilisque fio.

Gentlemen, I have said nothing to you of *topical remedies*, which all physicians agree in denouncing. There is, however, one local medication which has rendered me real service, of which I shall now speak. It is true that this medication is not indicated during the paroxysms (although it may be useful, and is free from danger at the termination); but it is applicable in the intervals of the paroxysms, the return of which it may prevent. I refer to *tobacco fumigations*.

Every eight days, from the moment the attack is over, the patient exposes the joints which have been affected, to the smoke of tobacco leaves burned in a chafing-pan. The heat ought to be strong. The smoke is received in a large stocking, or in woollen blankets, enveloping the affected parts.

The efficacy of a proceeding of this description may, it appears to me, be thus explained. Experience has shown that causes capable of exciting pains in a part of the body act nowhere more vigorously than upon those which have been the seat of gout. For example, the pressure of a tight stocking will often be sufficient to excite the return of a recent attack; and, in passing, let me add, that this fact may sometimes be advantageously employed to recall the gouty pains to the foot, when, having abruptly ceased, the patient experiences morbid symptoms in the viscera, which symptoms, it is important to remove. We can thus understand why an obtunding medication, such as tobacco smoke, may prevent the return of the paroxysms by diminishing the susceptibility of the parts.

Granting, gentlemen (that which I deny), that there is never danger in combating the manifestations of frank articular gout, to suppress the local manifestations is no more curing the gout than making syphilitic eruptions disappear by topical means is curing the pox. We no doubt accomplish a great deal when we render the external manifestations fewer and less acute; but as Cullen remarked, "gout, which is a disease of the entire economy, and very often depends on original conformation, cannot be cured by medicines, which only produce very transient effects." The diathesis continues to such an extent that, without being exposed to influences different from those which act on other men, the gouty patient will again be subjected to gouty paroxysms. To cure gout, is to destroy the gouty diathesis. This, it is alleged, can be done by the aid of certain medicines.

During the last century, physicians, struck with the affinity between gravel and gout, proposed the plan of giving gouty patients remedies which have been called lithontriptic washes [*lessives lithontriptiques*], alkaline solutions, such as Glauber salts, or solutions made with the carbonates of lime, of soda, &c., which appear to be so beneficial to individuals attacked by gravel and nephritic colic.

This alkaline method of treatment, soon abandoned even by those who had most extolled it, has, in our day, been restored to its place of honor by the defenders of the chemical theory of the uric acid diathesis.

I should not be telling you anything which you do not already know, were I to rehearse all that has been said on the employment of alkaline mineral waters in the treatment of gout. You are aware that while the waters of Carlsbad, of Vichy, of Vals, and other places have their ardent defenders, they have been rigorously proscribed by physicians of the highest eminence who had learned from experience that they were dangerous.

Gentlemen, I know no medication more perilous than that by mineral waters, when administered without reserve, without discernment, and without reference to the individual's conditions of health, the form of the gout, the time which has elapsed since the last attack, and the probability of a fresh attack not being imminent. Not a year passes, in which I do not see the evil consequences of the use of mineral waters in gout.

Do I mean by this statement to interdict absolutely their employment? Certainly not. With Dr. Durand-Fardel* I believe in their beneficial action; but in a degree which is very limited.

As a general rule, alkaline waters ought never to be taken for more than ten or twelve days consecutively; and only in small quantity at a time. It is well not to revert to their use more frequently than once a month for the limited period I have named. By continuing their use for a long period, or almost constantly, or taking them in enormous doses (as some do not hesitate to order them to be taken), is to run the risk of having a frank gout converted into a gout which is chronic, vague, and visceral. In relation to this point, allow me to quote the opinion of Prunelle, a man very competent to form correct views on such a subject. He says:

"Before venturing to prescribe Vichy or other mineral waters in the interval between the paroxysms of gout, it will be very important to be well informed as to everything which has taken place since the last paroxysm, and to ascertain to what extent the discharge of sweat and urine during the paroxysms had been looked on as critical. For, very evidently, if the gouty paroxysm has been imperfect, the elaboration perhaps preparing for one more complete, ought not to have been interrupted. An interruption of this kind is sufficient to change the mode of action of the medicament, and to begin a metasyncrasis. The results of the latter, as frequently happens, are the suppression of the articular gout; but all medical observers are agreed in considering this suppression as unfortunate, and as sometimes the source of very great danger. Hence it is, that as the waters of Vichy possesses violent perturbating powers, it is necessary to avoid employing them in the intervals between the paroxysms of articular gout."

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* DURAND-FARDEL: Dictionnaire Général des Eaux Minérales, et d'Hydrologie Médicale: 1860.

In articular gout, whatever might be its acute or chronic form, the celebrated physician of Vichy prohibited the use of baths, not only baths of the mineral water, but likewise baths of ordinary soft water. He restricted himself to prescribing the mineral water to be drunk in very moderate quantities, with a view to combat the diathesic manifestations which may arise in the digestive canal or urinary passages.

The practice of Prunelle is to-day the same as has long been the practice of Dr. Durand-Fardel, who prescribes the water to be taken in very moderate doses, so as not to fatigue the digestive organs and thereby attract towards them gouty manifestations. But, though Dr. Durand-Fardel recommends that in acute gout the use of baths should be abstained from, lest they act too energetically upon the general system, checking the articular manifestations, and determining metastases, which are the more dangerous that the action of the medicament cannot be directed, yet in chronic gout, he recommends them to be employed, provided the patients are carefully watched.

In thus advising as to the use of the waters, the administration of which he so ably directs, Dr. Durand-Fardel contests the explanation which chemists have given of their salutary action. Neither the alkaline waters of Vichy, nor of Vals, Carlsbad, nor Pougues—which, in certain cases, within narrow limits, it is true, find their indications—act beneficially by neutralizing the uric acid, which the chemists allege is the cause of all the morbid symptoms of gout. Admitting that changes take place within the living body as they do in the experimental vessel of the chemist, the blood and the secretions, in place of containing uric acid would contain urates, and we should hardly have obtained any change, because as Dr. Garrod has shown, it is in the form of urate of soda that the uric acid exists in gouty persons.

Alkaline mineral waters, then, do not act upon the uric acid, which is the consequence and not the cause of the disease, but on the diathesis itself: or at least, they act by combating the different pathological states which the diathesis induces, that is to say, disorder in the digestive function, in the urinary and cutaneous secretions. They act, in fact, by regulating the great functions which constitute nutrition.

It is in a similar manner that the waters of Plombières and Contrexéville, the mineral ingredients of which exist in very small proportions, have also a useful action upon some gouty persons, by improving assimilation.

In a similar manner act likewise the preparations of quinine, which constitute the basis of the remedies of Held and Giannini, the preparations of nux vomica and quassia amara, the tincture of guaiacum of the Codex,* and its analogue the ratifia of Caraibes: they are expressly indicated when it is necessary to restore lost tone to the organism, and particularly to the organs of digestion. It is, therefore, particularly in the treatment of chronic gout that these medicines will find their place.

In the treatment of this form of gout, certain mineral waters are appropriate. When the patients present all the symptoms of anæmia, when asthenia seems to predominate, the ferruginous waters such as Spa and Pyrmont are useful; and better still, when the gout is more visceral than articular, the chlorinated sodaic waters of Wiesbaden, Kissingen, Kreuznach, and Hombourg.

Wilbad, Néris, and Luxeuil are the most salutary to neuropathic individuals; but, when the object is to combat not only the gout itself but the lesions which it brings in its train, such as articular engorgements, the chlorinated sodaic waters, and certain sulphurous waters, are more useful. Aix in Savoy, the soft springs of Bagnères-de-Luchon, of Cauterets, and of Schinznach (in Switzerland) ought to be preferred. On more than one occasion I have satisfied myself as to the beneficial action of these waters, particularly the waters of Aix in Savoy. Let me add, however, that these waters have no efficacy, and their administration is not exempt from danger, unless the gout has been quiet for a long time. In atonic gout, and still more in regular gout, their unseasonable use might induce visceral morbid symptoms; and gout thus provoked might have the most deplorable issue.

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* Codex Medicamentarius, p. 376. Paris, 1866.

The chlorinated sodaic waters, the sulphated waters, the weak sulphurous waters, such waters as those of Néris, Wilbad, and Luxeuil, are also indicated in cases in which gout is complicated with rheumatism.

I may say as much for hydrotherapy. When methodically employed, it beneficially modifies the consecutive accidents of gout. By rousing the cutaneous and urinary functions, by opening all the emunctories, and stimulating the entire system, hydrotherapy augments the peptic powers.

Gentlemen, all the different means of treatment fail in any way whatever to constitute specific remedies: in this respect we are no more advanced than was Sydenham when he said: "*Therapeia* RADICALIS *et usque quaque perfecta, qua quis etiam a diathesi ad hunc morbum foret liberatus adhuc in Democriti puteo latet, atque in naturæ sinu reconditur; nescio quando, a quibus in lucem extrahenda.*" But although we cannot hope to destroy radically the diathesis, we can at least endeavor to weaken its injurious tendencies; and according to the opinion of all great practitioners, it is in the regimen that we find means of arriving at this result.

The rules laid down by Sydenham are still those which we have to follow in the present day: I cannot, therefore, do better than give you an abstract of the long paragraphs which he has devoted to this object.

It is essential to be very sober, and not to eat food difficult of digestion: but it is also necessary to avoid a too great abstinence, which leads to debility.

As to the nature of the food, we must consult the gout or rather the aptitudes of the gouty patient; and we must strictly regulate the hours of his meals. Wine may prove injurious as an ordinary beverage, but the exclusive use of water will be still more hurtful. Between the abuse of one or other, a middle course was recommended by Sydenham. He recommended the use of a mild ale brewed at London; and he allowed the wines of Spain in preference to those of France and the Rhine.

Every kind of excess being injurious to gouty persons, they ought to avoid immoderate venereal pleasures, and prolonged want of sleep; but they must not remain too long in bed, and as a rule, they ought to go to bed early and rise early.

Though fatigue is injurious, moderate exercise is one of the best means of assisting the nutritive functions. The exercise ought to be regular: and that which is taken in the country is much to be preferred to that taken in towns, where the air is close and filled with noxious exhalations. The gouty individual ought every day to take a walk, or, better still, according to Sydenham, to ride on horseback. If he can do neither, he ought to take carriage exercise; which for the aged is indispensable.

The religious observance of these rules has a favorable influence on the constitution of gouty subjects, diminishes the frequency of the attacks, and enables the system better to resist their effects; but is far from promising a cure.

Sydenham says: "Quamvis hujusmodi regulæ tam diætam quam cæterum regimen spectantes, si ab homine podagræ obnoxio religiose observentur, eum ab enormioribus morbi insultibus præservare queant, atque istam sanguine et partibus solidis firmitatem conciliare, quæ ab illa malorum iliade, unde morbus non solum supra humanæ potentiæ vires, sed et funestus tandem redditur, eundem immunem praestare possit: non tamen efficient ut non post quædam intervalla maxime exeunte hyeme, podagra quandoque sentiatur."

LECTURE LXXXIII.

NODULAR RHEUMATISM, ERRONEOUSLY CALLED RHEUMATIC GOUT.

The Disease is very rare in Men: it is more common in Women.—Generally Chronic, supervening all at once.—Sometimes Subacute at the commencement.—It is a manifestation of the Rheumatic Diathesis.—Pains and Muscular Retractions.—The Heart is seldom affected.—Rheumatic Complications, however, have been observed in the Heart, Pleuræ, Lungs, Brain, and Kidneys.—Essentially a Chronic Disease in respect of its duration.—Successful Treatment by different medicines.—Tincture of Iodine, given internally, ought to be preferred.

GENTLEMEN: Nodular rheumatism being a disease which you will seldom see in the clinical wards, I ought not to-day to neglect speaking to you of it, as in the wards of St. Bernard and St. Agnes you have an opportunity of studying its principal characters in two of our patients. Let me also add, that you will seldom see in an hospital the beginning of the disease, and that in the asylums for incurables in the Salpêtrière and at Bicêtre, you can only study it in an advanced stage, when it has existed for years, when the patient has been domiciled as an incurable.

In bed 3 of St. Bernard Ward lies a woman forty years of age, who complains of articular pains in the fingers, wrists, elbows, and knees. The painful joints are swollen, but the skin which covers them has retained its natural color; and on applying the hand to the affected parts, no appreciable increase of temperature can be detected. The most striking feature of the case is the deformity of the affected joints. The patient has no fever, and retains her appetite, though pale, and very much reduced in strength. She told us, that in consequence of the pains having successively invaded most of the joints of the hand, she had been obliged to abandon her occupation as a seamstress. Menstruation is regular. Neither gout nor rheumatism was ever known to have attacked any member of the patient's family.

This woman formerly enjoyed good health. She had, however, scarlatina eight years ago, when the joints of the hands and wrists were attacked by scarlatinous rheumatism, which only lasted a few days.

As one of the antecedents in this case, I ought also to mention periodical megrim accompanied by vomiting, which on each occasion greatly prostrated her strength. Megrim, you know, is a diathesic disease, often met with in members of the same family. It most frequently shows itself in youth and mid-age: after the age of forty or fifty attacks of megrim, having already become more rare, cease to occur, to the great satisfaction of the patient; but at that period of life, other morbid manifestations appear.

In our patient of St. Bernard Ward, the megrims showed themselves with their usual intensity up to the age of thirty-eight: for the last two years, they have been less frequent, less painful; and from that time, the first symptoms of a new malady appeared. Both knees became the seat of pains, which at first were transient, and the knees seemed to be rusty; but by the end of the day, the pain disappeared, and the play of the joints was more easy. Gradually, however, the pain became more tenacious, and the articular movements were impeded. At the same time, the pain invaded both wrists, without leaving the knees: it recurred at intervals, and on each occasion seized some other joint. It first attacked the metacarpo-phalangeal joints, and then the phalangeal articulations: finally, after five or six months, it seized the shoulder, elbow, and tibio-tarsal articulations, and several joints of the toes, to such an extent that the patient could no longer bring to her employers the work which her disabled fingers had painfully accomplished.

For three months, this patient has been confined to bed, and unable to move. At present, although the treatment has sensibly modified the state of the joints, you can still recognize the curious deformities which give the fingers the appearance of long pods. The knees are swollen: there is great stiffness in the haunches, elbows, and shoulders. The greatest articular deformities are those of the wrists. The slightest movement occasions great pain in the joints, which present soft, doughy swellings, unaccompanied by redness: on the dorsal aspect of the right wrist, there is a swelling as large as a hen's egg.

This tumor seems to be composed of fibrous tissue with large pouches containing a semi-fluid substance. Pressure is not painful: and though it somewhat modifies the form, it does not change the volume of the tumor. It is probable, therefore, that the fluid within the tumor does not directly communicate with the sheath of the tendons in the wrists, nor with the radio-carpal articulation; or else, the fluid is too viscid, too thick, to be easily displaced. Perhaps the tumor is situated in the cellular tissue which lines the synovial cavity, and sometimes, in such circumstances, becomes very thick. Under the influence of general treatment, and the use of some hot sand-baths, the swelling, so great at one time, has entirely disappeared.

Do not suppose, gentlemen, that the articular nodes of gouty rheumatism have always a fibro-cellular character: some are of a bony nature, from the heads of the bones being swollen, a condition which may have proceeded so far as to destroy the mutual adaptation of the articular surfaces, and induce almost complete dislocation. In our patient, the phalangeal joints presented slight osseous enlargements, which imparted to the fingers a state to which Sydenham has alluded in treating of chronic rheumatism.

Our patient, helpless from the articular pains, had an exceedingly anæmic appearance, and her muscles were wasted from inaction. There were no muscular contractions: the patient had never had cramps in the limbs. The heart, carefully observed on several occasions, never showed the slightest abnormality in the rhythm of the pulsations, or in the character of the sounds. As a rule, the heart is not implicated in nodular rheumatism.

I stated that this patient's condition was very much improved by the treatment to which I submitted her: in point of fact, the joints have recovered some of their movements; and the nodes have lost much of their size and sensibility. The general health has become better: good fare has caused the anæmia to disappear. The patient will soon be able to leave the hospital, and resume her ordinary occupations; but sooner or later, new pains will show themselves, and whatever measures be adopted, there is reason to fear that the ameliorations obtained will be only transitory.

Though nodular rheumatism be most common in women, it is sometimes met with in men. You must have observed a male attendant in St. Agnes Ward who discharges his duties well rather than badly. To obtain the right of residence in the hospital, he assists the regular servants: you must have observed his peculiar gait as he moves from bed to bed. By trade, he is a basket-maker. He has been a resident in the hospital for the last seven years. Prior to that date, he earned his livelihood by his trade; and according to his own statement was never given to excesses. There were, he said, neither gouty nor rheumatic subjects in his family. Little by little, the knees, feet, shoulders and wrists, and at last the hands, became the seat of pains which continued some days, recurring in paroxysms. The joints soon became swollen and deformed; and the patient was obliged to keep his bed. When admitted to St. Agnes Ward, he was bent double. For seven months previously, he was unable to leave his bed.

The vertebral articulations in the dorsal and lumbar regions appeared to be soldered together: the coxo-femoral articulations were rigid. This patient, before taking to his bed, acting under the advice of numerous physicians, went to Wiesbaden, Aix-la-Chapelle, and Bourbonne. From his residence at these different stations, he derived only temporary relief, the rheumatism continuing to hold its place in most of the joints. For three years, I submitted this patient to a combined treatment by the tincture of iodine in large doses, vapor baths, sulphur baths, sublimate baths, and the application of bags of hot sand to most of the joints. At the end of this period, the affection seemed to be arrested, and the patient was able to leave his bed.

He still suffers sometimes, particularly when there is a change of weather; but the pain has become bearable, so long as the patient abstains from movements which irritate his joints. The malady was only checked: the stiffness of the vertebral column continues, the haunches and knees have not completely regained their power of movement, the tibio-tarsal articulations and the joints of the toes are almost entirely immovable; and in walking, the patient describes with each leg arcs of a circle: he cannot run, and his walk is like the waddle of a duck. The movements of the shoulder and elbow are partially preserved, as are likewise those of the wrists, but the digital phalanges are soldered together, and the fingers are semi-flexed, and unable to execute any precise movement.

Whatever we may do, this patient will never be completely cured: the osseous nodes and the subluxations will never disappear: it is accomplishing much to have obtained an almost entire cessation of pain. His general state will always remain relatively bad; and the hygienical conditions in which he now is, from prolonged residence in hospital, will predispose him to have, sooner or later, new rheumatismal attacks.

When describing to you the principal details of the case, I did not at all anticipate that the death of the patient was to occur at an early date. In the winter of 1863, he complained of pain in the chest; for several months he had bronchitis; and when he asked me to examine him, I ascertained the existence of pulmonary tuberculization, already far advanced, and from which he ultimately died.

At the autopsy, we found much slighter lesions than we had expected. Correctly speaking, the joints were not dislocated: there only existed forced flexion of some of them, and relaxation with thinning of the ligaments in the sense of extension, consequent upon the traction to which they had long been subjected. The articular cartilages were eroded, worn, at the points where pressure on the articular surfaces had been strongest and most prolonged. At last, the heads of the bones became partially deformed by the production of small granulated osteophytes in the vicinity of the points from which the cartilages had disappeared. In this situation, there were other points in which the osseous substance had become very thin, friable, and in such a state as to admit of its being cut by a knife. There were evident indications of chronic phlegmasia having existed. The nodosities of the joints were due to a strong projection of the partially displaced heads of the bones, but not displaced, as had been supposed during life, by the tumefaction of the bones; for, I repeat, there were only very small granular osteophytes, which hardly projected beyond the surface of the bones.

Here, I may appropriately narrate the case of the woman who occupied the last bed in St. Bernard Ward, and which presented to you the first symptoms of nodular rheumatism. The patient, forty-five years of age, irregular in her menstruation for some months, had lived under favorable conditions up to the day on which she entered the Hôtel-Dieu; was seized, after a chill, with pains in the knees and right haunch. She was, nevertheless, able to continue her occupation as saleswoman in a haberdasher's establishment: the pains, however, became more acute and more constant, and pains soon invaded the elbows and wrists. She then asked to be admitted to the Hôtel-Dieu: I detected an obvious engorgement of the knees and wrists, in which parts, as well as in the right haunch, every movement excited acute pains. In the evening, for fifteen days, there was an exacerbation of the pain, accompanied by slight fever.

The patient was pale, anæmic, and had little appetite. On listening to the heart, a blowing-sound was heard, soft at the base, and extending into the vessels of the neck; but there were no signs of organic lesion of the valves.

After treatment by tincture of iodine, an obvious amendment was soon observed in her local and general condition.

In these three patients, you have seen the disease begin by sudden pains in the large joints, pains which after a variable period were accompanied by engorgement and puffiness. Then, the disease showing itself in paroxysms, the first affected joints became the seat of more decided pains and engorgements, while the other joints, large and small, were invaded by pain. By and by, from pain and an altered state of the articular surfaces, all movement became impossible; and the patients were doomed to immobility of the limbs.

From the first manifestation of the disease, the patients became pale and feeble, though each paroxysm was accompanied by only a slight acceleration of the pulse, and was not attended by any loss of appetite. I never neglected the examination of the heart; and never did I detect in it any organic lesion. This fact is important, and is in accordance with general observation.

The affection is essentially chronic: whatever be done, its invasion is progressive; and if we do not intervene in the first attacks, the patients will sooner or later be condemned to complete helplessness.

The cases which I have now described to you are certainly insufficient to give you a complete acquaintance with the disease termed nodular rheumatism [*rhumatisme noueux*]: indeed for a general description of the affection, I should require to derive materials from all the works published on the subject since the beginning of the present century.

At the beginning of this lecture, I remarked, that nodular rheumatism is a disease seldom seen in hospitals, because from its being incurable, the cases are generally removed at once to the asylums for the incurable. Let me add, that it is a disease of rare occurrence: it is most frequently met with in women, and in them it is most usual at the period when the catamenia cease to appear: nevertheless, it is met with in young girls who menstruate regularly, and also in young pregnant women. For more than thirty years, during which my attention has been directed to this disease, I have only once seen it in a male. The patient was between sixteen and seventeen years of age.

Sydenham, who devoted much of his attention to gouty and rheumatic affections, very correctly remarked, that nodular rheumatism is an apyretic form of chronic rheumatism, differing essentially from gout, althougk, like gout, it recurs in paroxysms, and may last a lifetime. Sydenham adds: "It also, sometimes happens that the pains, after having continued for a long time with cruel severity, at last spontaneously cease. The affected joints, however, remain entirely incapable of motion. The joints of the fingers are, so to speak, turned over, and there are nodosities, as in gout, particularly on the inside of the fingers. The appetite is good; and in other respects, the general health of the patient is good."* Observe! Sydenham says that the joints of the fingers look as if they were turned over; and the autopsy of which I have given you the principal details, fully confirms the statement of the illustrious observer.

I am anxious to show you, that (as has been proved by my learned friend M. Lasègue) Sydenham perfectly recognized the fact that this form of chronic rheumatism, accompained by deformity and pains in the joints, must be distinguished from gouty disease. It is to be regretted that Garrod and Fuller in England,† and Trastour in France,‡ should have retained for the malady now under our consideration the name of *gouty* rheumatism. It is right to state, however, that Garrod, in the last edition of his treatise on gout, proposes to substitute "rheumatic arthritis" for the term "gouty rheumatism."

It is not my intention, gentlemen, to give you a retrospective review of all the works which have been composed upon *nodular rheumatism*, *nodes of the joints*, *mild asthenic gout*, and *primary chronic rheumatism:* let it suffice to inform you, that Fuller, Garrod, and subsequently Lasègue, Charcot, Trastour, and Plaisance have contributed, in elucidation of this subject, the best historical documents and the most valuable views in relation to the symptoms, pathological anatomy, and treatment of nodular rheumatism.§

Garrod and Fuller admit the existence of an acute form of gouty rheumatism commencing with intense fever and acute inflammation of several joints. Ere long, it assumes the chronic form; and there appear, at a later stage, the deformities which peculiarly belong to gouty rheumatism.

Generally, it is true, the disease shows itself at once in the chronic form. If, however, you carefully interrogate the patients, you will find that at an earlier period, they presented symptoms of acute or subacute articular rheumatism: in other cases, they will recall to their recollection, that in their youth, they had experienced muscular pains, pleurodynia, or lumbago. Some women will tell you that they have suffered from periodic megrim.

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* SYDENHAM: Opera Omnia. [Syden. Soc. ed. 1844, p. 260]: and SYDENHAM: Traduction de Jault, 1866, t. i, p. 422.

† GARROD: Nature and Treatment of Gout and Rheumatic Gout. 2d edition, 1863.

‡ TRASTOUR: Du Rhumatisme Goutteux: Thèse Inaugurale, 1853.

§ CHARCOT: Thèse Inaugurale, 1853.

It is important to ascertain these antecedents, because they prove that nodular rheumatism may show itself in patients who had had previously different manifestations of the rheumatic diathesis.

The pain of nodular rheumatism is generally seated in the knees, wrists, and fingers. At the onset of the disease, the joints continue painful and swollen for only one or two days: then, after a lapse of a fortnight, three weeks, or five or six months, new attacks of pain occur. You will then find, that there is not only engorgement of the soft parts, but likewise swelling of the epiphyses. The bones have, at this stage of the disease, undergone a special modification in their nutrition, the result of which is swelling and attenuation of the osseous tissue of the epiphyses.

In the large joints, this osseous tumefaction is very marked, and is distinct from the tumefaction of the soft parts of the articular structures. The synovial cavity is often distended by hydrarthrosis, and its thickened parietes seem to send prolongations beyond the joint, as you saw occur on the external aspect of the wrist of the woman who lay in bed 3 of St. Bernard Ward. Synovial bulgings are also observed upon the lateral aspects of the knees; but, after the lapse of a longer or shorter period, the soft, doughy nodosities are absorbed, and there only remain nodosities of the epiphyses.

The articular deformities are specially conspicuous on the hand and wrist: the fingers present projections corresponding to the metacarpo-phalangeal articulations: the first phalanges are in a state of flexion, which renders the heads of the metacarpal bones still more salient. The middle phalanx is in a state of extension, and the first and third in a state of flexion. From this relative position of the metacarpal bones and phalanges, results a strange form of the hand, consisting in an alternation of broken lines and projections. The metacarpo-phalangeal joint appears as a protuberance on the back of the hand, while the articulation of the first with the second phalanx projects at the side of the palmar surface. The whole hand is generally in a state of semi-flexion, and inclined towards the cubital side.

The typical condition now described, in which flexion predominates, is the most common. It must be stated, that all the joints of the hand are not equally liable to be attacked. The index, middle, and ring fingers are often the seat of most marked changes, while the thumb and little finger remain nearly exempt.

There is another type, called extension, in which the first and third phalanges are extended, while the middle phalanx only is bent: in cases of this description, the projection of the heads of the metacarpal bones is situated at the side of the palmar aspect of the hand.

Finally, in several cases, as is convincingly demonstrated by a glance at the plates of M. Charcot's inaugural thesis, the fingers present the appearance of claws. In other cases, the deformities are of such a character as to baffle description. I must, however, remark that sometimes the fingers are altogether in a state of extension, and slightly separated from one another: it is particularly in this variety that the fingers resemble necklaces.

Though the disease frequently seems to respect the thumb, leaving unimpaired most of its movements, it is otherwise with the great toe, which presents much deformity. The metatarso-phalangeal articulation projects very much, particularly inwards; and the great toe, pushed above or under the next toe, is in a state of extension or forced flexion. The other toes may present deformities analogous to those met with in the hand; but as a general rule, they are much less marked.

We must now pass in review the alterations of the large joints. The forearm and leg are usually in a state of semi-flexion: this position, when once acquired, is generally persistent. The haunch and shoulders almost always which plays so important a part, even beyond the affected joints, is it not a justifiable hypothesis, that the peripheric nervous system is so injured as to cause the contractions? In 1853, M. Charcot doubtingly suggested the hypothesis that the contractions might be dependent on reflex action, his idea being that the seat of the excitomotor action was in the affected joints. This hypothesis must be rejected, at least in the cases in which muscular pain precedes the lesion of the joint, or continues after the joint has ceased to be painful.

I am inclined, therefore, to believe, rather that the muscular pain, soon followed by retraction, persistent, and independent of the muscular lesion, ought to be looked on as a manifestation of the disease. Again, this manifestation probably has its seat in the nervous trunks which supply one class of muscles, or in the ramifications of the nerves of each of the contracted muscles. Afterwards, when I come to discuss the nature of nodular rheumatism, we shall see whether this nervous manifestation, as well as the arthritis, is rheumatic.

Gentlemen, let me now return to the strictly clinical part of my lecture. When the malady is perfectly confirmed, that is to say, after it has lasted several months, the inflammation usually extends by several attacks to a great many joints; and then we seldom see retrocession of the disease. On the contrary, its course is generally progressive: joint after joint is successively attacked, and the deformities become more and more decided. The joints are anchylosed: and when an attempt is made to move them, the hand can detect frequent crepitation, which is produced by the fibrous parts being torn, and by the rubbing of the eroded osseous or cartilaginous surfaces. Moving the joints always occasions great pain: and the rupture of the anchyloses has never given favorable results.

I ought, however, to remark, that under the influence of constitutional treatment, it sometimes happens, not only that the disease does not progress, but that the arthritis is so greatly modified that on anatomical examination it is no longer possible to detect any, save slight, articular lesions. This occurred in the basketmaker whose case I described to you.

Be the case what it may, when the principal joints of the superior and inferior extremities have been the seat of the disease, to move is almost absolutely impossible; and the patients are obliged to remain either in the horizontal position or seated.

The manner in which the joints are invaded is a point to which M. Charcot has very judiciously called attention. In nodular rheumatism, *symmetry* is the rule, that is to say, the homologous articulations are simultaneously attacked, the exceptional cases being very few. This fact has been stated by Budd and Romberg.

Nodular rheumatism does not manifest itself only in the joints and muscles: during the course of the disease, there are observed sciatic pains of variable duration and intensity. In fact, we meet with a particular class of lesions to which attention has only been directed within the last few years. I remarked to you that, generally speaking, nodular rheumatism has no action on the heart: valvular lesions, in truth, are scarcely ever met with in this class of rheumatic persons; sometimes, however, by auscultation, we can detect blowing and rasping sounds, which seem to have their seat in the mitral and aortic valves. In 1846, Romberg, and at a later date, Trastour, Charcot, and Peter, observed undoubted examples of organic affection of the heart in individuals having nodular rheumatism, and who had never suffered from acute articular rheumatism. Moreover, autopsies have shown that the pericardium may also be the seat of very extensive inflammatory lesions. In four out of nine autopsies made by Dr. Cornil at preserve a certain degree of mobility. Observe, gentlemen, that the superior extremities only may be invaded by the disease, the patients preserving the use of their lower limbs; but, on the other hand, it is not uncommon to see the vertebral column anchylosed in different parts, the patients being unable to stoop, or turn the head; and when the dorso-lumbar region is invaded, the afflicted are often bent forward and unable to straighten themselves. Finally, the temporo-maxillary articulation may be the seat of chronic rheumatism, setting in abruptly, as M. Charcot had an opportunity of observing in six patients of the Salpêtrière.

I have already said that the articular pain, acute in its form and recurring at intervals, may vary in each individual. These pains depend upon the morbid alterations of the synovial membrane, cartilages, and epiphyses.

Pains are sometimes experienced in the continuity of the limbs: they are of a spasmodic character, occur chiefly at the time of the articular attacks, and are readily distinguished by the patients from the pains which accompany muscular fatigue: they compare them to cramps. These pains are seated in the muscles of the thighs and legs, arms and forearms. What is their cause? What is their nature?

You have observed, gentlemen, that the patients know how to place pained limbs in positions which lessen the pain. In inflammation of the psoas, the patients lie on the side corresponding to the affected muscle, the thigh being semi-flexed on the pelvis. The muscle is thus relaxed, and there is no dragging of the inflamed fibres: but to produce this flexion, it is necessary that the muscles which flex the thigh on the pelvis should contract, or, that the patient, leaving the thigh immovable, should bring down the pelvis in such a way as slightly to bend the thigh. In muscular wryneck, when the trapezius is affected with rheumatism, we see the sterno-cleido-mastoid muscle contract to obviate the necessity of traction by the pained muscles. In lumbago, to prevent the sacro-lumbar muscles from being dragged by the weight of the body, the oblique and straight muscles of the abdomen contract, so as to keep the trunk either immovable, or slightly bent forwards.

The unaffected muscles come to the aid of the pained muscles, to prevent traction of the latter, which might renew and aggravate the pain. Likewise, when joints are the seat of pain, the muscles act in such a manner as to lessen the pain, and keep the joint at rest.

In a similar manner, an attempt has been made to explain muscular pains in nodular rheumatism, by the fatigue resulting from the instinctive tutelar contraction of the muscles. But opposed to this explanation is the fact, that the muscular contractions sometimes show themselves before the joints become much affected; and it is not unusual to see them go on increasing, long after the joints have ceased to be the seat of pain.

In these cases, then, the muscular contraction is independent of, and not the consequence of, chronic rheumatic arthritis. When the muscular and articular pains seem to progress together, we shall on the contrary be disposed to think that the articular pain is sometimes augmented by the spasmodic contractions of the muscles.

As to the cause and nature of these muscular pains, soon followed by persistent retraction, it is impossible to regard them as the consequence of the arthritis; and to me it appears more rational to look upon them as a manifestation of the general local state, a manifestation which may be absent, but which is sometimes a predominating phenomenon of the disease.

Hitherto, in nodular rheumatism, no lesion of the central or peripheral nervous system has been found: but in a disease in which pain is an element which plays so important a part, even beyond the affected joints, is it not a justifiable hypothesis, that the peripheric nervous system is so injured as to cause the contractions? In 1853, M. Charcot doubtingly suggested the hypothesis that the contractions might be dependent on reflex action, his idea being that the seat of the excitomotor action was in the affected joints. This hypothesis must be rejected, at least in the cases in which muscular pain precedes the lesion of the joint, or continues after the joint has ceased to be painful.

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Landré-Beauvais and Pinel have observed pulmonary complications: their patients (patients at the Salpêtrière) succumbed under that ataxo-adynamic state, so common in the pneumonia of the aged. But although these pulmonary complications may be considered as diseases independent of the rheumatic diathesis, I do not think that a similar opinion is tenable in respect of pleuritic complications, particularly when pleurisy with effusion occurs simultaneously with pericarditis, and when the latter presents all the characters of an acute inflammation.

Patients affected with nodular rheumatism often become albuminuric during the latter years of life, a clinical fact for which we are also indebted to the work of Dr. Cornil.* Albuminuria, it is true, is then, frequently, only a symptom of chronic inflammation of the bladder and pelvis of the kidneys: in some cases, however, it has been shown by the autopsy, that there existed the alterations of the kidneys characteristic of Bright's disease.

Let me say, that you ought henceforth to examine carefully patients affected with nodular rheumatism to ascertain whether there exist any cardiac, pleural, or renal complications attributable to the rheumatic diathesis.

You know, gentlemen, that in cases of acute articular rheumatism, it is not unusual to observe the cerebral symptoms described under the generic name of "cerebral rheumatism." In previous lectures, I have addressed you at length upon the localization of rheumatism in the encephalon. It is a complication seldom met with in nodular rheumatism. I ought, however, to remind you that Dr. Vidal has related the case of a man aged seventy-three, who died from a cerebral affection, after having had symptoms of nodular rheumatism for several years.†

Let me also remark, that M. Charcot has seen, coincident with nodular rheumatism, certain affections of the nervous system, such as shaking palsy —tremors at least—and locomotor ataxy.‡ But such cases are very unusual; and there is no proof of there being any correlation in the pathogenesy of these nervous diseases and nodular rheumatism.

Nodular rheumatism is not immediately dangerous to life, when no complication supervenes. The patient who first excited the attention of Haygarth, and suggested his work on nodosities of the joints, was ninety-three years of age. In the dormitories of the infirmary of Salpêtrière, you will see a great many old women who have been for years affected with nodular rheumatism. They are infirm rather than unwell; that is to say, they have incurable lesions resulting from nodular rheumatism, but the disease itself is no longer in an active form. They are, however, placed in unfavorable hygienical conditions by the immobility to which they are condemned. They are almost always either lying down or sitting in an armchair, the appetite is poor, nutrition is badly performed, and they acquire a greater aptitude to contract the diseases of old age. In those in whom chronic has preceded the attack of subacute rheumatism, it is not uncommon for the articular pains to reappear accompanied by fever; under such circumstances, acute cardiac complications may occur.

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* CORNIL: Mémoir sur les Coincidences Pathologiques du Rhumatisme Articulaire Chronique. [*Comptes Rendus des Sciences et Mémoires de la Société de Biologie*, 4me série, t. i, année 1864.]

† VIDAL (E.): Thèse Inaugural, 1855.

‡ CHARCOT: Leçons sur le Rhumatisme Articulaire Chronique, published in the Gazette des Hôpitaux, 1867.

Pulmonary phthisis is another organic change which terminates the life of patients suffering from nodular rheumatism. Bear in mind, gentlemen, the case of the basketmaker who for many years had suffered but little from his nodular rheumatism, and who died with all the signs of rapid pulmonary phthisis. Numerous observers have noticed the existence of pulmonary tuberculization in rheumatic patients. I do not mean to say, gentlemen, that phthisis is necessarily a manifestation of the diathesis which induces nodular rheumatism, although there is a rheumatic phthisis; but I wish to call your attention to an unfavorable complication which may originate, to a certain extent, in immobility and a forced sedentary condition.

The anatomical articular lesions have been studied with great care. The affected joints present numerous traces of chronic inflammation; they are deformed, the deformity being due, as I have said, to alterations of the osseous, synovial, or periarticular cellular tissue.

The articular epiphyses are thick, their original structure is augmented in volume, and, in the form of stalactites, there are new productions of osseous tissue. These productions are generally seated at the peripheric line of insertion of the cartilage. Within the joint we can sometimes detect osseous union of the surfaces in apposition.

The cartilages present various alterations. They are attenuated, eroded in some places, or velvety. The latter change consists in a disintegration of the cartilage, so as to give it the appearance of Utrecht velvet. The attenuation is occasionally so great in some places as to constitute real ulcerations of the cartilage, showing the osseous tissue at their bottom.

The epiphyses are at the same time thick, spongy, and very attenuated; large rings filled with fatty matter are visible, and the attenuated tissue is easily cut with the scalpel. The whole synovial membrane of the joint is that which presents the most interesting alterations, alterations evidently demonstrating an inflammatory action. Sometimes there is a very intense vascular injection at the edges of the synovial membrane, which may present morbid prolongations, extending from one articular surface to the other. These prolongations are the origin of the cellular fibres often observed in the joints; and as they may become the seat of cartilaginous and calcareous products, they explain the presence of the intra-articular foreign bodies sometimes met with in nodular rheumatism. But it is a fact deserving of notice that we never find pus in the joints, and seldom even an excess of synovial fluid. This form of arthritis has been called "dry arthritis."

In describing to you articular nodosities, I said that they were never osseous, and that the soft parts shared in producing these deformities. In fact, anatomical examination shows that the cellular tissue which lines the synovial cavity may sometimes attain a great development. I have seen these fibrocellular deformities disappear under treatment; at other times, however, the hyperplasia has been so great as to leave marked permanent deformity. Moreover, the thickenings of the cellular tissue, by contracting adhesions with the surrounding parts, cause fibrous anchyloses of such a character that when the ligaments are cut, the joint still retains its anomalous position.

The inflammatory action seldom attacks the articular ligaments; and we never find deposits of urate of soda in them or in the periarticular cellular tissue. Neither extra-articular tophus, nor deposits of urate of soda in the cavity of the joints are ever met with in nodular rheumatism.

The muscles, I have said, are sometimes retracted, and in such cases, their tendons act like cords, which maintain the joints in their abnormal position. The immobility to which many rheumatic patients are condemned explains the fatty degeneration sometimes observed in the muscular fasciculi.

Such, gentlemen, are the principal lesions met with in nodular rheumatism. I ought, however, to add, that in certain cases, this disease leaves scarcely any specially characteristic alteration of the joints; that is to say, that the synovial membrane and cartilages do not present any of the lesions which I have been describing. There then remain only acquired articular deformities, partial luxations, and nodosities of the epiphyses. Probably, in those cases, the diathesis has been long quiescent, and under the influence of an improved nutrition, there has taken place reparation of the changes in the cartilages and synovial membrane.

I shall be brief upon the other anatomical lesions of nodular rheumatism. Those, however, which have been detected in the heart and kidneys seem so important that, without going into long details, I must at least mention them. Garrod and Fuller, in describing the acute form of nodular rheumatism, have called attention to the point of transition between the acute and the suddenly invading forms of rheumatism. All observers have been careful to seek for the cardiac lesions which are found in nodular rheumatism. I have told you that as a general rule chronic nodular rheumatism is not accompanied by affections of the heart; but I have also stated that heart diseases have been sometimes met with, and that autopsies have proved that in chronic rheumatism there may exist acute pericarditis with fibrinous deposits on the surface of the pericardium, and chronic pericarditis with complete adhesions of the serous membrane of the heart. In treating of the nature of nodular rheumatism, I shall employ those anatomical facts which decisively connect the nodular malady with the rheumatic diathesis.

Drs. Charcot and Cornil have found pretty frequently the lesions which belong to albuminous nephritis; which lesions differ from those met with in gout, and (as Dr. Charcot remarks) are constantly found in cachectic persons.

Gentlemen, what is the etiology of nodular rheumatism? Most authors assign an important part to damp cold. It is quite true that many of the patients have lived for a long time in damp places. It is true that the poor, who are often exposed to cold, are more subject than the rich to nodular rheumatism. It is likewise true that in certain damp countries, it is so common as to be almost endemic. We must also, however, bear in mind that a special individual disposition is required to enable the moist cold to determine the production of nodular rheumatism. Bad hygienical conditions have likewise their influence: they debilitate the system, and render it more impressionable to the causes of the disease. A similar result is produced by great bodily fatigue, profuse hemorrhages, and frequent pregnancies. Bean, while he admitted the undoubted influence of damp cold, considered that mental worry and dyspepsia had something to do with producing nodular rheumatism. He relates the case of a lady who, living in easy circumstances in a residence having a complete southern exposure, consulted him for articular pains with deformities of the joints. In this case, there seemed to be no damp cold; but on carefully interrogating the patient as to her antecedents, he discovered that the lady, in her youth, had lived in a very damp region, and had, when about fifteen or sixteen years of age, suffered from her first attacks of nodular rheumatism. Having soon afterwards removed to a situation where the hygienical conditions were more favorable, she continued thirty years without articular pains, but under the influence of deep grief, she became the subject of dyspepsia. This lady had had almost complete loss of appetite, and had been losing flesh for some months: she soon afterwards became rheumatic, the disease which had slumbered for thirty years waking up.

It is not unusual, gentlemen, to observe similar periods of arrest in the manifestations of nodular rheumatism. You ought always carefully to inquire whether or not the patients, at some anterior period, have suffered from articular pains, of which they may have almost entirely lost all recollection.

Nodular rheumatism, so rare in men, is so common in women that in the Salpêtrière, according to the statistics of MM. Charcot and Vulpian, from a fifteenth to a twentieth of the population of that asylum are sufferers from it. Nodular rheumatism generally has its beginning at the time of the establishment or cessation of menstruation; but sometimes also during pregnancy. Garrod believes that the ovario-uterine functions have no special action in originating nodular rheumatism, and act only when they are a cause of debility. Gonorrhœa, when it localizes itself in a joint, is sometimes the cause of calling forth and generalizing nodular rheumatism. Garrod gives a case in point: and in 1832, when acting at the Hôtel-Dieu for Professor Récamier, I had as a patient a young groom, most of whose joints had been invaded by nodular rheumatism, consequent upon an attack of gonorrhœa.

Let us now study the nature of nodular rheumatism. Gentlemen, I have long taught that nodular rheumatism is neither gout nor rheumatism. To prove that gout has nothing to do with rheumatism, it is sufficient to remark, that the persons affected with the latter present neither the condition nor the symptoms of gouty maladies. You have seen that nodular rheumatism chiefly attacks women, whereas gout almost exclusively attacks men. Gout is chiefly a disease of rich men, and of men who have lived luxuriously, and *suddenly cease*, as Sydenham remarks, to lead an active life. The conditions under which rheumatism originates are totally different; as it seldom attacks persons who are not enfeebled by some cause. Moreover, the gouty articular deformities are extra-articular and characterized by tophaceous formations of urate of soda, while, at the same time, the urine is often charged with uric acid. I have often, but always fruitlessly, sought for tophaceous concretions in our patient of St. Agnes Ward: the nodosities, I again repeat, which were so considerable, were wholly constituted by the projection of the articular portion of the bones, and were smaller than could have been supposed by the external appearance. The serum of the blood contains no uric acid as in nodular rheumatism. Nodular rheumatism, therefore, is not gout. Women, however, after the cessation of menstruation, when they assume some of the characteristics of the male sex, may present gouty manifestations. These remarks still retain all their original value: nodular rheumatism must not be confounded with gout, there being very many morbid symptoms to distinguish the one from the other.

Is it the same with rheumatism? May not the articular nodosities be consequences of the rheumatic diathesis? There was a time when I should have replied in the negative to this interrogation. It was, in fact, a doctrine generally accepted by myself and others, that nodular rheumatism was not a febrile disease, that the patients never had rheumatic affections of the heart, or pleuræ, nor metastases to the brain, stomach, or intestines; and according to the views entertained by me, as well as by all others, "gouty rheumatism" was neither gout nor rheumatism. I was no more disposed than Garrod and Fuller to consider it a hybrid disease originating in gout and rheumatism: and with them, whilst I was unable to say what the disease was, I was equally unable to say what it was not. I had "learned from clinical experience, that the circumstances under which gouty rheumatism showed itself, the inveterate obstinacy of the symptoms, the peculiar alteration of the joints, and the remedies most useful in the disease, all point to its being intimately associated with some peculiar constitutional alteration."*

These conclusions are still tenable. However, the fever, the acuteness of the pains, and the generalization of the disease over all the joints of the hand and wrist, as observed in some cases of nodular rheumatism—the acute form of the malady (which we have sometimes an opportunity of witnessing at the commencement), afterwards becomes chronic—the acute manifestations which occur in the heart and pleuræ, in the course of the chronic form, testify to the existence of a rheumatic diathesis, which will show itself at different periods of life by certain morbid alterations always seated in the fibro-serous tissue of the joints, heart, and pleuræ. Moreover, there have sometimes been observed in the paroxysms, cerebral disturbance, and disturbance of the digestive organs, apparently alternating with the articular pains. Finally, if the pathological antecedents of the patients be analyzed, it will sometimes be found that there were indications of a rheumatic diathesis in their progenitors, and that the patients will state that they themselves have, anterior to the existing attack, experienced morbid symptoms referable to a diathesic condition, such as megrim, eczema, diseases of the eyes, and successive attacks of erysipelas, which according to some physicians—M. Bazin in particular—are often attributable to the rheumatic diathesis.† Again, are not the pains and muscular retractions observed in nodular rheumatism the result of the morbid cause, rheumatism, acting pathogenically upon the muscles and nerves at the same time as upon the joints?

I do not believe, however, that we are yet in a position to arrive at any exact, precise conclusions in relation to this point. Still, I have considered it my duty as professor to lay before you the facts and arguments which appear to have modified the opinion of several clinical observers as to the nature of nodular rheumatism.

The discussion into which I have entered renders it unnecessary for me to speak at any length on the subject of diagnosis. It is impossible to confound nodular rheumatism with gout. There are great differential characters; and they are particularly marked when we compare the local affections of the two diseases. The articular deformities of nodular rheumatism bear no resemblance to those of gout. In rheumatism, they are not tophaceous concretions, but are produced by the projections of the ends of the bones, augmented in volume, incrusted by osteophytes, forming irregular angular excrescences: they are also in part produced by the retraction of certain muscles and the atrophy of their antagonists. The joints, partially dislocated, the articulating surfaces of which have lost their normal relations, become fixed in the vicious position which they have assumed: though we may hope to arrest the farther progress of these anchyloses, we cannot reduce those already formed. The aspect of the fingers turned back upon their external side, pushed backwards and resembling long pods, have no similarity to gouty fingers.

In nodular rheumatism, though the small joints may be affected, it is not, as in gout, the articulation of the great toe of one foot which is first affected, but more frequently, the metacarpo-phalangeal articulations of the index and middle finger in both hands, in conformity with the law of symmetry pointed out by Dr. Charcot. This law of symmetry certainly does not exist in gout.

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* Garrod: On Rheumatism, Rheumatic Gout, and Sciatica. 3d edition, p. 345. London. 1860

† Cornil: Mémoire sur les Coincidences Pathologiques du Rhumatisme Articulaire Chronique, lu à la Société de Biologie. [*Comptes Rendus des Sciences, et Mémoires de la Société de Biologie*, 4me série, t. i, 1861.]

In nodular rheumatism, the feet and inferior extremities are not first affected as in gout. In both our patients, you have seen, that although the knees were first attacked, the feet were not attacked till long after the hands. Generally, the malady first shows itself in the hands. In gout, as a rule, all the joints do not at the same time suffer with equal severity: in rheumatism, all are seized in succession, and continue to be affected, without excepting the vertebral and maxillary articulations.

Nodular rheumatism, insidious in its beginning, declares itself by pains, which are less characterized by violence than by constancy, very different as you know from what occurs in gout: when once it sets in, it has an onward progress marked by exacerbations.

It is much more common than gout in women. Like acute articular rheumatism, its development is very much under the influence of immediately exciting causes, such as cold, particularly damp cold. It is very doubtful whether heredicity has any share in producing nodular rheumatism

It is a very important fact, that in nodular rheumatism we do not find symptoms characteristic of the uric diathesis, which is nearly always associated with gout—the *uric* diathesis, which we must be careful not to confound with the *gouty* diathesis, as some have done.

The remarks which I have made, gentlemen, upon the tenacity of the symptoms, the paroxysms of the disease, the onward progress, the articular deformities, and the persistent contractions, sufficiently prove that medicine cannot suppress attacks of nodular rheumatism. However, hygienics, and sometimes different forms of treatment, may impede the onward progress of the disease, render longer the intervals between the paroxysms, and prevent the invasion of other joints. A general, profoundly modifying treatment is the only means of attaining a satisfactory issue.

Thirty years ago, when doing Professor Récamier's duty at the Hôtel-Dieu, there was admitted to my wards, a young lad, to whose case I have already alluded when treating of the etiology of nodular rheumatism. This young man, consequent upon an attack of gonorrhœa, was seized with pain in very many joints, many of which gradually became stiff and deformed.

At that period, there existed a much stronger disposition than at present to believe that gonorrhœa might be the origin of syphilis; and as I then held that pox had a share in causing alterations in the joints, I subjected the patient to antisyphilitic treatment. Sublimate baths were ordered to be taken three times a week, and to my great satisfaction the attacks became less acute and less frequent, and finally completely disappeared. So much was this the case that the joints recovered freedom of movement, and ceased to present any appreciable deformities. It is true, that in this young man the malady was incipient, and that the deformities were chiefly located in the soft structures of the joints.

I do not hesitate then to prescribe the same baths in new cases of rheumatism, but I confess that I do not always obtain the same success from their use. Be that as it may, sublimate baths, even irrespective of the existence of any syphilitic diathesis, have rendered me great service: and I have continued to prescribe them, taking care to discontinue them when they did not produce satisfactory results. You saw the beneficial effects they produced in the case of the woman who occupied bed 3 of St. Bernard Ward. Mercurial preparations, on account of their action on the osseous system, may be administered internally, provided recourse be had at the same time to stomachic remedies, and in preference to all others to powder of cinchona. Preparations of colchicum have always produced mischievous consequences, because they cannot be long continued without causing disturbance of the stomach and intestines. They have now been entirely abandoned in the treatment of nodular rheumatism.

During the last few years, Dr. Noël Gueneau de Mussy, having remarked that most mineral waters which act beneficially in rheumatism, contain arsenic, has proposed to substitute baths of the subcarbonate of soda and the arseniate of soda for the sublimate baths. This mode of treatment has been made the subject of a memoir to the Academy of Medicine by my hospital colleague. In the patients in whom nodular rheumatism is still acute, baths containing 100 grammes of subcarbonate of soda and one gramme of arseniate of soda, produce stimulating effects which contraindicate their use. In these cases, Dr. N. Gueneau de Mussy employs the arseniate of soda only in the proportion of from one to five grammes in a gelatinous bath.* Baths of arseniate of soda have sometimes, though seldom, induced slight toxic phenomena: they may be employed for several months without causing any serious symptoms.

I have not neglected to employ arsenical baths; and yet I have been obliged to discontinue them from their not seeming to lead to much improvement in the state of the joints.

When you wish to prescribe arsenic internally, have a clear conception of your doses, and prescribe in such a manner that no error of a patient can cause his death. Prescribe—as I have done for many years—five centigrammes of the arseniate of soda to 120 grammes of water: of this solution, order a teaspoonful to be taken morning and evening; and do not increase the dose till you are certain of the tolerance of the stomach for the medicine. If you prefer the form of pill, order pills of arsenious acid, each containing two milligrammes: four, five, or six of such pills may be taken daily. These arsenical medicines ought to be taken at mealtimes: it is the best means of securing rapid absorption, and avoiding irritation of the stomach.

Gentlemen, there is no specific for nodular rheumatism. Almost every individual patient requires his own special remedy; and, probably, that is because nodular rheumatism bears an intimate relation in its manifestations to an individual diathesic state, which may demand mercurial, or arsenical preparations, or simply alimentary and pharmaceutical tonics.

There is, however, a medicine which, although it cannot be looked upon as a specific, appears to act beneficially with more uniformity than any other upon articular nodosities: I refer to iodine.

My friend and learned hospital colleague, Dr. Lasègue, in 1852, when my *chef de clinique*, thought of employing iodine. He had, he said, "obtained beneficial results from this medicine in the least rebellious forms of osseous rheumatic swellings."

In Dr. Lasègue's opinion, nodular rheumatism is a form of rheumatism. He gave iodine with complete success to a patient affected with nodular rheumatism. "This man, who occupied bed 11 of St. Agnes Ward, had all the joints of the feet and hands deformed; the wrists, elbows, and shoulders were slightly attacked; the knees were swollen and painful, and even the articulations of the cervical vertebræ were not spared. The patient was forced to remain almost entirely in bed. He was treated by tincture of iodine: at an after period, in addition, as a calmative and auxiliary resolvent, bags of dry hot sand were applied. After some weeks the affection was arrested in its progress; at the end of a month, some joints had recovered their mobility: the cure proceeded slowly, but continuously, and after four months of treatment by iodine, the patient was performing the laborious duty of attendant on the sick in an hospital."

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* NOEL GUENEAU DE MUSSY: Du Traitement du Rhumatisme Noueux par les bains arsénicaux. [*Bulletin de Thérapeutique* for September, 1864.]

This quotation, which I take from M. Lasègue's memoir on the subject, published in the Archives Générales de Médecine for August, 1856, leaves no room for doubt as to the therapeutic value of iodine in cases of nodular rheumatism. Add to that statement the fact, that, since 1852, M. Lasègue and I have often used this remedy with decidedly beneficial results.

The case of the woman who lies in bed 3 of St. Bernard Ward is another to be added to the list of ascertained recoveries. Have recourse, therefore, gentlemen, to the tincture of iodine; use it, and you will often have occasion to congratulate yourselves on having employed it. But to obtain success with this medicine, you must know how to manage it, remembering, at the same time, that every chronic disease demands a long course of treatment. Order the iodine to be taken daily at the morning and evening meals, prescribing, in the first instance, ten drops of the tincture, in sherry wine or sugared water, as recommended by M. Lasègue. You may progressively augment the dose to one, two, three, four, five, or six grammes, without occasioning any inconvenience to the stomach; and more surprising still, you will often be astonished to discover that digestion goes on with remarkable activity. How does tincture of iodine act upon nodular rheumatism? I cannot believe that it possesses a specific action, as it does not produce equally beneficial results in all patients. I am inclined to think that its action is complex; that is to say, that it acts upon general nutrition by assisting digestion, and that in this way, perhaps, it exerts an indirect action, in some patients, upon articular engorgements.

We have at our command then a certain number of medicines, which act as general alteratives, which may be of great use in nodular rheumatism; but with a view to promote resolution of the articular inflammation and assuage the pains of the stomach, it is useful to employ douches of hot sand. That is a resolvative and calmative measure of great power, provided it be judiciously employed. You must plunge the affected parts into hot sand, or allow the sand as hot as can be borne to fall on them. The patients then complain of a very painful sensation of burning. You can, however, always discover, by the thermometer, the degree of heat which each patient can bear. This may be from 60° to 70° Centigrade. Douches or local hot sand-baths ought to be given three times a day, lasting for one or two hours each time. It is important that the sand be kept at the same temperature: this can easily be done, because sand cools slowly, and can readily be replaced when it begins to get cold. By attending to this rule in the use of the hot sand, the patients soon experience decided relief; and it is easy to detect a rapid diminution in their articular engorgements.

LECTURE LXXXIV.

ACUTE ARTICULAR RHEUMATISM AND ULCERATING ENDOCARDITIS.

Very great frequency of Acute Articular Rheumatism.—A Diathesic Disease.—Peculiarly an affection of the Fibro-Serous Tissue.—Rheumatism of the Large and Small Joints.—Primary or Secondary Rheumatism.—Rheumatism of the Heart, the origin of organic diseases of the organ.—Rheumatism of the Pleuræ, Lungs, and Membranes of the Brain and Spinal Marrow.—Rheumatic Metastases.—No Specific Treatment for Acute Articular Rheumatism.—Rheumatic Ulcerative Endocarditis.—Ulcerative Endocarditis Independent of the Rheumatic Diathesis.—Atheromatous Endocarditis.—Visceral Emphraxis.—Capillary Embolism.—Alteration of the Blood consequent upon Ulcerative Endocarditis.—Typhoid Symptoms.

GENTLEMEN: We have now been engaged in the study of gout and nodular rheumatism: and at the beginning of the year, I devoted several lectures to the clinical study of cerebral rheumatism. To-day I wish to discuss acute articular rheumatism, without intending, however, to give you a complete systematic description of the disease. Let me remark, that you will not see the disease at the bedside of the patient always presenting itself with the retinue of symptoms and complications described in works on pathology. At the close of the lecture, I shall call your attention to a newly described complication of articular rheumatism to which the name *ulcerative* endocarditis has been given. Let me here remark, however, that this modification of endocarditis may occur irrespective of any rheumatic manifestation.

Hardly a month passes during which you have not an opportunity of studying rheumatic arthritis in our clinical wards. The action of cold, particularly when the surface of the body is covered with sweat, is a very frequently determining cause in persons of the rheumatic diathesis. When you interrogate the patients at the hospital, nearly all of them will tell you that they have been struck with cold, either when hard at work or immediately after discontinuing severe labor. Some will say that they have been exposed to a draught of air, and that they have felt the whole body enveloped in cold. Others will tell you that they have been exposed to cold when passing from a hot to a cold damp atmosphere. In the evening, in the night, or during the next day, after experiencing these sensations, the patients are seized with shivering, succeeded by great heat and perspiration, and at the same time, nearly always, with acute fever and profuse sweating.

Rheumatic arthritis is continuous, hardly ever presenting paroxysms. The pulse is rapid, large, and resisting. The sweating is always profuse, and when the patients are uncovered, you can see the perspiration collected in little drops over nearly the whole body. The perspiration has a peculiar odor, such as is hardly ever met with in other febrile diseases. You will also be struck with the paleness of the complexion: the face itself is often of a dull blue, and this general pallor is in strong contrast with the bright red hue of the skin.

There is loss of appetite: the tongue is white, but only slightly saburral: there is no tendency to vomit: constipation is the rule in such cases. The only complaint made by the patients is of pain in the joints. You will observe that they assume the dorsal decubitus, and remain motionless in bed, in dread lest the acute pain in the articulations be aroused by the slightest movement. The pain often first declares itself in the knees and insteps: afterwards, at the end of a period varying from some hours to three or four days, we find numerous joints invaded by the rheumatic inflammation. The pain has sometimes an upward progress: that is to say, that it ascends from the insteps to the knees and haunches, or from the wrists to the elbows and shoulders. It is not unusual for the disease to declare itself, in the first instance, only upon one side of the body, and subsequently, to invade, in similar order, the joints of the other side.

This course of the disease has never seemed to me to have any notable importance in relation to its duration and termination; but it is otherwise when the rheumatism simultaneously attacks the large joints, the wrist, the insteps, and, even also, it may be, the small articulations of the hands and feet. When the latter are invaded by acute rheumatism, and particularly when that invasion occurs at the commencement of the disease, one may conclude almost with certainty, that the articular rheumatism will be of long duration; and consequently, that the disease will have a degree of gravity relatively greater than in other forms of articular rheumatism. In fact, the manifestations in the hands and feet testify to the greater power of the disease, which in less severe cases generally remains limited to the large joints.

In acute articular rheumatism, all the joints, large and small, may be attacked in succession. The articulations of the clavicle with the sternum and acromion, the maxillary articulations, and those of the vertebral column, may be seats of rheumatic pains.

Rheumatism, then, has a special predilection for the joints, but, in general, it is the large joints which are most commonly invaded. Gentlemen, I have frequently pointed out to you the characters of rheumatic arthritis. At the bed of the patient, we have seen that the affected joints were swollen and painful. The swelling is in the tissues which surround the joints; but this swelling is principally an intra-articular effusion. You cannot put your hand on the joint or cause the slightest movement of the articular surfaces without occasioning very severe pain. By applying the hand gently to the diseased joints, a very appreciably increased temperature can be detected. In a very few cases, redness may be remarked around the large joints, as is seen in arthritic affections of another kind. In rheumatism, there is a white swelling of the superficial tissues, but when the rheumatism is in the wrist, hand, instep, or small joints of the foot, the swelling has a rosy hue. In the course of the numerous tendinous sheaths of the wrist and instep, we see red streaks indicating the part which the sheaths have in rheumatic inflammation. In these cases, the wrist and hand are deformed. All the fingers are immovable, swollen, separated from one another, and have the shape of large spindles. The dorsal surface of the hand is rounded: there exists a state of true acute œdema, and between the hand and forearm, there is perfect continuity without any line of demarcation. Similar remarks are applicable to the foot, when it and the tibio-tarsal articulation are simultaneously invaded.

When hand and foot are the seat of rheumatic inflammation, the disease may remain stationary there for a long time, giving rise to the fear that the articular tumefaction may become the beginning of a white swelling. This serious complication, however, is seldom observed except in scrofulous and tuberculous subjects. It is much more usual to see the small joints retain a very great stiffness, which can only be got rid of by producing, several times a day, slight movements of flexion and extension in the rigid fingers.

When the rheumatism has been seated only in the large joints, it is usually found that the local inflammation lasts only for a few days—from three to seven days. It often happens that the pain, inflammation, and effusion disappear from one day to another, but in such cases, other joints are seized. The rheumatic matter, to use Van Swieten's expression, has a very migratory tendency; and it is not uncommon to see it return to the joints which it had abandoned, as if it could not exhaust its action without either attacking in succession a greater or less number of joints, or returning several times to the same joints.

Rheumatism seldom assails any of the articular tissues except the fibro-serous, as is proved by microscopical examination; and it frequently leaves no other anatomical lesion behind it than synovial effusion. This constant tendency of rheumatism to affect the fibro-serous tissue of the joints sometimes causes rheumatism, either primarily or secondarily, to attack the fibro-serous tissue of other organs; and the preference of the disease for that tissue explains the affections called rheumatic metastases.

I shall afterwards recur to these alleged complications of rheumatism. First of all, however, I wish to make some clinical remarks upon the manifestations of rheumatism in the sanguineo-vascular system.

Gentlemen, you are acquainted with the important works of MM. Bertin and Bouillaud upon rheumatic endocarditis and pericarditis. In his treatise on diseases of the heart, and also in his treatise on articular rheumatism, my learned colleague of the Faculty has set himself to demonstrate the law of coincidence of inflammation of the heart with articular rheumatism. According to the celebrated professor of La Charité, in acute, violent, generalized articular rheumatism, *it is the rule* to meet with coincident endocarditis, pericarditis, or endopericarditis. Gentlemen, there is no one more disposed than I am to render justice to the great labors of Professor Bouillaud, yet here I must state that an attentive study of the heart in a certain number of cases of acute articular rheumatism has not enabled me in all of them to discover symptoms of endocarditis, pericarditis, or endopericarditis. In fact, in several rheumatic patients who had acute fever with swollen and painful joints, I could not detect by percussion the dulness caused by effusion, nor by auscultation the friction-sound of pericardial inflammation. A similar remark is applicable to the symptoms of endocarditis; for in a large number of cases of acute multiarticular rheumatism, I have not been able to hear a blowing sound at the apex of the heart: I have more frequently heard a blowing sound at the base, but it was a soft sound, and it extended into the vessels of the neck with the same softness; from which circumstance, and the fact that rheumatic patients are all very anæmic, I am inclined to connect this blowing sound with anæmia. I think then, that the law, as laid down by Bouillaud, in relation to the coincidence of acute cardiac lesions with rheumatism is not so absolute as he has assumed; but while I say this, let me add, that in a great many cases, I have been fortunate enough to be able to verify fully the truth of the law of coincidence so well established by him.*

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* Bouillaud: Traité Clinique du Rheumatisme Articulaire, et de la Loi de Coincidence des Inflammations du Cœur avec cette maladie. Paris, 1840.

Be that as it may, gentlemen, you ought always most carefully to search for the cardiac symptoms which characterize acute lesions of the heart in articular rheumatism; and often, I repeat, you will have an opportunity to confirm by your own observation the law of coincidence laid down by Bouillaud. Recollect, however, that this coincidence may be wanting in acute polyarthritic rheumatism.

The rheumatic diathesis manifests itself upon the fibro-serous tissues of the heart in virtue of the same title by which it attacks the similar fibro-serous tissue of the joints. And, again, in the same way that all the joints are not invaded by rheumatism, it may happen, in a certain number of cases, that the heart remains intact. It would be interesting to study the influence of occupations upon determining the seat of rheumatic affections.

And were it possible, by comparative examination, to prove that the employments which occasion most fatigue to certain joints are those most frequently affected by rheumatism, should we not be justified in admitting a determining local etiology? A similar remark might probable be made in respect of the heart, an organ which is not equally susceptible in all rheumatic persons. Is it not well known that in healthy men the moral emotions and fatigue go to the heart, and that in others, it is protected from every moral and physical disturbance? In the former, the rheumatic diathesis, so to speak, preferentially strikes the heart. In support of these general considerations upon the localizations of the acute rheumatic diathesis, I ought to remind you that in my lectures upon cerebral rheumatism, I have been careful to demonstrate to you that rheumatism attacks the membranes of the brain, particularly in men whose brain had been previously the seat of inflammations proceeding from various causes. In the antecedents of my patients, I have in fact been able to trace the etiology of their cases in their intellectual fatigues, mental distresses, alcoholic excesses, or in certain anterior mental dispositions which had produced a change in the organ of thought. Two of my patients had previously given evidence of insanity, another had been addicted to alcoholic excesses. Some patients had shown a congenital or acquired cardiac idiosyncrasy, and were consequently more disposed than others to endocarditis and pericarditis.

These remarks, however, gentlemen, must be made under reserve, and whatever confirmation they may one day receive from your own observation, do not forget that rheumatism often attacks the heart, and is often the primary cause of the organic lesions to which the complex term of "aneurism of the heart" was formerly applied, and the varieties of which we now describe under the names of contraction and insufficiency of the cardiac orifices.

Valvular lesions of the heart have often assuredly for their primary cause an attack of acute articular rheumatism: clinical observation, however, will teach you that lesions of the mitral and aortic orifices may likewise exist without there being a possibility of discovering in the antecedents of the patients any articular manifestation of the rheumatic diathesis. While it is true that rheumatism often causes diseases of the heart, it must also be admitted that there are cardiac lesions originating in wholly different causes. To mention only one of them, alcoholic intoxication, which determines most remarkable alterations of nutrition in the fibro-serous coverings of the liver and brain, has certainly its part in the etiology of organic affections of the heart. This etiology is, moreover, proved by the coexistence, so frequent in alcoholic drunkards, of lesions of the heart and cirrhosis of the liver.

How then does acute rheumatism act upon the heart? The pericardium is a fibro-serous membrane which completely envelops the heart, and isolates it from the neighboring organs. It consists of two portions: one, the parietal, is free, and constitutes the pericardial pouch: the other, almost wholly serous, is called "visceral," because it covers the heart, and is closely united to it. The serous fold of the parietal and visceral portions is composed of epithelial cells, which lie on the conjunctive tissue. The epithelial layer is non-vascular, and is nourished by infiltration of plasma from the more deeply seated vessels. When rheumatic inflammation attacks the heart, modifications of the functions of nutrition become apparent. The capillary vessels which ramify in the cellular tissue become more numerous: the plasma which they allow to transude through their walls no longer supplies normal epithelial cells, and the cellular tissue becomes infiltrated with new products, and with fat. The serous surface loses its natural smoothness, the membrane itself becomes thickened, and deposits new products on its surface—false membranes—which assume various forms, and may present all the characters of new membranes. The membrane which lines the cavity of the heart is, like the pericardium, composed of two layers, a serous and a fibrous: in it, rheumatic inflammation determines lesions analogous to those which it produces in the pericardium. However, the modifications of nutrition caused by rheumatism are most remarkable on the folds of endocardium which constitute the mitral and aortic valves. Not only does the serous surface of these valves lose its smoothness, but it also often becomes the seat of fibrinous deposits, and of conjunctive cells resembling mulberry-shaped granulations, either scattered or collected in groups. At other times, the thickened valves become the seat of calcareous deposits. These deposits, as has been shown by chemical analysis, are composed of carbonates of lime and soda. These transformations of primary organic tissues are consequences of the transformation of the plasma; and there are found, as you know, analogous transformations on the pleuræ and serous surface of the dura mater.

When the valves have become altered in this manner, they present puckerings of the cardiac orifices which prevent the normal play of the valves. There are other lesions of the valves characterized by atheromata, or by a vascular injection which may lead to actual ulceration. I shall recur to the subject of ulceration in endocarditis at the close of this lecture.

When the joints have been the seat of rheumatic inflammation, they may, after a period of variable duration, recover their functional integrity; but there is no similar recovery after endocarditis: the lesions, be they ever so slight, are irremediable, and usually they become worse and worse. It is necessary, however, to remark that this evil progress may be very slow, leading neither necessarily nor immediately to great disorder of the cardiac function. The thickened valves may retain sufficient flexibility to continue to act as perfect valves, rising and falling regularly in such a way as to afford free passage to the blood and prevent its regurgitation into the cavities of the heart.

Though there be no functional lesion, and though the valves be but slightly thickened, and have lost little of their normal smoothness, an attentive ear can detect blowing sounds, which do not exist when the valvular apparatus is in a state of integrity.

The structural modifications of the valves may remain very slight for many years: at other times, the nutrition of the valves has been so much modified by the rheumatism that the lesion goes on constantly increasing, presenting to the ear rough and rasping blowing sounds. The circulatory function may, nevertheless, not appear seriously impaired, from, simultaneously with the contraction of the cardiac orifices and the insufficiency of the valves, the cardiac muscle redoubling its efforts to overcome the contraction of the orifices, and to struggle against the insufficiency of the valves. We then have *physiological* hypertrophy of the heart. But sooner or later, the efforts of the muscle become exhausted, and are powerless to overcome the constantly increasing obstacles. From this time, the physician detects all the general symptoms of chronic disease of the heart. But do not believe, gentlemen, that the heart quickly abandons the struggle. Clinical observation will inform you, that the disorder in the pulsations of the heart may disappear, and that for a variable period it may recover an energetic power, especially if a critical evacuation by the intestines or (as more frequently occurs) by the kidneys relieves the vascular system from dropsical pressure. We can very frequently prolong the life of the patient by exciting an abundant diuresis, and so restoring some of the power of the cardiac muscle.

In the immense majority of cases, the endocardium is affected after the articulations; but it sometimes happens, quite exceptionally, that the order of events is reversed, and that the law of Bouillaud is verified in an inverse sense; or in other words, that the rheumatism first attacks the endocardium, and then the joints. I had three such cases during the course of the year 1864.

A young man was admitted to St. Agnes Ward with high fever, and great general discomfort, in whom the only local morbid symptom was a somewhat intense blowing sound at the apex of the heart. Had this sound been heard only at the base of the heart, I might have attributed it to anæmia; but as it was, I had no hesitation in regarding it as evidence of endocarditis. Four days after admission, the knees became swollen and painful, and subsequently the shoulders were affected in a similar manner; and finally, the young man had all the symptoms of acute multiarticular rheumatism of average intensity. The patient recovered, but when he left our wards had a blowing sound, the very characteristic roughness of which sufficiently proved both the exactness of the diagnosis as to the anterior acute attack, and the existence of a contracted state of the left auriculo-ventricular opening, with insufficiency of the auriculo-ventricular valve.

The second case was that of a woman, aged 38, who occupied bed 11 of St. Bernard Ward. She had never had rheumatic arthritis at the date of her experiencing, three years ago, palpitation of the heart: she had a small, rather frequent cough, with dyspnœa, but without hæmoptysis. The cardiac disturbance became at length so great, that she resolved, a year ago, to go into Professor Bouillaud's wards in La Charité. At that time, the distinguished Professor detected a cardiac affection, for which he subjected the patient to active treatment. I beg your special attention to this case, because it shows you that the signs of which I have just been speaking have been observed by Bouillaud, and that like me he has attributed them to a lesion of the endocardium.

I shall now describe the affection for which this patient was admitted to my wards. Fifteen days before admission, she—*never before having had pains in the joints*—felt acute pain in the left knee, which became red and swollen; and she was unable to walk. On admission, the knee had ceased to be painful; but there were swelling, redness, and pain in the tarsal articulations, extending along the synovial sheaths of the tendons of the left foot and left thumb.

On examining the heart, I perceived, at the apex, accompanying the first sound, a blowing sound, which was very strong and rasping. This blowing sound was not heard at the base of the heart, and was not continued into the aorta. The pulse was small, and exceedingly irregular. The heart was considerably increased in volume. The liver, also, was very large.

The respiratory sounds were everywhere natural, and, notwithstanding the cough, there was no pulmonary congestion.

The intensity and roughness of the blowing sound, its maximum intensity being at the apex, and the smallness and irregularity of the pulse, concurred to lead me to diagnose an affection of the heart; the view I adopted being, that there was contraction of the left auriculo-ventricular opening, and insufficiency of the mitral valve. The diagnosis, supported by the existence of congestion of the liver, was confirmed by the previous diagnosis of Professor Bouillaud.

I have no doubt that this woman has an organic disease of the heart: that this disease had endocarditis as its starting-point: and that the endocarditis, which was of old date, had been latent: finally, that it was proved to be rheumatic endocarditis by the arthritis which you had the opportunity of observing. The point in this case to which I desire specially to direct your attention is the existence of the cardiac prior to the articular manifestations.

Cases of this kind are perhaps not so unusual as might be supposed. When treating the case now described, I was seeing in consultation a young matron of Brest, then suffering for the first time from subacute articular rheumatism, and who had had previously two attacks of pleurisy. In this young lady, I detected a *superficial* double blowing sound at the base of the heart, and a single rasping blowing sound at the apex. I had no hesitation in attributing the first of these sounds to the friction of old false membranes, the result of a previous attack of pericarditis; and attributing the second, that is to say, the single sound at the apex, accompanying the first sound of the heart, to an organic lesion consequent upon an attack of endocarditis. I considered myself fully justified in attributing to one cause, that is to the rheumatic diathesis, the existing rheumatic attack, both attacks of pleurisy, the pericarditis, and the endocarditis.

Observe then, gentlemen, that *the rheumatic affections of visceral serous membranes may precede rheumatic affections of the articular serous membranes.*

Let me mention a case in which we observed endocarditis without consecutive arthritic manifestations. I refer to the young woman, aged 26, who occupied bed 30, St. Bernard Ward. She was admitted on February 9th, suffering from excessive dyspnœa, which began with shivering, and lasted for two days. The respiration was very frequent, though the pulse was not more than 92. Nothing morbid was appreciable in the lungs; but a rough intense blowing sound was heard at the apex of the heart. There was evidently endocarditis accompanied by dyspnœa, which is not a usual symptom of that disease. Four days later, the pulse was 112, and the respiration 60 in a minute. It was not till eighteen days after admission, that she first complained of acute pain at the apex of the heart, shooting through the whole of the epigastrium. The spinal apophyses of the fourth, fifth, and sixth dorsal vertebræ did not become painful till four days later. This woman during the whole of her sojourn in our wards, had no articular pains, and she left the hospital without any functional disorder, retaining, however, the blowing sound at the apex.

You see, then, that this strong young woman, who had never had articular rheumatism, was suddenly seized, when in a good state of health, with initiatory rigors, such as occur at the onset of the phlegmasiæ. Dyspnœa, attended by fever and unaccompanied by pain in the side, then set in: and this dyspnœa continued for a long time before there was any manifestation of pains. The pain, inflammatory in its nature, became the immediately exciting cause of intercostal neuralgia and epigastralgia. The proof that originally the pain was of purely inflammatory origin is that the painful points over the apophyses, characteristic of neuralgia, did not appear till a period considerably later than the pain in the side. The existence of pericarditis was not only made evident by the *bruit de souffle* at the apex, but also by that sound being rough and persistent after the other symptoms had disappeared. This woman has an organic lesion, which must sooner or later manifest itself by disastrous consequences.

I now wish, gentlemen, to speak to you of a connection which exists between rheumatism and erysipelas. There is not only an analogy between the two diseases which are only seemingly inflammatory, but there is likewise a correlation. Not only have they the same migratory character, but the one may replace the other; as, for example, rheumatism may succeed erysipelas. At present, we have a simultaneous epidemic of erysipelas and rheumatism. A young girl who lay in bed 8, St. Bernard Ward, was suddenly seized by rheumatic pains, when convalescent from severe erysipelas of the face. The patient, aged twenty-two, had already had, according to her own statements, very frequent erysipelatous attacks. The convalescence from the rheumatism did not go on satisfactorily: there was a vague feeling of discomfort, and some fever in the evening. After two days of these undecided symptoms, she had acute pain in the knees, and an exacerbation of fever in the evening. Next day the joints were swollen. Two days later there was heard a *bruit de souffle* at the base of the heart, accompanying the first sound: it was not a soft blowing, due to anæmia, but a rough blowing which was evidently endocardiac. After the knees, the elbows, then the wrists and fingers were seized: in turn, the ankles and toes were attacked. At the present moment, the patient is in a really alarming state: in the evening, her pulse is about 120: her pains are excruciating: her appetite is gone: and during the eleven days which have elapsed since the rheumatism broke out, I have accomplished nothing by the treatment employed.

I cannot resist placing side by side the phenomena which we observe here after erysipelas with those so often seen after *scarlatina*, and (though more seldom) after nodular rheumatism. I have told you how common it is to see acute articular rheumatism, as well as pericarditis and endocarditis, during the convalescence from scarlatina. Now, erysipelas is an affection in which the skin is implicated as in scarlatina; erysipelas, which presents stronger affinities with fevers than with the phlegmasiæ, possesses, like scarlatina and nodular erythema, a tendency to be followed by rheumatism associated with endocarditis.

You no doubt recollect what I said regarding the relations of *chorea* to rheumatism. You know that rheumatism is one of the most powerful predisposing causes of chorea. In the majority of cases adduced in support of this law of correlation, the rheumatism precedes, at a longer or shorter interval, the appearance of the chorea. I have, sometimes, however, been able reciprocally to announce the more or less speedy advent of articular rheumatism in children brought to me with chorea; and the result has justified my unfavorable prediction. Extend the induction to its extreme limits, and consider that since chorea (a rheumatic affection), may precede arthritis of the same nature, it may quite as well, and for the same diathesic reasons, precede or accompany endocarditis. This conclusion, derived from the most legitimate induction, is confirmed by clinical observation. You recently saw in bed 25, St. Bernard Ward, a girl of sixteen with a first attack of chorea. On admission, the chorea was of eight days' duration; but she had had previously about three weeks of general discomfort, feverishness, insomnia, and loss of appetite. Three days afterwards, she experienced slight pain at the heart, unaccompanied, however, by palpitation or dyspnœa. This young woman has never had articular pains. The chorea is of average severity and is most intense on the left side, where there is anæsthesia and great debility. Guided by analogy, I auscultated the heart with much care; and had no difficulty in hearing a rough blowing sound at the apex. This patient, therefore, has had endocarditis at the same time as chorea. The rheumatism in this case, in place of attacking the articulations, attacked the endocardium, the law of correlation being therefore only indirectly confirmed. This case is in harmony with an observation made by Dr. Henri Roger. In his clinical lectures at the Hôpital des Enfans Malades, this judicious observer has recently stated that in at least one-fourth of all the cases of chorea which he has observed, there were heart complications, either with or without rheumatism.

It might appear that as the internal covering of the heart is so frequently affected during a course of acute articular rheumatism, and as the similar tunic of the arteries and veins is identical with it in structure and functions, that they must possess the same morbid affinities, and become also often implicated like the endocardium. Nothing, however, is more different than this from what actually occurs. Arteritis and rheumatic phlebitis are exceedingly rare. Dr. Bouillaud has well remarked that phlebitis may occur in the course of rheumatism; but he adduces no case in support of the statement; and it is easy to see that his statement is based rather on analogy than on observation. We, however, have had an opportunity of seeing some of these exceptional cases; I refer particularly to the patient whom you have seen in bed 16 of St. Agnes Ward.

This man was thirty-six years of age, pale and debilitated. His skin was white and soft. He was in the habit of drinking to excess, particularly brandy. He was admitted to the hospital on 11th March, 1864, with pain and swelling of the right elbow and both knees. This state had existed four days, having been immediately preceded by an attack of intense fever. There was no room for doubt in forming the diagnosis; the malady was articular rheumatism. It was the first time the patient had suffered from it.

The amount of fever was moderate; and there was little in the general state of the patient to occasion anxiety. Both knees, the right elbow, wrist, and shoulder were swollen and very painful. There was neither blowing, nor any other abnormal sound of the heart. Respiration was regular, and the respiratory murmur was heard everywhere.

There was *one* circumstance mentioned by the patient in giving the history of his malady which struck me: he stated, that on the first day of the attack, he experienced acute pain in the calf of the right leg, and on the following day, he similarly suffered in the left. Both calves, in fact, were tense, as if swollen, hard, and very painful on pressure; but the pain was chiefly caused by pressure made in the course of the saphena veins, which could be traced under the skin like two hard cords. The pain was also very acute in the favorite seat of phlegmasia alba dolens; that is to say, in the posterior part of the calf.

There did not appear to be any indications of muscular rheumatism. The idea naturally suggested itself to my mind, that there was inflammation of the deepseated veins of the calf, and that the painful induration of the saphena veins indicated a change in their condition. But as there was no œdema, I resolved to wait before I positively declared the existence of endophlebitis.

Six days afterwards, on the 18th, there was no change in the state of the legs, but the feet were evidently swollen. For five days, the left arm had been swollen, and throughout the whole extent of its internal aspect, it presented the yellow hue of ecchymosis. Pressure over the biceps occasioned great pain; and in the axilla, a hard painful cord could be felt, which was evidently the obliterated axillary vein. It is remarkable that the arm is very greatly swollen, the forearm much less swollen, and the hand hardly at all swollen. There was great effusion into both knee-joints, which were but slightly painful in comparison with the legs. The radio-carpal articulation, which has been affected, is now free.

There was no blowing sound of the heart.

On the day after admission, there was increased tumefaction of the right calf, which at its greatest diameter measured thirty centimetres in circumference. The left calf, which also was swollen, did not measure more than six centimetres. The superficial veins were attacked in turn: they became painful to the touch, and lost their elasticity. There was great improvement in the arms; but the left arm continued to present large ecchymotic stains.

There was neither fever, heat of skin, nor sweating. The most careful auscultation did not enable me to detect any abnormal cardiac sound.

It is, therefore, evident, that this rheumatic patient has not endocarditis, but that he has numerous phlebitic affections; that is to say, that the diathesis in place of striking the endocardium has attacked the internal coat of the veins, constituting endophlebitis.

On the 19th, the patient suffered from headache and muscæ volitantes. The pain, which was localized in the median line of the head, in the course of the superior longitudinal sinus, led me to suspect inflammation with consecutive obliteration of that venous canal, and to be on the outlook for the development of formidable cerebral symptoms. However, the headache and visual phenomena, after continuing three days, disappeared without my anticipations being realized.

On the 21st, both femoral veins, and both brachial veins from the elbow to the axilla, were completely indurated, without being more than very slightly painful. In the inferior extremities, the branches of the saphena vein, and that vein itself, were hard and painful. Thus, the principal venous trunks, and some of the superficial veins of the four limbs were obliterated, the result being œdema of the four limbs. On the 22d, there was detected painful obliteration with superficial redness of the external aspect of the inferior third of the left forearm. Between the 23d and 26th, the superficial veins of the forearm became implicated in succession; and the superficial veins of the legs became similarly affected. With a view to study with more precision the progress of the phlebitis, Dr. Peter, my *chef de clinique*, adopted the following measures: with nitrate of silver, he traced the course of the superficial veins, which were distinctly red, so that on the following days the changes which had taken place might be seen. When the experiment was made, these veins, which were red in color and painful to the touch, were still permeable, and could be emptied by pressure. Two days after the experiment, they were hard and considerably prominent, could no longer be emptied by pressure, were less red, although they still remained nearly as painful as before. The redness and pain, therefore, were shown to have preceded the obliteration of the vessels: consequently, the inflammation of the veins preceded the coagulation of the blood. Consequently also, phlebitis existed, not radiating phlebitis caused by the contact of the coagulum, but phlebitis which had, on the contrary, determined the formation of the coagulum.

I shall not dwell upon the alternations of increase and diminution of the œdema, arising from the greater or less impediment to the venous circulation: I prefer to direct your attention to a new phenomenon, one of most unusual occurrence, viz., the appearance on the 30th March, the twenty-third day of the disease, of petechiæ in every part of the left thigh. There was at that time increased pain with loss of mobility in the limb, its temperature being lowered instead of being augmented: the fever, though moderate in degree, had returned, the pulse, which was very small, ranging between 92 in the morning to 100 in the evening. The patient groaned incessantly; and his general condition seemed very bad.

On the following day, the œdema had gained the scrotum and lower part of the abdomen. Next day, the inferior part of the chest was œdematous; and the internal mammary vein was painful throughout its entire course.

On the 3d April, the twenty-seventh day of the disease, the fever had ceased: but there was increased œdema of the abdomen and chest. On that day, a large ecchymosis appeared on the dorsal surface of the left foot, which was enormously swollen. On the succeeding days, phlyctenæ appeared in the situation of the ecchymosis, and a petechial eruption accompanied by acute pain became visible on the knee and right thigh: at this time, nearly the whole of the lower extremities were covered by petechiæ.

In the latter days of April, the whole of the skin of the dorsal surface of the left foot was sphacelated, the corresponding skin of the right foot being sphacelated in part. The skin of the calves of both legs was extensively ulcerated, as was likewise the skin of the scrotum and prepuce.

However, as I pointed out to the students who followed the daily visit, the indurated radial and ulnar veins were gradually diminishing in volume, and a similar change was taking place in most of the superficial veins of the lower extremities. I predicted a speedy return of circulation through these veins, and said that it would take place before the large venous trunks became permeable. The event verified my prediction. A long time elapsed before the large venous trunks became permeable. At the end of June, four months from the commencement of the affection, the axillary veins were still indurated; and although they had been free from pain for a long time, there was no ground for supposing that the circulation had been resumed in them. It was not till the middle of July, that the large venous trunks had ceased to be obliterated.

The loss of substance in the feet and legs degenerated into ulceration of a bad character. The ulcers were sordid, bled on the slightest contact, and did not cicatrize till the middle of September. Even at that period, six months after the phlebitis commenced, the ulcerations on the calf of the left leg were not quite cicatrized.

It would have been difficult to have seen a more anæmic patient; but all the functions were pretty naturally performed. The fever had disappeared about the end of the first month of the illness, the functions of the heart were unaffected, respiration was good, the appetite sufficiently keen, and digestion well performed. The patient had neither albuminuria nor diarrhœa. His resisting the unfavorable impression of the serious local symptoms must be imputed to the plastic functions not having been impaired. He left the hospital at the middle of September with persistent œdema of the calf of the left leg, and an imperfectly cicatrized ulcer of the same limb. I believed that residence for some time at Vincennes would hasten recovery which it would be difficult to bring about in an hospital.

I particularly insisted upon part of the treatment consisting in the administration of sulphate of quinine and tonics. I had for a long time kept the patient upon good fare. He had been taking the diuretic wine for a long time.

If we now analyze the series of morbid changes which took place in this patient, we shall find that the rheumatic affection almost simultaneously attacked the joints and the internal coat of the veins, respecting the endocardium; so that we had endophlebitis in place of endocarditis. This is a fundamental fact which is rather unusual in its character, although in respect of this particular instance the histological well explains the morbid analogy.

Phlebitis, as usual, caused obliteration; but the obliteration, which was on an extensive scale, led to an unlooked-for result, the production of petechiæ and ecchymoses, and to sphacelus, which under such conditions is an unusual occurrence.

You are aware that articular rheumatism is the morbid state in which the blood contains the greatest proportion of fibrin; and you also know that that is a condition of the blood which certainly does not predispose to hemorrhage. May it not be then that in this disease petechiæ and ecchymoses greatly depend on the numerous obliterations of veins, the blood (notwithstanding these obliterations) flowing through the capillaries so as to distend and ultimately rupture them—may not this be the explanation of the petechiæ and ecchymoses? The arterial blood having passed into the capillaries, and being unable to leave them by the veins, the necessary result was rupture. This is the explanation of the occurrence of hemorrhages accompanied by pain, at those places where there had been the greatest amount of œdema.

In respect of sphacelus—so seldom met with in venous obliteration—it was evidently the result of the great distension of tissues; and also probably, of the disturbance of interstitial nutrition at those points where the venous circulation was almost entirely suspended by obliteration of the deep and superficial veins.

The progress of the multiple phlebitis presented peculiarities which it is important to point out. For example, the inflammation proceeded from the large venous trunks to the branches. It was the deep veins of the arms and calves of the legs which first became painful, while at the same time œdema was observed to proceed towards the distal extremity of the member. Resolution, on the contrary, and unblocking, proceeded from the branches towards the trunks. We had opportunities of seeing the superficial indurated veins diminish in volume little by little, and then become permeable, the collateral circulation being thus slowly re-established. The symptomatic œdema disappeared much more rapidly in the upper than in the lower extremities; and it left the thigh long before it disappeared from the calf and the foot. Even when the patient left the hospital, the calf and foot retained a diffuse puffiness.

For more than a month, the circulation was almost completely suspended in the four members, and we could imagine how the interstitial nutrition must have been interfered with by the very fact of the arrest of local circulation. During the whole of this period, the patient had in a permeable state scarcely more than the large venous trunks of the abdomen and head; and in respect of the venous circulation, the four extremities were as if they had been amputated. For a short time, I was afraid that the cranial sinuses would in their turn become implicated: when the patient was suffering from pain in the course of the sagittal suture, I was even under the impression that the superior longitudinal sinus was obliterated: but it turned out, happily for the patient, that his sinuses were not implicated similarly to the veins of his limbs; for it is easy to understand the serious consequences to the brain which must have followed such an implication. It is remarkable that this patient, notwithstanding the existence of such causes of embolism, had no pulmonary complication.

In terminating the history of this case, I wish only briefly to discuss the two hypotheses which present themselves in relation to entire venous obliteration. Was the obliteration primitive, and did the coagulation of the blood determine the irritation of the vein? Or, was the obliteration of the vein consecutive to its irritation, and did it cause the coagulation of the blood? In favor of the first hypothesis, we may urge that in rheumatism there is a great augmentation in the quantity of fibrin, and allege that this hyperinosis would sufficiently account for the spontaneous coagulation of the blood at the most distant parts of the circulation, where the current is slowest, that is to say, in the limbs. But the very simple experiment made by my *chef de clinique* demonstrated that the redness and pain in the veins preceded their obliteration; and consequently, that the affection commenced in the parietes of the veins, so that the coagulation of the blood was consecutive to the venous affection. It is, therefore, very evident that our patient has had attacks of phlebitis, and that these attacks were *rheumatic:* indeed, the number of veins affected is a sufficient proof of the existence of a diathesis assailing not one isolated point, but the entire venous system. There is only one general affection which can produce lesions so extensive and so symmetrical.

Having said this much regarding the clinical peculiarities of acute articular rheumatism, let me now return to its most usual complications.

Acute rheumatism produces, we have seen, diseases of the heart: it is, therefore incumbent on the practitioner to prevent the continuance of the rheumatic inflammation of the serous membrane of the heart. Revulsive treatment is assuredly the best means which we possess of accomplishing this object. In conjunction with it, we must direct the patients to use all hygienical precautions likely to prevent the rheumatism abandoning the articulations. We must prescribe scarifying cupping over the region of the heart, or better still, the application of irritants to the skin, such as sinapisms and blisters, which will diminish fluxion towards the heart. I have little liking for therapeutical theories, and have no confidence in the theory of revulsion; but I believe, in accordance with the statements of all clinical observers, that the application of revulsives to the region of the heart first calms and then often puts an end to pains and palpitations so as to enable the heart to regain the regularity of its pulsation. You must, however, at the same time carry out the general treatment of the disease, which varies according to the prevailing medical constitution, and the individual's constitution; and which will demand, it may be, bleeding, purging, alkalies in large doses, sulphate of quinine, aconite, or digitalis.

How can such different medications tend to the same end, the cure of the patient? So distinctively characterized a disease as rheumatism ought to have only one remedy; but no such specific is within our reach. When a man has grown old in the practice of the art of medicine, he accepts, with Sydenham, Boerhaave, and Stoll, the fact that acute rheumatism is a special pyrexia requiring for its development the existence of certain individual and atmospheric conditions. It presents many characteristics. It only occurs in certain individuals, and in certain families: the fibrous tissue is its favorite seat: it imparts a peculiar aspect to the person it attacks. No clinical observer can deny that it is a disease which possesses the inflammatory element; chemical analysis shows us that the blood is charged with fibrin; but it also contains another morbid element, with the nature of which we are not acquainted, and the existence of which is revealed to us only by the constancy of its manifestations. Again, these manifestations are generally transient, and usually leave no trace, *except on the heart.* Rheumatism is essentially migratory: though it constantly attacks the fibro-serous tissue of different organs, it leaves one articulation to seize upon another, and often returns to that which was first stricken. Moreover, it may assail organs the functions of which cannot be disturbed for a certain time without causing death; and necroscopic examination discloses no lesion of these organs adequate to explain the fatal issue. The old physicians, by whom rheumatism was well studied, perceived in it a fluid which pervaded the whole organism, and assumed the right of temporary domicile in different organs.

They observed that this was a precise indication: and, in relation to this point, it is very interesting to read Van Swieten's commentary on aphorism 1493 of his master, Boerhaave. This able observer regarded it as essential to eliminate, at any price, the morbid matter; and that as it circulated in the humors, it was sometimes necessary to open a vein to afford a free and abundant exit to the sanguineous fluid: by this proceeding, a great part of the rheumatic matter was supposed to be discharged. But Van Swieten recommends that this means of treatment should not be abused, as it cannot be long employed without incurring a risk of syncopic attacks and convulsions. Purgatives and sudorifics were also recommended by the illustrious Professor of the University of Vienna, with a view to the elimination of the morbific matter by a natural emunctory.

This morbific matter, circulating in the normal humors of the economy, may be deposited in the viscera after having left the joints. Hence originates the theory of metastases, that is to say of change of situation in the matter which is producing the disease in any part of the organism. Likewise when the old physicians saw a coincidence between the disappearance of the articular pain, and the patients complaining of dyspnœa, oppression at the chest, and cough, they unhesitatingly affirmed that the rheumatic matter had been conveyed to the lungs and pleuræ. Van Swieten informs us that, under similar circumstances, it has often happened that the autopsy has revealed dropsy of the chest and pulmonary engorgement. The same clinical remark was made in respect of cerebral symptoms supervening in the course of rheumatism; and in these cases, there has been recognized serous effusion around the brain and in its ventricular cavities.

I am, therefore, gentlemen, justified in saying that, towards the middle of last century, rheumatism was profoundly studied in its different manifestations: also, that the works published in later years on visceral rheumatism, particularly on cerebral rheumatism, have had their own share of merit, by recalling the attention of the medical world to the secondary manifestations of rheumatism.

It sometimes happens, gentlemen, that the viscera are primarily invaded by rheumatism. There is, as you know, a form of pneumonia called *rheumatic pneumonia*, which sometimes occurs with all the physical and rational signs of inflammatory pneumonia—the stitch in the side, cough, difficult breathing, bloody expectoration, dulness, râles, and blowing, in fact, every symptom and every physical sign. But that which gives this kind of pneumonia its distinctive character, and makes it a species, is that all the symptoms of pneumonia may suddenly disappear without the gradual decrease observed in inflammatory pneumonia. At other times, the patients have a stitch in the side, oppression, crepitant and subcrepitant râles, and blowing, while the expectoration is merely viscous or catarrhal, and presents no trace of blood. The rheumatic disease may remain confined to the lung; but it is not unusual for articular pains to occur on the cessation of the symptoms of pneumonia.

Again: the membranes of the brain and spinal marrow may be the primary seat of rheumatism. When lecturing on cerebral rheumatism, I brought before you several cases in which rheumatism primarily invaded the coverings of the cerebro-spinal axis. Recall to your recollection the case of the patient who was admitted to St. Agnes Ward with fever, and lumbar pains so violent, as to lead me to believe, in the first instance, that I had to do with the rachialgia of the invasion stage of small-pox. On the morrow of my first examination, I detected a rheumatic inflammation of one of the knees, while the patient at the same time stated that he no longer suffered in the lumbar region. Recall, also, the case of the female attendant of St. Bernard Ward, who, after having been subject to rheumatism for many years, was at a later period seized with rachialgia and incomplete paraplegia, then with pains in the head accompanied by visual disturbance and feelings of general discomfort, followed by the sudden supervention of articular pains, and the disappearance of all the cerebral and spinal symptoms. I cannot, gentlemen, too strongly urge upon you to imprint these facts upon your memory, because they will unquestionably assist you in the diagnosis of rheumatic manifestations, and place you on your guard against adopting therapeutical proceedings which are often useless when they are not injurious.

I conclude, gentlemen, these general considerations regarding acute rheumatism by remarking, that it very seldom passes into a chronic state, except when it has attacked only one joint. It seems as if it then exhausted all its morbid action on a single articulation, and by its intensity and continuance in one situation made up for that which it lacked in the extent of parts affected. Be that as it may, in such cases, the rheumatism causes chronic arthritis with alteration of the synovial membrane, cartilages, and osseous tissue: the local affection, termed *white swelling*, is frequently the consequence of similar localizations of the rheumatic principle. In our clinical wards, you have had an opportunity of studying these very painful chronic arthritic affections which have their seat by preference in the knee and wrist: you have also seen in these cases the advantage which the patients derive from poultices of camphorated bread-crumb with the addition of belladonna and opium. These poultices must not be renewed more than once in eight or ten days. Under the influence of absolute immobility, of temperature, and of the absence of pain, I have almost always obtained a cure of these chronic rheumatic arthritic affections.

Though acute multiarticular rheumatism seldom becomes chronic, it is subject to relapses. When you see a patient suffering from rheumatism, you ought to discover whether he has not had previously some acute rheumatic affection: it is important to ascertain whether there has been a previous attack. You ought to tell the patients that they have reason to fear new attacks of rheumatism; and must consequently avoid all determining causes of the disease.

There is perhaps no acute disease which so rapidly induces anæmia as rheumatism. The extreme pallor, the vascular blowing, and the hydræmic fulness of the pulse declare the existence of a state of acute cachexia quite independent of the medical treatment which has been employed. But though the blood loses a notable quantity of its red globules, it acquires a large amount of fibrin during the acute stage of the disease. I am not prepared to say that the excess of fibrin increases the plasticity of the blood; but the fibrinous productions deposited on the valves show that there is a great tendency to coagulation. Are we to believe that this excess of fibrin in the serum of the blood is the consequence of the supposed inflammatory element in rheumatism? It seems useless to discuss this question, wherefore I prefer simply to state the fact that along with the acute cachectic state of the patients there is very great fibrination of the blood. The crasis of blood is so much modified by acute rheumatism that months are required before the patients entirely recover their health.

I need not dwell at much length upon the treatment of acute articular rheumatism. Clinical experience has taught me that the wisest plan is not to employ a plan of treatment determined upon beforehand. The fever, pain, and inflammatory turgescence are, to a certain extent, necessary to enable the patient to come well out of the attack. Therefore we ought, generally, to do no more than try to imitate nature, that is to say, try to maintain the rheumatic sweating, ease the articular pains, and above all, avoid promoting visceral metastases by adopting improper treatment.

I do not at all believe that the sulphate of quinine has ever caused cerebral rheumatism. I know very well that metastasis to the brain may take place in cases in which sulphate of quinine has never been prescribed. Nevertheless, I consider it prudent, particularly when the antecedents or habits of the patients lead us to fear that there is some cerebral predisposition, to avoid the use of any medicine which can cause congestion of the encephalon, and consequent inflammation of the brain.

In England, for some years past, influenced by the views of Dr. Garrod, several physicians have successfully employed the bicarbonate of soda in doses of from fifteen to thirty-five grammes. This treatment according to the statements of the same physicians moderates the fever, and lessens the inflammatory turgescence without determining visceral inflammation, and likewise shortens the duration of the attack. Let us hope, gentlemen, that farther observation will confirm these important statements. But when I remember how easily the Vichy waters induce anæmia in healthy persons, I cannot help fearing that the bicarbonate of soda will increase the anæmia of rheumatic subjects.

There is nothing calculated to carry conviction to the minds of practitioners in the writings which advocate the treatment of rheumatism by large doses of sulphate of quinine, nitrate of potash, and antimonials. In most of the reported cases, a sufficiently precise account has not been taken of the onset of the disease, its intensity, its duration, and its relapses, to justify the responsibility of my advising you on this subject. Gentlemen, as you are entitled to receive from me the result of my experience, I confess to you that I do not regard any one method of treatment as absolutely preferable to all others. The practitioner must mark attentively the progress of the disease, and be in no haste to interpret the performances of nature. Rheumatism has a cause, the essential nature of which is concealed from us. Generally, the disease declares itself in several articulations, and visceral complications seldom occur. Our duty, therefore, is not to interfere very actively, at least not till the indications of treatment are very exact.

I have already described the plan I pursue in cardiac complications. When cerebral disturbance leads us to fear that there is localization of the rheumatism in the membranes of the brain, the gravity of that complication justifies our daring much; and in such cases, too, we must not too long delay interference. While by employing revulsives, we try to bring back the inflammation to the joints which were first attacked, we must employ saline purgatives so as to produce copious stools by subtracting from the blood a great part of its fluid constituents, thereby at the same time causing the discharge of a certain quantity of the morbific matter. I cannot confidently affirm that this kind of intervention will prove beneficial, but I can say with certainty that it will not produce any serious consequences to the patient; and this treatment has the advantage of not introducing into the organism any new element unknown to us in its mode of action.

When convalescence has once been established, never neglect to recommend great hygienical precautions; and be specially careful to get the patients scrupulously to avoid all causes which induce chills. Regarding rheumatic anæmia, I may remark that it hardly calls for any special treatment. Good food and the open air will restore to the blood its deficient red globules; and the excess of fibrin which has been observed will soon disappear.

It still remains for me, gentlemen, to speak of *ulcerating endocarditis.* That term does not imply a new disease, occurring as a complication of acute articular rheumatism, or independent of the general disease. But the attention of the best observers is sometimes at fault, as I remarked when lecturing to you on exophthalmic goitre, locomotor ataxia, labio-glosso-laryngeal paralysis, and progressive muscular atrophy. Ulcerating endocarditis has evidently been always liable to occur; but it required an altogether fortuitous concurrence of circumstances for a lesion which perhaps had already been remarked without much importance having been attached to it, to become the subject of that special study, which was soon so prolific in results.

Prior to Bouillaud's discovery of the law of the coincidence of acute diseases of the heart with acute articular rheumatism, there has sometimes been observed functional disturbance of the heart during a rheumatic attack. In these cases, the patients may complain of palpitation, dyspnœa, and pain in the region of the heart. In the Letters of Morgagni and the *Sepulcretum* of Bonet, you will find indicated and described alterations of the pericardium and cardiac valves in patients who had died during the progress of acute articular rheumatism. All the clinical and anatomical facts were seen, but their relationship was not recognized, so that the observation of the facts remained a dead letter. The glory of discovering and demonstrating this relationship belongs to Bouillaud.

Nor is this all: my accomplished colleague having observed that the patients die with typhoid symptoms, the consequence of gangrenous endocarditis, called attention to the fact, and so led the way to a new discovery.* Dr. Bouillaud was led to this view by a number of cases published in his Traité des Maladies du Cœur, particularly by the cases communicated to him by Saussier, Gigon (d'Angoulême), and Rivière.

Dr. Stenhouse Kirkes was, I believe, the first who followed in the path opened by Bouillaud: in an able work, he treats in a very remarkable manner many of the consequences of valvular alterations of the heart. Though Kirkes has not described ulcerating endocarditis, he has shown by his cases the nature of the principal consequences of the migration of fibrinous concretions formed in the heart, and mingling with the blood. At the date to which I refer, Kirkes had recognized the possible consequences of the migration of valvular concretions to the peripheric and pulmonic circulation. In the same patients, he likewise observed, on the one hand, softening of the brain with sudden hemiplegia, aphasia, and obliteration of the middle cerebral artery; and on the other hand, infarctus of the spleen and kidney coincident with obliteration of the large vessels of these organs and small ecchymoses of the skin and mucous membranes, the result of sanguineous effusion, having in their centre a small spot of a yellow or chamois color caused by obliteration of the capillaries. On the other hand, Kirkes had observed that lesions of the tricuspid valves produced serious alterations in the parenchyma of the lungs. Finally, he perceived that the broken-up fibrin might cause an infection of the blood, manifesting its presence by typhoid symptoms.

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* Bouillaud: Traité Clinique des Maladies du Cœur. Paris, 1841.

Dr. Kirkes looked upon all these lesions as similar in their nature, and similar in their origin; viz., the obliteration of an artery by a fragment of fibrin detached from the heart, if the fragment were large, the obliterated artery was proportionately considerable—the middle cerebral, renal, or splenic, for example—softening, or some serious morbid alteration of the organ, being the final consequence: if the fragments were of medium size, there were only ecchymotic spots at the points where they had been stopped: finally, he said that the disintegrated fibrin, by its admixture with the blood, could give rise to adulteration of the blood sufficient to induce typhoid symptoms. So much for the left side of the heart. Again, should the fibrinous mass be detached from the tricuspid valve, it is in the cycle of the right side of the heart that untoward symptoms are observed—obliteration of the pulmonary artery and consecutive lesions of the lungs. Thus we see that almost to the very name, Stenhouse Kirkes had discovered embolism, and though he does not make special mention of ulcerating endocarditis, he had the great merit of calling attention to the functional disturbances induced by it, and even indicating the pathogenic filiation of the typhoid symptoms to which it gives rise.[*]

It appears then that although Bouillaud had called attention to gangrenous endocarditis and its consequences, it was certainly Stenhouse Kirkes who discovered the vascular obliterations, the consequent local lesions, the diffusion in the blood of disintegrated fibrin, and the general disturbance of the economy. After him, I must name Virchow, Bamberger,[†] and Friedreich,[‡] as well as the French physicians, Charcot, Vulpian,[§] and Lancereaux.[||] It was in 1861, that MM. Charcot and Vulpian, in an admirable work, made known generally to the profession the doctrines of the German authors, and demonstrated the inflammatory vascularity of the aortic valves, and elucidated all the effects produced on them by inflammation. Subsequently, numerous cases of ulcerating endocarditis have been reported from the Parisian hospitals.

Inflammation of the valves may depend upon a previous inflammation, or may supervene without the pre-existence of any appreciable inflammation: it has consequently different modes of pathogenesis. Generally, nothing more than hyperæmia of the valves can be detected: there exists only a loss of substance, variable in extent of surface and in depth, velvety, and having sometimes irregular edges, free, or covered with fibrinous vegetations. At the bottom of the ulceration, there are new plastic productions, consisting of pyoid corpuscles, granular bodies, and cells of conjunctive tissue. Sometimes, the valve is perforated: at other times, the ulceration extends to the free margin of the valves, the edges being checkered by small thready or laminated prolongations.

The ulcerations are generally situated in the mitral and aortic valves: they are, however, also met with in the tricuspid and pulmonary valves.

When there is an abundant purulent secretion from the valves of the left side of the heart, it is obvious that purulent infection may ensue: on the other hand, in those cases in which the exudation consists exclusively of conjunctive tissue, of fatty and fibrinous bodies, the exudation may occasion capillary embolism and consecutive purulent infection. It is, therefore, necessary to draw a distinction, both from a pathogenic and clinical point of view, between the sanguineous mixture of the purulent product of an ulcerous endocarditis and the fibrinous disintegration of an exudation either formed or in process of formation. An attempt has been made to establish this distinction anatomically: it has been said, for example, that the fibrin is soluble by alkalies and acids, but that the cellules of conjunctive tissue and the plastic corpuscles resist these agents. In fact, embolia only give rise to local phenomena, provided the obliterating body has not itself undergone a previous pathological alteration. When the valve is in a state of hyperemia, it may be granted that the inflammatory process has been the cause of the ulceration; but in those cases in which there has been no appreciable capillary injection, there is reason to suppose that the valve has been the seat of an atheromatous softening, which has become disintegrated, or that an alteration of nutrition analogous to gangrene has taken place in the tissues of the valve. Be that as it may, we are almost always able to detect small fibrinous granulations which are either isolated or agglomerated.

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[*] Bouillaud: Traité Clinique des Maladies du Cœur, t. ii, p. 308. Paris, 1841. Kirkes: Archives de Médecine for March, 1853.

[†] Bamberger, H.: Lehrbuch der Krankheiten des Herzens. Wien, 1857.

[‡] Friedreich: Krankheiten des Herzens. Erlangen, 1861; 2d edition; 1867.

[§] Charcot et Vulpian: Note sur l'Endocardite Ulcéreuse Aigue à forme Typhoide. [*Mémoires de la Société de Biologie*, 1861. Paris, 1862, p. 265.]

[||] Lancereaux, E.: Recherches Cliniques et Anatomo-pathologiques pour servir à l'histoire de l'Endocardite Suppurée, et de l'Endocardite Ulcéreuse. Paris, 1863.

It was necessary, gentlemen, before speaking of the symptoms of ulceration of the valves, to describe to you the anatomical lesion. It generally happens that in the course of a rheumatic affection, sometimes also in the latter months of pregnancy, or a few weeks after delivery, or at other times as the sequel of a cachectic state, or of an old or recent heart affection, or finally, under circumstances depending upon any decidedly marked morbid condition, the symptoms declare themselves by a single fit of shivering, the patients complaining of general discomfort, articular pains, and feverishness. During the first days of the attack, there is loss of appetite, accompanied sometimes by nausea and pain at the epigastrium. Diarrhœa and tympanitic distension of the abdomen have also been observed. The prostrate condition of the patient and the abdominal symptoms may lead to the supposition that the case is one of typhoid fever in its initiatory stage.

There are other cases, in which the patients, after some days of discomfort, are suddenly seized with shivering, frequent attacks of intractable vomiting, and profuse diarrhœa. One case is recorded in which there occurred cramps, coldness of the limbs, alteration of the countenance, and extinction of the voice. There is also observed, as in certain cases of cholerine, a period of reaction; and the symptoms of gastro-intestinal disturbance may disappear; then, cerebral symptoms supervene, the patient falls into a comatose state, and death follows about eight, ten, or fifteen days from the date at which the first symptoms of the malady showed themselves.

In several cases, jaundice of a more or less decided hue has been observed to occur in the course of the disease. Phenomena akin to the initiatory symptoms of typhoid fever may have led to the belief that the case was one of severe jaundice, a conclusion all the more probable, from the presence of petechiæ and extensive ecchymoses.

It appears, therefore, that, under various circumstances, the patients are seized with feelings of general discomfort, with symptoms similar to those which indicate blood-poisoning, with a shivering fit (which may recur in an irregular manner), prostration, diminution of the vital powers, intestinal and hepatic functional disturbance. These symptoms may lead to the conclusion that the case is one of adynamic typhoid fever, cholerine, or severe jaundice.

But a slight auscultation of the heart is sufficient at once to testify that that organ presents modifications in its rhythm, in its pulsations, and in the intensity of its sounds, bearing no relation to those usually met with in typhoid fever, cholerine, or aggravated jaundice. We will find the first or the second normal sound of the heart replaced by a blowing sound, attributable only to the existence of valvular lesion. Now, autopsies have shown that ulcerating endocarditis may determine lesions of the mitral and aortic valves, and that these lesions may cause stricture of the orifices, or insufficiency of the valves. Finally, it may happen that the fibrous deposits which cover or encircle the valvular lesions may be disposed in such a way that the sounds may temporarily cease to be audible, or that only one of the two morbid sounds may remain.

Be that as it may, a daily examination of the heart made with the requisite care, will soon remove all doubts as to the existence of a cardiac lesion. Hence it follows, that it is necessary to inquire as to the part played by this lesion in the production of the local and general phenomena which are observed.

We see then that the typhoid symptoms are dependent upon general purulent infection produced by an admixture of the blood with organic matters detached from the ulcerated valves. These organic matters will also produce effects by causing obliterations of arteries: they will determine vascular obstructions, infarctus of the spleen or kidneys, small capillary obliterations, or small intestinal, cutaneous, or mucous hemorrhages—in the centre of which obstructions yellow fibrin will be found. Finally, it is probable that the organic detritus, acting as foreign bodies, may determine small miliary abscesses.

Metastatic abscesses resulting from purulent infection have a preference for particular organs: and perhaps further observation will show that capillary embolia occur most frequently in determinate parenchymata. It is known that infarctus occurs most frequently in the spleen, next most frequently in the kidney, and that it is quite exceptional to meet with it in the liver. Does the rarity of infarctus in the liver depend upon the manner in which the hepatic artery arises from the cœliac trunk? This is possible: but, in any case, although fibrinous vegetations of a certain size have no tendency to become impacted in the hepatic artery, this exemption does not extend to molecular detritus, which being suspended in the blood, can circulate as easily as the red globules. This circumstance explains how it is that disturbance of the hepatic functions and jaundice are produced by the presence of vitiated blood through the liver.

The numerous ecchymoses observed on the mucous membrane of the gastro-intestinal canal, and on that of the air-passages, as well as in the pleuræ and pulmonary parenchyma, are sufficient to explain, to a great extent, the various disorders observed in these organs, in connection with ulcerating endocarditis.

Gentlemen, I ask you to note that in some cases of ulcerating endocarditis the cerebral arteries have contained embolia, and that this has led to paralytic hemiplegia.

From these anatomical and clinical facts, then, it appears, that in the cases in which there occur sudden signs of obliteration of the arteries, of ecchymoses, of jaundice, and symptoms of an alteration in the constitution of the blood, we must very carefully inquire whether there be not some lesion of the valves of the heart.

I regret that I have not collected cases from my clinical wards in support of the preceding remarks: but let me fill up this gap by giving you the principal facts of the case which forms the subject of the memoir of MM. Charcot and Vulpian, and of another case recorded by Dr. Chalvet, and explained in a remarkable manner by Dr. Lancereau.

The case of ulcerating endocarditis for which we are indebted to MM. Charcot and Vulpian, has this remarkable peculiarity, that the valvular lesion was situated in the right side of the heart, and that it gave rise to local morbid changes limited to the lungs, and to general typhoid symptoms.

The following is an abstract of this case:

A man, aged 30, a plumber by trade, who had always enjoyed perfect health, after having been subjected to great fatigue, was suddenly seized with violent shivering, headache, and acute lumbar pains. Five days later, he applied for admission to the Hôtel-Dieu, where the existence was ascertained of most of the symptoms of typhoid fever: there was high fever, the tongue was covered with a saburral coat, there was extreme prostration, some headache, and a little cough. It is noteworthy that this patient had recently attended upon his wife whilst she passed through an attack of typhoid fever.

On the seventh and eighth day of the disease, the same symptoms existed; and in addition there was one rose-colored lenticular spot on the abdomen, which was very decidedly tympanitic. The tongue was dry and cracked, and the mouth pasty; there was constant thirst, and frequent vomiting. The pulse was full and rapid. Over the middle region of the heart there was heard a blowing sound, between the two normal sounds; it was different from the blowing sound perceived at the base on the day of admission. Respiration was difficult; and crepitant râles were heard in both lungs.

On the ninth day, the same typhoid symptoms existed; and there was also an inability to urinate. The urine drawn off by the catheter contained no albumen; it had been found in the urine on the fifth day of the disease. There was no diarrhœa similar to that which is met with in dothinenteria. The subcutaneous veins of the upper extremity were greatly distended. The pulse was rapid, full, and bounding. Both sounds of the heart were accompanied by a blowing sound.

On the tenth day, there was stupor, and a pale countenance. There was sweating from the whole cutaneous surface. No thoracic nor precordial pains existed. There was strong pulsation in the jugular veins; and great distension of the veins both of the superior and inferior extremities. The pulse retained the frequency and fulness it presented on the previous days. Auscultation of the heart gave positive indications of extensive valvular lesion.

On the eleventh day, there were alternations of quiet and agitation, with slight delirium. The stools were liquid and copious. The countenance was pale, and indicated stupor. The profuse sweating continued. Subcrepitant vibrant râles were heard. Posteriorly, throughout the whole extent of both lungs, there were subcrepitant and vibrant râles, without blowing sound, and without dulness. The tympanitic distension of the abdomen continued. There were no rose-colored lenticular spots. Pressure of the abdomen did not give rise to any pain. The articulations presented no structural changes. The urine (obtained by catheterism) showed no traces of albumen.

On the twelfth day, the patient had had several liquid stools. During a great part of the night, he had delirium. The countenance had a weary aspect. Questions were answered slowly but correctly. The patient said that he felt very well. The pulse was feeble. Auscultation continued to give the same cardiac and pulmonary signs.

On the evening of the thirteenth day, he died. Death was not preceded by convulsions. To the very last, the patient retained his consciousness.*

What conclusions are we to draw from this clinical history? A man, who considered that he was in good health, was suddenly seized with shivering, after experiencing moral and physical depression. The aggregate of the general symptoms soon justified typhoid fever as the diagnosis. On the seventh day, a single rose-colored lenticular spot appeared. It must, however, be remarked that there never was any epistaxis; and that although the tongue and stomach afforded signs of intestinal lesion, the patient had not had during the early days of his illness the frequent liquid stools usually observed in typhoid fever.

After the eighth day, had the case been one of typhoid fever, the general symptoms, particularly the intestinal symptoms, would have become more decided; but up to the thirteenth day, these symptoms remained stationary. On the eighth day, auscultation revealed the existence of an organic lesion of the heart; and at the same time, râles louder and more diffused were heard throughout a great extent of the chest. The tension, moreover, of the peripheric venous system, and the persistence of pulsation in the jugular veins, scarcely left any room to doubt the existence of valvular lesion of the right side of the heart. Finally, the absence of that aggravation of symptoms usually observed in the second seven days of dothinenteria indicated that the case was one rather of a typhoid than dothinenteric character. That was the view entertained by MM. Charcot and Vulpian: and the sudden manner in which the cardiac affection began, and the rapidity of its progress led them to believe—under all reserve, however,—that this was ulcerating endocarditis.

The autopsy confirmed their opinion: there was no morbid change in the intestinal canal deserving of notice. *Peyer's patches and the solitary glands were in a perfectly normal condition.* The liver, kidneys, and brain presented no morbid appearances; that is to say, there was neither capillary embolism nor infarctus. The spleen was enlarged, and a little softer than natural. The heart was slightly hypertrophied. On the visceral portion of the pericardium, there was a white patch of old date. There was no imperfection of the aortic valves, nor of those of the pulmonary artery; no important alteration of the left side of the heart existed.

Though the valves of the pulmonary artery were normal, the tricuspid valves were damaged. One of them was perforated, and on the edges of the perforation, were small, grayish, fibrinous concretions of a granular appearance. Moreover, one of the largest of these granulations was retained by a pedicle which was also fibrinous: on lowering this vegetation upon the ventricular surface of the valve, it entirely obliterated the perforation. The auricular surface of the same valve was more altered; it presented small and easily detached whitish-gray mammillary prominences, which seemed principally to consist of fibrinous deposits, and appeared also to be partially composed of valvular tissue in the incipient stage of disintegration.

On examining the valvular vegetations with the aid of the microscope, they were found to consist of fibrin in the fibrillary state, containing amidst it some red blood-globules and a very small number of white corpuscles. The diseased portion of the valve contained a fibrinous, fibrillary deposit, a very few oblong cell-germs, and a still smaller number of fusiform bodies. In the fibrinous substance there was a large number of very minute granules of fat. There did not exist any germs of pus-globules, nor any pus-globules. There were very few white corpuscles in the cardiac clots.

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* CHARCOT et VULPIAN: Note sur l'Endocardite Ulcéreuse Aigue à forme Typhoide. [*Mémoires de la Société de Biologie, année* 1861. Paris, 1862.]

Gentlemen, observe that the dominant constituents of the morbid valvular products are fibrin in the fibrillary state, and fat-granules. The anatomical disposition of these morbid products renders it probable that similar products may be carried in the sanguineous current through the pulmonary artery.

In point of fact, the pulmonary parenchyma contained numerous abscesses, the largest of which were about the size of a hazel-nut, the majority being not bigger than hemp-seed. They were all covered with a tolerably thick false membrane. These abscesses contained a yellowish-gray substance, in which were found a great many purulent and pyoid corpuscles, and likewise numerous cells with several cell-germs. On the surface, and in the substance of the lungs, were observed irregular patches of ecchymosis, in some places resembling apoplectic clots. At a number of points, a yellowish-white matter was observed filling the canals. These canals were turgid, and seemed to be branches of the pulmonary artery. The matter with which they were distended was in the form of cylinders, and consisted of fibrin: the cylinders contained innumerable pus-globules.

How are we to explain the succession of morbid phenomena in this case, the details of which are so complete? Probably, as MM. Charcot and Vulpian remark, the ulceration of the valve proceeded very slowly; and the shivering fit was the signal of poisoning produced by the blood becoming mixed with the valvular detritus. The numerous metastatic abscesses in the lungs were the result of arrest of fibrinous matter in the minute ramifications of the pulmonary artery. Moreover, the fibro-purulent substance, which obstructed several ramifications of the pulmonary artery, is an anatomical proof of the cardiac origin of the pulmonary abscesses, although in no part of the peripheric venous system did there exist any cause of embolism or purulent infection.

In Dr. Chalvet's case the diagnosis was made by Dr. Lancereau on the dissecting-table.* The ulcerous endocarditis was in this case situated in the left side of the heart, and the autopsy was so carefully made, that the advance of the general infection was followed (so to speak) step by step; then, by chemical and microscopical examination, the matter itself which was the cause of that infection was demonstrated in the blood.

A woman, aged twenty-two, who had been in impaired health for a year, was suddenly affected with anxiety, epigastric pain, and nausea. She was desirous to continue at her work, but was soon obliged to desist, being seized by a violent attack of shivering, which lasted two hours, and was followed by sweating. Next day, there was a return of the shivering: four days from the first onset of the symptoms, the patient was taken to the Hospital Saint-Antoine, where she presented all the symptoms of a violent attack of cholerine—vomiting, diarrhœa, coldness of the extremities, cramps, feeble voice, and exhausted strength. There was no albumen in the urine.

The diarrhœa became less abundant, and the pulse rose; but the vomiting continued. On the eighth day of the disease, the face and eyes were injected; the fever was more intense, and the skin was hotter. On the ninth and tenth days, symptoms the same as those now described were present, and the patient was restless and very anxious. On the eleventh day, jaundice appeared, which soon spread over the whole body. The stupor and adynamia accompanying the appearance of the jaundice afforded some ground for thinking that the case was one of malignant jaundice. Next day, some ecchymotic spots were visible upon the limbs. The countenance had a more anxious cast. On the thirteenth day, the patient died.

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* CHALVET: Case reported in Lancereau's memoir—"Sur l'Endocardite Ulcéreuse," published, in 1861, in the Comptes Rendus des Sciences et Mémoires de la Société de Biologie.

No examination of the heart was made; none at least is recorded in the history which we have of the case. No reference is made to the functional condition of the organ. The inference to be deduced from this fact, as well as from many others of a similar character, is that a lesion of the valves of the heart occurring without any symptoms to attract notice, became a cause of general infection, under the influence of which infection the patient succumbed after presenting typhoid symptoms.

At the autopsy, small ecchymotic spots were observed on the surface of the lungs; but the parenchyma of the organs contained neither apoplectic clots nor metastatic abscesses. The liver, on the contrary presented remarkable morbid changes: a great number of cells were destroyed, and an infarctus was seen near the centre of the organ, and many branches of the hepatic artery were partially obstructed by small clots, chiefly formed of fibrin.

The spleen, which was enlarged and diffluent, contained an infarctus in its upper and middle regions, pretty near the hilus. On the surface of the kidneys, there were small ecchymoses and slight depressions. Some of the larger renal capillaries were filled with a matter, which when examined under the microscope seemed to be finely granular dust.

The mucous membrane of the intestine presented small ecchymoses which probably depended on the same cause as the ecchymoses on the pulmonary and renal surfaces. There was no morbid alteration of Peyer's glands.

Here then, gentlemen, was a number of lesions, existing in different organs, which exactly corresponded with the symptoms observed during life. The mitral valve was thickened, and presented a very marked injected appearance: on its auricular surface, there was an excavation sufficiently deep to receive the extremity of one of the fingers. There were granulations at the bottom of the ulcerations; and the substance covering the ulceration, when examined through the microscope, was found to be composed to a great extent of fine grayish granulations, and detritus of a more or less formed description. The entire substance, says the observer, resisted the action of acids and alkalies.

Gentlemen, this vast ulceration was probably the result of the opening of an atheromatous cyst. This opinion is strengthened by the following fact. At a point very near the ulceration, there existed a slight mammillary prominence, from which, on being cut into, there flowed a thick yellow matter composed of numerous very fine granulations, fragments of cells, cell-germs, and conjunctive tissue. All the parts were pale; but they resisted, to a great extent, the action of acids and alkalies, a circumstance which seems to show that fibrin enters only to a very limited extent into the composition of these exudations. Finally, the microscope likewise revealed in their parts the presence of fat-globules and some crystals the products of the fatty matters.

What are we to conclude from this clinical and microscopical analysis? The inference is, that the valvular ulceration had poured into the blood fibrous and other products, which in the spleen, liver, and kidneys, had obliterated the vessels of medium size, so as to produce infarctus and obstruction of the capillaries to such an extent as to cause ecchymoses on the surface of these organs, and in the thickness of the cutaneous and mucous derma. Again, the organic and other exudations, except the fibrin, had probably caused a modification of the blood, which would explain the diffluence of the spleen and the typhoid symptoms.

To conclude—and here is the point of greatest interest in this remarkable case—the microscopic and chemical examination of the blood showed that it contained products analogous, in all respects, to those detected in the mitral valve and obstructed vessels.

As is observed in septicæmia, the blood was everywhere nearly in a viscous state—almost thready—and resembling treacle or gooseberry-jelly. The blood contained in one of the femoral arteries, having been collected in a tube, was submitted to microscopical examination: besides the red globules, the white globules, and the fibrin, the presence of molecular granulations, granular globules, fat-globules, and fragments of the fibres of conjunctive tissue were detected. Similar elements existed in the centre of the clots in the splenic, renal, and hepatic arteries. Similar elements were also met with, though sparingly, in the blood of the coronary arteries. These elements, like those which constituted the small valvular abscess, were rendered pale but were not dissolved by the acids, while they entirely resisted the action of the alkalies.

Such, gentlemen, are the two cases of which I have been desirous to explain to you the principal details. They are of a nature to fix in your minds the morbid changes which occur in ulcerating endocarditis; and will likewise explain how the blood becomes infected in such circumstances. Such are the facts which have to be added to the septicæmic class of affections, when the patients present typhoid symptoms which cannot be referred to dothinenteria, to well-marked infectious fever, or to malignant jaundice, and when therefore you must investigate with care whether the heart does not present some valvular lesion to explain the presence of typhoid symptoms.

Finally, if you are of opinion that the symptoms are independent of any rheumatic affection, this special organic etiology of blood poisoning will justify you in suspecting that a valvular alteration of the heart is the cause of symptoms which could in no other way be satisfactorily explained.

Gentlemen, I have been constrained to place before you the doctrine of ulcerating endocarditis professed by a certain number of most remarkable physicians who attribute to embolism the local and general symptoms observed in this affection. I cannot, however, conceal from you the fact that this doctrine does not command the suffrages of all observers—I cannot conceal from you that savans of the highest distinction, at the head of whom are Bouillaud, Hardy, and Béhier, attribute the general and typhoid symptoms of endocarditis to a primary morbid condition. Hardy and Béhier, in their "Traité de Pathologie Interne," say: "The ulceration and softening of the endocardium are the results of a general vitiation of the economy at the time when the inflammation of the membrane manifests itself; and which inflammation seems unable to produce any other than an infecting ulceration and the general phenomena indicated. It is, in fact, simply an endocarditis in a cachectic subject." Duguet and Hayem, distinguished young physicians, take a very similar view of the question; they hold that in these cases there is an aggravated malignant embolism characterized by multiple visceral lesions, particularly by lesions of the valves of the heart. The existence of the typhoid state from the beginning of the attack would be thus explained, and the visceral infarctus would only be regarded as secondary lesions, the remote consequences of ulcerations of the endocardium.*

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* DUGUET et HAYEM: Mémoires de la Société de Biologie: 1865.

In favor of the doctrine that the typhoid state arises from spontaneous embolism supervening suddenly, and not in any way the consecutive result of the passage into the blood of disintegrated fibrin coming from a valvular lesion, may be cited perfectly authentic autopsies performed in the hospital service and under the eye of Dr. Bouillaud, in which it was found that there was neither any sort of ulceration of the endocardium, nor any vestiges of partially eroded fibrinous vegetations, although during life there had existed a most decidedly typhoid condition. This occurred in the case of a female patient whose history was communicated by Dr. Auguste Voisin to Dr. M. L. Martineau.*

Consequently, we must not suppose that the doctrine of embolism explains all the possible phenomena of malignant endocarditis.

LECTURE LXXXV.

MARSH FEVERS—INTERMITTENT FEVERS.

The manifestation of a Diathesis.—Causes which produce that Diathesis.—Marsh Cachexia.—Organic Lesions: Engorgements of the Spleen and Liver.—These Lesions are both the Consequence and the Cause of Accidents.—Regular Intermittent Fevers.—Their Three Stages.—Their Different Types.—Marsh Fevers may be Continued at their commencement.—They must not be confounded with Continued Fevers, nor with Pyrexiæ beginning in marshy districts with Intermittent Paroxysms.

GENTLEMEN: In 1858, we received into St. Agnes Ward three persons with intermittent fever: one of them contracted this disease in the Crimea, and the other two in Africa. They returned to France, and came to reside in Paris, in 1857; but it was not till 1858, six months afterwards, that these individuals suffered from a recurrence of paroxysms of intermittent fever for which they had been treated, and apparently with complete success, in the country in which they were first attacked.

We frequently see recurrence of the paroxysms of marsh fever in persons for a long time removed from the influences which first produced the symptoms. This recurrence, common though it be, is not the less deserving of the attention of physicians: it is not without importance in relation to nosology, but its relations to therapeutics are still more important.

We must be careful not to confound these recurrences, more or less distant in respect of time, and more or less possessing the character of periodicity, with morbid phenomena proceeding from a single persistent cause, with a diathesis of the organism, or with relapses of pyrexiæ and phlegmasiæ.

A person, for example, after the lapse of weeks, months, or years, takes, a second time, a purely inflammatory peripneumonia: this second attack does not consist of new manifestations of an existing disease, but is on each occasion a new disease, exactly similar to the first in its seat and nature, like it, too, running through all the stages, and accomplishing, *uno tenore*, its complete evolution. If we admit the existence in the individual of a peculiar predisposition to pulmonary inflammation, we must suppose that in each attack, there is a new intervention of the same cause, or of some analogous cause, the effects of which would entirely exhaust themselves in a series of uninterrupted morbid acts, till there either is a return to previous health, or a fatal issue.

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* MARTINEAU: Des Endocardites: Paris, 1866. An excellent work, in which modern theories are discussed and interesting cases narrated.

It is not so, however, in respect of diathesic diseases, with which marsh fever has so much analogy, that I feel some difficulty in not placing it in the same class.

At some particular epoch, an individual is attacked by gout: some months later, he has a second and then a third seizure: each of these seizures is not a new disease, but only a new manifestation of the same disease of which the cause, never completely exhausted, though remaining quiet for a longer or shorter period, has not the less existed in power within the economy—existed *in posse*, to make use of an old medical expression. Though, under certain circumstances, an immediate cause becomes the starting-point of the accidents, the intervention of that cause is not absolutely necessary, as in peripneumonia, of which I have just now been speaking.

A similar remark is applicable to pox. Whatever time may have elapsed between the different outbreaks of characteristic symptoms, these symptoms are always subordinate to one and the same cause.

A similar statement applies to marsh fever.

It certainly never would occur to any one, that the periodic paroxysms of a quotidian, tertian, or quartan intermittent constitute so many distinct illnesses. It is palpable to every one, that it is always the same disease, the manifestations of which are separated by longer or shorter intervals, during which intervals, the morbific cause remaining latent, the economy has seemed to regain its perfect equilibrium of health. It is not, however, so generally believed, or at all events is not so generally stated, that the periods of respite may be exceedingly prolonged. I am not alluding to intermittent fevers said to have monthly or annual paroxysms, cases of which have been collected by excellent authors, among whom may be mentioned Schenck: I refer only to cases similar to those presented by our three patients in St. Agnes Ward.

You observe that in them, after an interval of six months, during which the individuals remained quite well, the manifestations of intermittent fever which they had had on the first occasion, and of which they had been apparently completely cured, have not been reproduced without an appreciable determining cause. In them, it has been the same fever which has made a new attack; and this has occurred, as on the first occasion, under the influence of an infection, the germ of which had not been wholly destroyed.

The germ of marsh fever not only sometimes remains quiescent for months or years after its first manifestation, but it also happens that individuals who have received the germ in countries where marsh fevers are endemic, do not experience the first apparent symptoms till long afterwards, when perhaps living in countries where such fevers do not generally prevail. Cases of this kind are by no means rare; and two have lately presented themselves to your notice, almost simultaneously, at this hospital. In one case, the patient was a child, admitted in July to St. Bernard Ward with intermittent fever, evidently contracted in Burgundy, where he had been reared, and whence he was brought six months ago to Paris. In the other case, the patient was a man, whom some of you must have seen in the wards of my friend and colleague, Dr. Delpech. A certain time has elapsed since this man returned from Africa. Enlarged spleen and a straw-yellow color of skin indicated that he was under the influence of serious paludal cachexia; and he came to the hospital to be treated for intermittent fever, which he assured us had first declared itself a few days previously, not the slightest paroxysm of fever ever having occurred in Algeria, where he had received the germ of the disease.

Gentlemen, from these remarks, you will already have seen the analogy which I pointed out between marsh fevers and diathesic diseases: in them, as in every diathesis, you will find that there is a morbific cause which may remain quiescent, for a longer or shorter period, in the organism which it affects. The analogy will present itself in a still more striking light when I explain to you the diversity of the manifestations of this morbific cause.

Bear in mind my definition of a diathesis. It is a special state, a peculiar disposition of the economy, hereditary or *acquired*, but essentially and invariably chronic, in virtue of which are produced disturbances in general pretty intense, which are identical in their nature, but variable and fluctuating in their form.

These disturbances of the system, variable and fluctuating in form, resting on the same basis, depending on one and the same cause, are met with in the disease now under our consideration. Though this disease manifests itself generally by intermittent and periodic attacks of fever, though its periodic character is so generally present that the term *intermittent* coupled with the designation of the fever is sufficient to define the nosological species, still, it frequently happens, that it assumes other features: the diathesis, and the paludal poison clothe it in forms essentially different from those in which it is generally clothed. It behooves the physician to be able to recognize the disease under this diversity of form, so as to be able to combat it.

The term *paludal* sufficiently indicates the nature of the diathesis, which in fact it describes. It recognizes that its ordinary cause is sojourn in places poisoned by paludal miasmata. In passing, let me mention, as a remarkable circumstance, that the marsh miasm seems to be a poison only to the human species. Few human beings can expose themselves to it with impunity, yet the lower animals, speaking at least from what we observe in respect of domestic animals, are not at all affected by it.

Though paludal affections are nowhere more common than in countries which are hot, low, and damp, you must not conclude that they are engendered by humidity of climate. Humidity only acts when there is considerable extent of surface covered with water, or when the soil is impregnated with water, which evaporates under the influence of certain atmospheric conditions, this evaporation producing telluric emanations, which perhaps constitute what we call the "marsh miasmata."

To so small an extent is humidity the cause of intermittent fevers, that one may without much inconvenience reside in a marshy district during the cold season, and during the rainy season, so long as the ground is submerged. But whenever the ground dries up, or even when it becomes partially exposed by the action of the heat of the day, there is danger. Whether it be a marsh properly so called, the bed of a river or rivulet, or an inundated meadow, marsh miasmata are developed, which poison those exposed to their emanations.

Again, even in dry and parched regions, similar results follow great disturbance of soil which has not been stirred for a very long period. Hence it is that in highly cultivated districts—on the elevated *plateau* of the

Beauce, for example—where there is a constant scarcity of water, very many cases of intermittent fever are met with.

The paludal affections which so cruelly ravaged our army during the war in the East, had no other cause than the great upturning of the soil necessitated by the siege works before Sebastopol.

Even here, in Paris, where intermittent fevers are of such rare occurrence that we hardly know them, they have shown themselves on several similar occasions. In 1811, during the digging of the Saint-Martin Canal, a veritable epidemic of intermittent fever prevailed in the districts of the Temple, the Villette, and Pantin. In 1840, there was a similar epidemic when the fortifications which now surround the capital were being raised. The diggings of later years required in piercing the town by new streets and boulevards, the construction of sewers and underground passages for the gas-pipes, have caused numerous paludal affections; and more than one physician has been surprised by meeting with cases of pernicious fever which he was little accustomed to encounter in his practice.

It must be stated that these paludal maladies, occurring accidentally as it were in countries which are usually exempt from them, are generally of a much less serious character than those which prevail as permanent endemics in localities where unfavorable conditions of humidity and temperature coexist. It is in such localities that we observe all the different manifestations of the diathesis, ranging from the intermittent fevers, which constitute the simplest expression of the diathesis, up to cases representing the very highest degree of the pathogenic action of poisonous miasmata.

The prejudicial influence of these insalubrious localities sometimes extends to a considerable distance beyond them, in consequence of atmospheric currents frequently transporting the marsh effluvia to places considerably remote.

The following fact, mentioned by Lancisi, and repeated in your textbooks, is one with which all of you are acquainted. Thirty inhabitants of Rome were walking towards the mouth of the Tiber: all at once, the wind arose, blowing across the marshes and carrying with it their emanations: twenty-nine of the thirty were seized with intermittent fever. Similar examples of the remote diffusion of paludal miasmata are not uncommon in the French districts of Sologne, Bourbon, and Bresse, as well as in other permanent centres of intermittent fever. If it be sufficient in many cases for a stranger to traverse a marshy country to take marsh fever, how much more liable to do so must be those who habitually reside in them: the diathesis impresses its stamp, or if I may use such an expression, becomes much more deeply rooted in the inhabitants, who, besides the miasmata, have other deplorable hygienical conditions to contend against.

The paludal diathesis does not always manifest itself by fevers: in very many residents in districts where marsh fevers are endemic, it announces its existence by organic changes, more or less characterized, and by an assemblage of morbid phenomena obvious to the observer.

There is a loss of color in the integuments, corresponding to a particular alteration in the blood: it is not merely the paleness perceived in persons exhausted by hemorrhages, nor is it the green hue of the chlorotic: it is a more or less deep tint of yellowish bistre.

This special cachectic tint, when strongly marked, generally coexists with engorgement of the spleen and liver, particularly of the spleen, which becomes much enlarged, and can easily be measured by percussion and palpation. Its increased bulk may be so great as to cause a decided projection through the abdominal parietes; and the enlarged organ may fill, as it were, the whole abdominal cavity, extending from the right beyond the median line, descending into the left iliac fossa, ascending into the cavity of the thorax, pushing up the diaphragm so as to embarrass the breathing, and, by exerting pressure on the vessels, assisting to produce those serous effusions, which I told you are frequent complications of this deplorable condition of the economy.

It is not unusual, as I observed in Sologne, for infants to be born with these visceral engorgements and the cachectic tint of the skin, testifying that even in the womb they had been under the same evil influence as that in which their mothers had lived.

Generally, however, before reaching this state of profound cachexia the individuals have experienced acute symptoms of paludal poisoning, acute phenomena of fever, which, according to the intensity of its cause, assume either the intermittent type, which we are accustomed to consider as the truly characteristic expression of miasmatic poisoning, or the remittent or continued type: this was clearly pointed out by the older physicians, particularly by Lancisi and Morton; and to our accomplished brethren of the army, Boudin, Laverau, and Maillot, we are indebted for having recalled attention to the fact.*

It is when these marsh fevers are of old date—even when the patients have left the countries in which they were first attacked—that the consequent anæmia and cachexia increase, becoming more and more marked. Then also supervene the serous effusions of which I have been speaking; œdema of the extremities, general anasarca, peritoneal and pleural dropsy, œdema of the lungs—affections which depend upon an alteration of the blood, and which (ascites and œdema of the lower extremities at least) have, to a certain extent, as their determining cause, the impediment to the portal circulation and to the passage of the blood through the vena cava produced by the enlarged liver and spleen.

When I come to speak of pernicious fevers, it will be seen that the spleen is sometimes the seat of very serious symptoms, of hemorrhages similar to those which occur within the encephalon, of softenings more or less extensive, and of more or less considerable ruptures which bring speedily mortal maladies in their train.

Every writer on intermittent fever has called attention to this cachexia and its accompanying engorgements of the abdominal viscera. Van Swieten, in noticing what his predecessors have said on this subject, adds, that in some cases, the dilatation and gorging of the colon, in its portions adjoining the spleen, are so great as to lead one to believe that the organ is tumefied.

You know, gentlemen, the part which has recently been assigned to the hypertrophied spleen: some physicians, as you are aware, resting upon the notion put forth by Dr. Audouard, and developed by him in different memoirs, hold that congestion of the spleen is the cause of the periodicity of the paroxysms.† This theory, which does not maintain its ground when the facts are seriously examined, has very few defenders in the present day, and consequently I shall not stop to discuss it. Let me only remark to you that this is no new idea, Galen having said, in several places in his works, that the spleen is the headquarters of intermittent fever.

This remark brings me to the consideration of the great question of the *cause of intermittence*, a question often discussed, but never yet solved.

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* BOUDIN: Traité des Fièvres Intermittentes, Rémittentes, et Continues. Paris, 1842.

MAILLOT: Traité des Fièvres ou Irritations Cérébro-Spinales Intermittentes. Paris, 1838.

† AUDOUARD: Des Congestions Sanguines de la Rate. Paris, 1848.

Some years ago, the following explanation was proposed by Dr. Masurel, a military physician. He said that the beginning of an intermittent fever is a vitiation of the blood by poisonous miasmata: by the ganglionic nervous system receiving an impression from the altered blood comes the paroxysm of fever, or rather the shivering, which is simply a symptom indicating the existence of a true neuropathic condition, a depressing heteronervation of the circulatory system: the second and third stages of the paroxysm, and the hot and sweating stages, are results of the reaction of the organism from the profound disturbance occasioned by the two conditions which presided at the development of the paroxysm. When the paroxysm is past, there still remain miasmatic principles in the blood, which, by their ceaseless round in the circulation, induce a new change in the nervous system; whence come a new paroxysm and a new circle of fever and remission.

This theory, which seems to derive its inspiration from Cullen's views regarding the cause of fever, has a great similarity to the opinion of Sydenham, who believed that the paroxysm of intermittent fever returns because the morbific matter, after accumulating in the system during the apyrexial period and being eliminated by the sweating, reaccumulates, till a renewed effort of nature again solicits its elimination.

To these statements, Van Swieten made the following peremptory reply: Although evacuations bring on crises in acute diseases, imperfect evacuations induce only imperfect crises, which never occur within such a period of time and at such regular intervals as in intermittent fevers.

Again, were the recurrence of the paroxysm dependent upon a remaining excess of morbific matter not expelled in the previous crisis, it would still remain to be discovered, why the potency of this morbific matter increases with greater or less rapidity according to the different kinds of fever—why, for example, it acts only after twelve hours in the quotidian, and only after thirty-six hours in fevers of the tertian type. How does it come to pass that an individual struck by a quartan should feel perfectly well a quarter of an hour before the return of the paroxysm, although the morbific matter then brought into action must have been previously quite ready to act? How can we admit that in one and the same variety of intermittent fever, affecting individuals differing in age, sex, and temperament, the morbific matter should take exactly the same time to accumulate and produce its effects? So inconsiderable are the evacuations essential to these fevers that, when they are treated by cinchona, recovery takes place without sweating, purging, or any unusual flow of urine, although it has been maintained by some persons that a cure obtained without critical evacuation is neither radical nor certain.

Finally, gentlemen, although everything leads to the belief that the cause of intermittence is in the nervous system, we are not acquainted with that cause. However, this much at least we know, that intermittence pertains essentially to the organism, and not to the action on it of an external cause, although we see it occur in physiological order without appreciable causes, and in the pathological order under the influence of the most diverse causes. Some of these causes, however, are more susceptible than others of exciting it, even of soliciting it with a constancy and regularity which is very often periodic.

The poison of marsh miasm is one of these causes. Though under certain circumstances, to which I have already alluded, it manifests its action by phenomena invested with the continued type, intermittence usually characterizes the phenomena induced by this morbific cause. The accidents of this description which are most common, or at least which seem to be best known to the majority of physicians, are those which constitute REGULAR INTERMITTENT FEVERS.

In exceptional cases, these fevers have precursory symptoms, such as general discomfort, accompanied by a feeling of debility, by lassitude and headache: the patient has pandiculation, and eructations. Generally, the attack begins abruptly; its paroxysms being composed of three distinct periods, called *stages*—the *cold, hot*, and *sweating* stages.

The *cold stage*, which ought rather to be called the *shivering stage*, because the sensation of cold experienced by the patient is merely the result of perverted sensibility, the temperature in reality changing but little, and even sometimes rising several degrees in certain parts of the body, a fact established by the experiments of Haen, which have been repeated by others. The cold stage is characterized by a shivering fit of more or less violence, of variable duration, but always rather long.

The shivering, which seems to begin in the lumbar region and ascend along the vertebral column, is soon trembling and real convulsive movement, affecting, in the first instance, the maxillary muscles, and so strongly as to cause the teeth to strike one another with a chattering noise. The trunk and limbs are then, and almost simultaneously, seized in such a manner that the rapid shocks by which the whole body is agitated shake the bed on which the patient is lying. The dryness of the skin, with its projecting papillæ giving it that appearance called *goose's skin:* the decoloration of the skin, nowhere more evident than in the face and extremities, which sometimes assume a slightly bluish tint, testifies to the disturbance which has supervened in the capillary circulation; while, at the same time, the smallness, feebleness, frequency, and irregularity of the pulse, show that there is embarrassment of the arterial circulation.

There is great anxiety, marked oppression, intense thirst, the tongue remaining moist and sometimes bluish: there is complete loss of appetite: and this state of extreme discomfort is not unfrequently increased by fits of vomiting. The urine is scanty, pale, and aqueous.

These phenomena continue for one or two hours, and in some rare cases for three or four hours; but never longer, unless when the fever is an algid intermittent, which does not belong to the legitimate intermittents, of which alone I am now speaking, but is one of the pernicious fevers, which we shall afterwards have to consider.

The *hot stage* commences by the rigors becoming more and more transient, and alternating with warm flushings. The heat, slight at first, grows increasingly intense, and becomes sharp, biting, and very painful. The patients vary their position in bed, seeking a somewhat cooler place. The degree of heat is far from being in relation to the preceding shivering, and is often much less in reality than it would appear to be from the statements of the patients. Modern researches have shown that the temperature of the body, as ascertained by the thermometer, is, in many cases, not more than one degree above that which it indicated during the cold stage. However, to the touch, the skin is dry and hot: the face is red and animated—the eyes brilliant, and sometimes very sensitive to light.

The pulse loses its hardness, without diminishing in frequency: it gains in amplitude and power. The respiration is less anxious, deeper, and more frequent. The headache, with which the attack began, increases in place of diminishing; and in some cases, there is a little delirium. The anorexia and thirst continue. The urine has a more or less deep color. The average duration of this stage is from one to two hours. In some patients, the skin becomes moist as soon as the shivering ceases.

In due course, these symptoms moderate; and the *sweating stage* sometimes announces itself by a general feeling of comparative well-being. The skin relaxes, little by little, becomes moist, and is soon covered by a profuse sweat. In some subjects, vomiting and slight diarrhœa seem to indicate that a similar emunction is taking place from the internal integument. The urine, red when it is passed, deposits on cooling a sediment of the color of brickdust. This sediment, however, which some have looked upon as an essential phenomenon in all attacks of intermittent fever, is not always found: the urine may be thick or yellowish-white, resembling mare's urine, or slightly cloudy, or finally, it may differ very little from normal urine.

The cessation of the fever is announced by a diminution in the frequency of the pulse, which preserves its fulness.

Generally, as soon as calm is restored to the system, the patient falls asleep: this restorative sleep sometimes comes along with the sweating.

So terminates the paroxysm of a legitimate intermittent fever: it is succeeded by a period of repose, the *apyrexial period*, which, according to the kind of fever, lasts for a longer or shorter time.

When the apyrexia is of long duration, the patient entirely forgets, so to speak, the paroxysm which he has traversed, and fancies that he is restored to perfect health; but when the period of repose is short, or when the paroxysms for a time recur frequently, the repose is never absolute. There may then be observed general discomfort, an indescribable feeling of fatigue, slight heaviness of the head, a certain degree of anorexia coincident with a saburral state of the tongue, thirst, and irregularity in the alvine evacuations, showing that the functions have not entirely regained their normal action.

Turgescence of the liver and spleen, particularly of the latter, in persons who have been for a considerable period affected with these fevers, is a very significant phenomenon.

The apyrexial period, according to its duration, is characteristic of the different *types* which intermittent fever may assume. Gentlemen, you know what these types are.

The *quotidian* fever has its paroxysms nearly the same, returning daily almost at the same hour. Let me here call your attention to a very important fact. In intermittent marsh fevers, the paroxysm generally commences in the morning, or at least before noon: on the other hand, the attacks of certain *symptomatic* intermittent fevers, also quotidian, such as those so often accompanying tubercular disease and the commencement of some pyrexiæ, supervene towards evening. The *tertian* consists of paroxysms returning every two days, that is to say, on the third day, counting that on which the first attack showed itself.

The *quartan* returns every three days, that is to say, on the fourth day, reckoning the day on which the preceding paroxysm occurred.

Besides these, the more ordinary types of intermittent fever, there have been mentioned quintan, sextan, septan, octan, and novan fevers; but during my long practice I have never seen them.

The principal types, however, the quotidian, tertian, and quartan, present numerous varieties known as duplicated and reduplicated fevers.

The duplicated, more common than the reduplicated, are those in which there are two paroxysms, occurring daily in the *quotidian*, every two days in the *double tertian*, and every three days in the *double quartan*.

As to the reduplicated fevers—there is in the *double tertian* one paroxysm every day, and in the *double quartan* two consecutive days with a paroxysm, and then one apyrexial day. Finally, in the *triple quartan*, the paroxysm occurs every day.

You no doubt, gentlemen, expect me to explain the difference between quotidian fevers and those of the double tertian and double quartan types, as in all of them there is a daily paroxysm. The difference is this: in the quotidian, all the paroxysms are nearly identical with each other: in the double tertian, the paroxysm of the third day resembles that of the first day in form, intensity, duration, and hour of occurrence, and the paroxysm of the fourth resembles that of the second: in the triple quartan, the paroxysm of the fourth day resembles that of the first, the paroxysm of the fifth resembles that of the second, and the paroxysm of the sixth resembles that of the third day. For example, suppose a double tertian, the first day's paroxysm of which began about noon, and continued eight hours, while the paroxysm of the second day commenced only two hours later, and lasted only seven hours: the paroxysm of the third day, like that of the first, will begin also at noon, but will not terminate till eight o'clock, while the paroxysm of the fourth day will begin at two o'clock, and will not be ended till nine o'clock, like that of the second day.

A remark, the full value of which you will understand when you have to treat a case of intermittent fever is—that the more the type is removed from the quotidian, the less amenable is the case to treatment. The quartan, therefore, is that which goes on for the longest time. The Latin imprecation, "*quartana te teneat*," for which it would not be difficult to find a French equivalent, shows, that the observation had in ancient times attracted the attention of the general community, as well as of physicians.

The same remark implies that the quotidian is the least obstinate: and that is the fact. Generally, a quotidian fever undergoes spontaneous cure without the intervention of the physician. This arises from quotidian fevers being seldom of marsh origin. They are met with everywhere, and at all seasons, at the commencement of certain pyrexiæ. In those countries in which intermittent fevers are endemic, most diseases in their earliest stage assume the quotidian type. I must add, however, that it is not unusual to see legitimate marsh fever take this type in young persons attacked for the first time: usually, however, in these cases, the fever soon becomes double tertian and then distinct tertian, thenceforth exhibiting all the characters of ordinary marsh fever.

The type seems to be derived much more from the nature of the miasm, or to speak more correctly, from the locality which it infects, than from conditions inherent in the individual.

Tours and Saumur, both situated on the left bank of the Loire, seem to me to present the same climatological and telluric conditions. Nevertheless, at Tours, tertian fevers are seldom seen; and the few cases of quartan fever which I there met with occurred in persons coming from Saumur, Rochefort, and other places where they had contracted the disease.

The following is an account of one of the most striking of the cases to which I am now referring. Fourteen soldiers living in barracks at Saumur went to Tours to give their evidence before a council of war. They had been hardly ten days in Tours, when nine of them were obliged to enter the hospital for *quartan* fever, the germ of which they had evidently contracted at Saumur; but all the fevers were tertian which I met with among the inhabitants of Tours and its environs.

An intermittent fever is called *regular*, when the paroxysms return at almost exactly the same intervals—*retarded* when the apyrexial period is prolonged—*anticipatory* when the apyrexial period is shortened.

In anomalous intermittents, the paroxysms may be *subintrant*—that is to say, one beginning before the other has terminated.

The diagnosis of an intermittent fever is certainly very easy; but no one, even the most experienced and talented, can pretend to that accuracy of judgment which would have been exacted by Galen who considered that the physician ought, from seeing the first paroxysm, to be able to say whether the fever was tertian or quartan: "*tertianam quidem a quartana qui primo statim die, nescit distinguere, neque omnino medicus est.*" Although Galen has collected the differential signs which, in his opinion, ought to guide us, their value is too disputable to allow us to place any reliance upon them. Before we can know the type of the fever, we must wait till several paroxysms have occurred: nay, we must even wait to be sure that the malady really is intermittent fever.

Often, in fact, as I have just been saying, and as is observed particularly in countries where marsh fevers are endemic, some continued fevers commence as intermittents, though they soon assume their proper type. This is a point to which I directed your attention when lecturing on dothinenteria, in relation to the cases of two women we had in St. Bernard Ward, in whom typhoid fever had commenced with the symptoms of intermittent fever—in the one as a quotidian commencing abruptly, and in the other, first as a tertian, then as a double tertian, then as a remittent, and finally, as a well-marked continued fever.

The subject is so essentially clinical, that I am not afraid of repeating the remarks I made on the occasion I refer to.

If your practice lie in a locality where marsh fevers are endemic, or among persons who have formerly inhabited such countries, I advise you to avoid concluding hastily that a fever is intermittent, unless the type be tertian or quartan: be likewise distrustful of appearances when the fever is double tertian, and be still more distrustful if it be quotidian. Before administering preparations of cinchona, which would fail in such cases, wait to see whether the type do not change. Should the fever prove to be continued, the intervals between the paroxysms will become shorter and shorter, and the paroxysmal manifestations will be less in this way: for example—if the shivering, chattering of the teeth, and general discomfort have continued an hour on the first three or four days, towards the fifth, sixth, or seventh day, it will not continue more than half an hour, and about the eighth or ninth day, the shivering will be quite transient. But while the paroxysms are less distinctively marked, the whole paroxysm will every day become more and more prolonged, the continued type will become more and more decided; and ere long, the disease will be exactly characterized. In addition to the manifestations of fever, there are symptoms which give a clue to the diagnosis. On interrogating, and attentively examining the patient, there are observable a certain number of phenomena which are not generally met with in marsh fever, and which belong to dothinenteria: I refer to the general softness of the pulse, vertigo, insomnia, and a feeling of discomfort, greatest in the interval between the paroxysms: then, there is the tendency to diarrhœa, and the gurgling produced by making pressure over the right iliac fossa.

Intermittent fever symptoms may also supervene at the beginning of phlegmasiæ, pleurisy, and pneumonia; and a still more prejudicial mistake may occur when the symptoms are those of inflammation of the pulmonary parenchyma, as they may lead us to believe that we have to do with one of the forms of pernicious intermittent fever.

On the other hand, in districts poisoned by marsh miasmata, you will see individuals suddenly seized with exceedingly violent symptoms of continued fever, which from their course and severity seem to indicate the onset of an attack of dothinenteria. After a certain time, these symptoms become complicated with shivering, which at regular intervals increase, and recur with characters more or less marked, the intermittent paroxysms following a gradation inverse to that which we have seen them presenting in the previously described cases, becoming quotidian, then double tertian, tertian, and even quartan.

You can now understand the full value of the Hippocratic precept, to which I drew your attention a few minutes ago,—not to interfere with intermittent fevers till there have been a certain number of well-marked paroxysms.

By conforming to this rule, when you have to do with a dothinenteria clothed at its commencement with the symptoms of intermittent fever, you run no risk of employing inappropriate treatment, and of blaming the cinchona for having changed a generally mild into a severe malady. In an attack of mild synochus, such as we frequently meet with in Paris, assuming at first the aspect of an intermittent fever, and from which there is generally spontaneous recovery, we must not fall into the mistake of supposing that we have cured a marsh fever by small, badly administered doses of cinchona, or sulphate of quinine, nor by some of the supposed febrifuges, such as kitchen salt or the bark of the *Æsculus hippocastanum*, remedies recently lauded, the apparent success of which is attributable to their having been employed in cases similar to those of which I have now spoken.

Finally, when we have to do with intermittent fevers, assuming at first the continued type, when we are waiting before we interfere, we must not suppose that we have reduced an incipient dothinenteria to a legitimate intermittent admitting of being cut short by cinchona.

Pernicious Intermittent Fevers.—What is the meaning of the term "Pernicious?"—Different kinds of Pernicious Fevers, such as the Algid, the Hot, and the Sweating; and those characterized by Coma, Delirium, or Convulsions.—They are usually of the Tertian type.—They are Anticipating or Subintrant.—Coloring of Organs, particularly of the Liver and Brain, by Pigmentary Embolia.—The Pernicious Symptoms may be due to Embolism.—Flagrant Insufficiency of the Mechanical Theory.—Masked Fevers.—Affections termed Neuralgic and Neurotic: Flux.

GENTLEMEN: Regular intermittent fevers, like those of our two patients in St. Agnes Ward, are not generally of a serious character, except in so far as they lead to dangers inherent in that extreme cachexia which is induced by their prolonged duration.

It is otherwise in respect of *pernicious intermittent fevers*. They frequently involve immediate danger; and when not treated promptly and energetically, almost inevitably have a fatal issue.

They are of rare occurrence in Paris; though, as I have already said, they have been seen here rather more frequently of late years, in consequence of the disturbance of the soil necessitated by the improvements which have been going on in the town. They are very common in Algeria, and in some parts of Europe, such as in the environs of Rome, in the Pontine Marshes, as well as in some departments of France; they are still more frequent in the equatorial latitudes both of the old and new world.

Intermittent fevers are called *pernicious*, when there is a perturbation of the economy very greatly jeopardizing the life of the patient within a few hours or days.

The pernicious symptoms of marsh intermittent fevers show themselves by exaggeration of the usual phenomena of the disease: when there is exaggeration of the shivering, the fever is called *algid;* when there is exaggeration of the febrile reaction, it is called *burning;* and when excessive sweating occurs, the term *sudoral* is employed. Functional disturbance of the organs essential to life is also met with; to pernicious intermittent fevers of this kind, the epithet *comitatæ* was applied by our medical predecessors.

I may now say in a word, to prevent the necessity of recurring to the topic, that the *pernicious* element of the fever depends less upon the disturbance excited in the economy by the affection of a particular organ, than upon the essential nature of the disease. It does not consist in the intensity of the functional disturbance of this or that organ, but in the insidious approach of imminent death—the malignity, the veritable, inherent, protopathic malignity with which in nearly every case the attack at once declares itself. The truth of this remark is shown by the fact, that the extent of this danger is far from being proportionate to the importance of the organ specially affected: this we see in pernicious intermittents characterized by cardialgic and dysenteric symptoms, and still more in the algid, burning, and sudoral forms just named, which do not seem to be concentrated in any particular organ.

Pernicious intermittent fever assumes the most diversified forms, which, I repeat, are characterized either by exaggeration of one of the usual phenomena of the disease, or by functional disturbance of some organ. The most common forms are the *algid* and the *sudoral.*

In algid fever, the cold continues from the beginning to the end of the paroxysm. It sets in with shivering of much more than ordinary violence, which rapidly increases in intensity: it lasts for a good many hours: the temperature of the body is really and obviously lowered: the tongue becomes icy: the skin when pinched retains the fold made in it, as it does in the algid state of cholera morbus. The thirst is intense; and the anxiety extreme. The face has a cadaveric expression—*cadaveris imaginem refert,* says Borsieri. There is no acceleration of the pulse; and if the symptoms subside, the patient very slowly regains his heat.

In the sudoral fever—the *febris diaphoretica* of the ancients—sweating commences at a stage rather earlier than usual, and soon becomes so profuse as to bathe the surface of the body. Coincident with this cold sweat, there is a rapid, small, weak pulse, accompanied by quick, painful breathing. The fingers seem as if macerated: the countenance is livid. The coldness is so great, that it is necessary to warm the patient, who may die in the first paroxysm. Should he emerge from it, his state of physical and mental prostration is extreme.

Among the forms of pernicious fevers, called *comitatæ* by the older physicians, we have the comatose, soporose, apoplectic, lethargic, delirious, convulsive, tetanic or epileptic, syncopic, cardialgic, hemorrhagic, petechial and scorbutic, peripneumonic and pleuritic, gastralgic, hæmatemesic, choleraic, and dysenteric. These terms are used to indicate the particular system of organs specially implicated, whether, for example, it be the nervous, respiratory, or digestive systems. They sufficiently point out the nature of the symptoms which characterize these different forms of fever.

In the *pernicious comatose fever,* the most striking phenomenon is a somnolence, which, showing itself at the beginning of the cold stage, or with the hot stage, goes on progressively augmenting during that second period up to the beginning of the sweating stage, being at last the profound stupor called carus. If an attempt be made to get the patients out of this state by giving stimulants, they open their eyes, but immediately shut them, moaning plaintively like persons roused out of their first sleep. In certain cases, the state to which I am referring is a veritable lethargy: in others, it is like an apoplectic stupor; and may be mistaken for apoplexy, if no notice be taken of the intensity of the fever, the heat of the skin, and the acceleration of the pulse, and if the duration of the initiatory shivering be not taken into account: the symptoms are scarcely so violent as in true apoplexy. The comatose symptoms gradually disappear within a time proportionate to the duration of the fever paroxysm, whether that be eight, ten, twelve, fifteen, or twenty-four hours. The patients then begin to take cognizance of the external world, and are amazed at what has occurred to them, of which they do not retain the slightest recollection. Though sometimes continuing to have a drowsy tendency, they seem completely restored to health, till the moment of the commencement of a new paroxysm, the recurrence of which will be more or less distant according as the fever is quartan, tertian, or quotidian.

The *delirious form* is characterized by a delirium, which, often announced by hallucinations, likewise shows itself at the beginning of the shivering, increases in intensity during the hot stage, and terminates with the termination of the sweating stage.

In *pernicious convulsive fever*, the convulsions are generally at once tonic, clonic, and epileptiform; but sometimes, though seldom, they are exclusively tonic and tetaniform.

The *syncopal*, so wonderfully described by Torti, is perhaps the worst of all the forms of pernicious intermittent fever.* It is the worst, in this sense, inasmuch as sometimes there is produced a state of apparent death, subjecting the patients to the risk of being left to die, when they might have been saved by timely medication.

A station-master of the Avignon Railway, subject for some time to paroxysms of intermittent fever, had repeated fainting fits. The syncope on the last occasion was so complete, that the absence of pulse led to the belief that life was extinct. Upon what seemed evidence of death, the body was taken to the autopsy theatre. It had been there for only a few hours, when it was the will of Providence, that a servant should require to enter. This lad hearing groans perceived the error which had been committed, placed the unfortunate individual again in his bed, and summoned M. Chauffard, physician to the hospital. Cinchona was immediately administered in large doses. The untoward symptoms ceased; and the patient regained his health.

Another individual, having fallen into a faint, was likewise accounted as dead, and had the face covered with a sheet: M. Chauffard, when making his examination, observed, that although the radial, axillary, and carotid arteries had ceased to beat, some slight movements of the heart were still perceptible. He immediately ordered a quinine lavement: the patient was saved.

In some cases, the fainting is preceded by acute precordial pain called *pernicious cardialgia*. It generally begins abruptly, without any apparent cause, the patient falling down in a state of exhaustion, induced either by a movement made in attempting to change his posture, or sometimes even by merely moving his arms. The pulse becomes small, accelerated, difficult to be felt, and then entirely ceases. The eyes are hollow; and, as in the cases I have laid before you, the person is apparently dead. Death may occur at any time subsequent to the first paroxysm.

Epistaxis, hæmaturia, a more or less abundant petechial eruption, covering a greater or less extent of surface, accompanied by a small frequent pulse, characterize *pernicious hemorrhagic fevers*, which are petechial and scorbutic.

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* TORTI: Therapeutica Specialis ad Febres Periodicas Perniciosas. Nova editio, curantibus Tombeur et Brixhe: 1821.

The symptoms commence with shivering, followed by a more or less severe hot stage, which is always accompanied by precordial anxiety, and sometimes by pain extending from the loins to the back, and to the pit of the stomach.

Peripneumonic pernicious fever, or, to borrow from Torti and Morton the more appropriate name, *pernicious catarrhal fever*, is characterized by pulmonary symptoms. The respiration is difficult, embarrassed, and disturbed: the face is turgid, and the eyes are injected: the forehead and chest are covered with sweat. In addition to these external signs of suffocative catarrh, there exists copious expectoration of a mucous bloody character, like that seen in certain cases of pulmonary apoplexy; and upon auscultation, crepitant and fine subcrepitant râles are heard in all parts of the chest.

Pleuritic pernicious fever declares itself by a stitch in the side, in some cases acute and poignant, and in others obtuse: the pain is increased by the respiratory movements, which are short and embarrassed. The pulse is small, hard, and often unequal. On percussion and auscultation, it is found, that in the corresponding side, there is pleuritic effusion, which is generally absorbed during the interval between the paroxysms.

In *cardialgic pernicious fever*, the pain at the pit of the stomach is so dreadful—so ferocious, to adopt the epithet applied to it by Borsieri*—that the patients utter terrible cries. Vomiting, at first mucous, then bilious, supervenes. There are other cases in which there is hæmatemesis; and in such cases, the disease is said to be a pernicious *hæmatemesic* fever.

When the intestine is attacked, there may be an alvine flux resembling the water in which raw meat has been washed, and as profuse as the discharge from the bowels in cholera. This form of the disease is called *pernicious choleriform fever*; or *pernicious dysenteric fever* when the flux is a sanguineous secretion. In both of these forms there is great prostration of strength.

Gentlemen, it is essential to bear in mind, that all these different forms of pernicious fever, however various, have characteristics in common.

In respect of type, they are generally tertian, sometimes quartan, exceptionally quotidian. Cases are likewise mentioned in which the type was remittent; and when the rigors are recognized with difficulty, continued fever may be simulated.

Though generally tertian or quartan, pernicious intermittent fevers do not assume these types till after several paroxysms have occurred. However, in countries where the poisonous miasmata have great power, as in Africa, or in individuals who after living in these countries, come to other countries, carrying with them the morbid germ, the fever may at once become pernicious, and carry off the patient in its first paroxysm. At the autopsy, in these cases, as in the others, the spleen is found to be enlarged, softened, pulpy, and sometimes ruptured.

Generally, I repeat, the disease does not so rapidly assume its pernicious character. The patient has first had many regular paroxysms of fever, which after a certain time change their character. The different stages are prolonged beyond their ordinary duration and leave the patient in a state of debility, contrasting remarkably with the amelioration—the return to almost perfect health—which followed the previous paroxysms. Finally, there supervene the morbid phenomena which mark the pernicious character of the malady.

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* Borsieri: Institutiones Medicinæ Practicæ. Lipsiæ, 1825–1826.

When the symptoms from the first paroxysm in which they show themselves do not indicate that the case will have a fatal termination, they follow the course which I have pointed out to you, beginning, that is to say, with the cold stage, increasing with the hot stage, and gradually decreasing from the commencement of the sweating stage, till they at last spontaneously cease. It is very necessary to be aware that this is the course of events; for if, mistaking the nature of the phenomena, the physician is eager to combat the symptoms—the coma, convulsions, or delirium, for example—by local or general bleeding, or by any other measure which is at least useless if it be not dangerous, he will, from the moment that calm begins to show itself, take credit for having obtained a fortunate result, which his ill-judged intervention has only not prevented. At the very time that he is congratulating himself upon having achieved a success, the patient may be carried off by a paroxysm which might have been averted by appropriate treatment.

We must also beware of not being misled as to the nature of the malady, when the paroxysms of pernicious fever are separated by intervals shorter than those of regular intermittents.

When the paroxysms are prolonged beyond their usual duration, the fever is called *anticipatory* or *subintrant—anticipatory* when, for example, a first paroxysm having commenced at noon, the succeeding paroxysm begins six hours earlier; and *subintrant* when the second paroxysm begins before the first has terminated, the paroxysm imbricating in such a manner as to prevent the patient recovering from the shock of one attack before he has to sustain a second. Were it for this reason alone, anticipatory, and still more subintrant, intermittent fevers, irrespective of any complication, demand the active intervention of the physician. But although we may, or rather ought, to adopt the precept of the older physicians—to wait, in a regular intermittent, till several paroxysms have occurred—we must beware of delay when the stages exceed their normal duration. In such cases, even when the stages have not presented anything unusual, act without loss of time, particularly if you perceive any disturbance of important organs, for then danger is imminent, and there is no time to lose.

I must not omit to mention a pathogenic theory promulgated in Germany by men whose works I exceedingly esteem. The theory to which I refer attributes the pernicious symptoms to *pigmentary embolia*. For the sake of those among you who are not acquainted with these views, I think it will be useful to enter somewhat into detail.

In the bodies of persons who have been under the influence of marsh miasmata and have succumbed during a paroxysm of pernicious fever (intermittent, remittent, or continued), we frequently discover certain lesions of the liver, that organ presenting a steel-gray or blackish color—sometimes a chocolate color. This change of color is due to the accumulation of pigmentary matter in the vascular apparatus of the organ: the liver is pigmented.

The spleen presents exactly similar lesions: it has a sombre or bluish-black hue, which is either uniform or disseminated in patches. In its interior, we find a still greater quantity of pigmentary matter than in the liver.

We also always find black pigment in the capillaries of the lungs.

It is likewise found in the brain, where it is still more easily detected, from its impinging upon the white color of the organ. The cortical substance then assumes the color of chocolate or graphite, while the medullary substance is not in any degree modified; or at least only in those cases in which there is a great amount of pigment. Under the microscope, the capillaries are to be seen full of granules and black particles, sometimes uniformly distributed, and sometimes accumulated in masses.

What then is this pigment? What is its mode of formation within the organism? It is a matter of a deep-black color; or, it is ochrish-brown; or, in exceptional cases, it is yellowish-red. It is formed to a great extent of granules, with which are found a small number of true pigmentary cells containing within them a greater or less number of black granules. The pigmentary granules are amorphous; but the agglomerations which they form are sometimes irregular, and sometimes cylindrical, from their being moulded in the interior of the vessels.

The pigment is the result of the transformation of the hæmatin; and this transformation may take place *anywhere, even external to the vascular system.* I beg that you will remember this fact. The metamorphosis is purely physical, or is, in other words, absolutely independent of the vital laws.

So much for fact: let us now attend to the theory.

The pigment is manufactured in the spleen, which organ decomposes the blood-globules, and transforms their hæmatin into pigment. The pigment thus formed passes into the vena porta, then into the liver, then into the vena cava, and then into the heart, whence it is carried into the general circulation. Here, we find ourselves brought back to the old atrabiliary doctrine; but also to a doctrine which is highly scientific and thoroughly armed.

Intermittent fever determines hyperæmia of the spleen: under the influence of this hyperæmia, the spleen manufactures a very large quantity of pigment. "In fact," says Frerichs, "in the normal state, the blood passing at once from the narrow capillaries into the venous cavities, flows slowly, and sometimes stagnates at certain points: then forming agglomerations of blood-corpuscles, it is, little by little, metamorphosed into pigment. In the hyperæmia of the spleen consequent upon intermittent fever, the stagnation is exceedingly marked, and *the result is the formation of masses of pigment.*"*

Frerichs is likewise of opinion, "that the pigment is developed at the expense of the blood which remains in the venous sinuses: the cellules, spindle-like and clubbed, of this pigment are formed from the internal parietal epithelium of the sinuses impregnated with decomposed hæmatin: the globular cellules are white blood-corpuscles charged with molecules of coloring matter: the pigmentary masses are detached fragments of concretion."† I have quoted the exact words of Frerichs, so that I might be certain to place correctly before you the German theory.

This extensive destruction of blood-globules by the spleen directly, necessarily impoverishes the blood; and this explains the anæmia which is so common a sequel of marsh fevers.

The excessive production of pigment will lead to a series of consequences, both of an organic and functional character, depending upon two causes entirely of a mechanical nature—the crowded state [*encombrement*] of part of the capillary system, and the damming [*barrage*] of the circulation. Indeed, Frerichs admits that the masses of pigment are stopped in the capillaries of organs, and there constitute a species of embolism.

To this mechanical doctrine, which is essentially only an application of the doctrine of embolism to the theory of pernicious symptoms, a doctrine far too superficial, and even in contradiction to the facts observed by the authors who support it, Dr. Peter and I make the following objections:

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* FRERICHS: Traité des Maladies du Foie: traduit de l'Allemand par Louis Duménil et J. Pellagot, p. 497. Paris, 1866.

† FRERICHS: Op. cit.

This crowding [*encombrement*] induced by embolism must be, and in fact is, first produced in the capillaries of the liver, the organ first in the course of the blood returning to the spleen. The obliteration of a part of the hepatic capillaries, and the consequent stasis of the blood, produce disturbance in the process of hæmatosis, because the liver is a hæmatopoietic organ; and Frerichs has very nearly adopted the belief that the gastro-intestinal hemorrhages, the attacks of profuse diarrhœa, and the vomiting, are the ultimate consequences of functional disturbance of the liver.

However, some of the pigment traverses the liver, passes into the right side of the heart, and reaches the lungs, where it ought to induce pigmentary crowding [*encombrements pigmentaires*]; but it would appear that this mischief-working material causes no bad effects, for Frerichs observed that the appearances found at the autopsies do not allow us to attribute the dyspnœa, or other functional disorders of the respiratory function, to pigmentary obstruction of the capillaries of the lungs. As you know, the capillaries of the lungs are smaller than those of any other organ. One cannot, therefore, understand how it is that the pigment which has been able to pass through the hepatic capillaries, which have a larger diameter, should not be stopped in the pulmonic capillaries which have a smaller diameter: but this is not the only absence of natural consequence involved in the iatro-mechanical system.

It appears then, according to this theory, that, notwithstanding seeming improbability, pigment traverses the lungs, reaches the right side of the heart, whence it is discharged over the whole organism: hence results *melanœmia*, that condition of which you have heard me speak, which explains the peculiar color of the face in marsh cachexia. We ought to find pigmentary embolism in every organ; but this is not the case, for the brain and kidneys almost exclusively enjoy this unfortunate privilege.

Frerichs says: "In the most minute capillaries of the brain, principally in those of the cortical substance, there collect particles of pigment which have traversed the vessels of the liver and lungs, while none have been arrested in their passage through these last-named organs. These mechanical impediments to the circulation, often occasion numerous lacerations of the vessels and capillary apoplexies."

Albuminuria is the indication of functional disturbance of the kidneys which is met with.

A host of objections present themselves to the embolism or blockade theory of pernicious intermittent fevers. Every embolism necessarily supposes an aggregate too voluminous to traverse the capillaries of organs, particularly those of the brain. But as anatomy shows that the capillaries of the brain and lungs are the smallest in the body, it follows, that the pigmentary aggregate must in the first instance encounter a material impossibility to traverse the capillaries of the lungs. There is no escape from this dilemma: the pigment either has been arrested in the pulmonary capillaries, there producing embolism, all the pigment which has traversed the liver being stopped in the capillaries of the lungs; or the pigment has found its way through the pulmonary capillaries, in which case, it must have passed through the capillaries of all the organs, because they are either as large or larger than those of the lungs.

It is, however, impossible to deny the existence of pigmentary color in the organs, and the presence of masses of pigment in their capillaries. A fact does not admit of being denied; but then this fact can be explained in a perfectly natural manner, that is to say, by the pigment being formed *in situ*, and being a consequence, not a cause. Thus, Frerichs himself admits that the pigment may be produced in any situation, even external to the capillaries. Pigmentation, then, is a physical phenomenon, arising simply from the destruction *in situ* of a certain number of globules by the stasis of the blood in the vessels. This stasis is itself a result of repeated visceral congestions, which are passive and proportionate in intensity to the intensity of the fever. The blackish color of the organs, particularly of the nervous centres, had been described by the authors who had studied pernicious fevers, particularly by Maillot and Bailly; but they attributed this color to congestion connected with the condition existing during the paroxysm. Is it not more simple to consider the "pernicious" symptoms as the effects of a sanguineous *raptus* in the nervous centres and their envelopes;* and to see in the pigmentation the physical result of a consecutive alteration of the blood *entirely cadaveric*, and which has taken place *in situ* from the local destruction of globules crammed into the capillaries distended during life? This doctrine, so simple and so naturally present to the mind, is certainly more true, or at least more like truth, than that which invokes the migration of embolia, which, as I have shown, is physically impossible.

There is also a purely clinical objection to this, viz., that the *intermittence* does not correspond with the *permanence of the lesion*. If the pigmentation be due to embolism, it ought to be persistent, for otherwise, there could be no embolism. Why should not the symptoms be as permanent as the lesion?

Finally, there is the therapeutical objection involved in the all-powerful action of preparations of cinchona which is quite inexplainable by the pigmentary theory. How, in fact, are we to understand the sulphate of quinine dispelling, as it does, the pernicious symptoms, if their proximate cause be the blocking up of the vessels of the brain, to remedy which condition cinchona has absolutely no power?

In my opinion, the pigment found in the vessels is formed in the place where it is found, and is the result of great and repeated congestions.

To sum up what I have said: I look upon pernicious intermittent fever as a general disease producing many visceral congestions, particularly in the nervous centres.

From an organic point of view, there results from these congestions sometimes an alteration *in situ* of the blood-globules (pigmentation), or, sometimes, an interstitial hemorrhage. Hence result, looking at the case from a dynamic point of view, more or less severe functional disturbance.

Let me add, in conclusion, that there may exist pigmentation of the brain without pernicious symptoms; and also, pernicious symptoms without pigmentation. Frerichs admits this, with a loyal truthfulness to science which cannot be too highly praised. He says: "If we compare the facts ascertained by microscopical examination with the symptoms observed during life, we will find, on the one hand, cases in which, notwithstanding the dingy color of the brain, there has been no cerebral disturbance, and on the other hand, cases in which there has existed disorder of the brain, although the organ was free from pigmentation." In eighty cases of cephalic intermittent fever, this occurred six times. The older observers, including Lancisi, Sénac, and Bailly,† made the same observation, which has been confirmed by Maillot* and Haspel.† It cannot, therefore, be doubted, that the cerebral symptoms already described cannot occur in intermittent fever unless melanæmia exist, and no other cause can induce these pernicious accidents.‡

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* MAILLOT: Traité des Fièvres ou Irritations Cérébro-Spinal Intermittentes. Paris, 1836.

† BAILLY: Traité Anatomico-pathologique des Fièvres Intermittentes Simples et Pernicieuses. Paris, 1825.

Gentlemen, under all the forms assumed by pernicious fevers, we find, either separately or united, three conditions, viz., pain, sanguineous congestion, and flux, characterizing *masked* fevers, of which I have now to speak to you.

Pernicious fevers—those at least which the authors of past ages called *comitatæ*—are, in fact, masked fevers [*fièvres larvées*]. You must not use the epithets *masked* and *latent* as synonymous.

A latent is in reality a hidden disease: if it be a pleurisy, for example, it does not reveal its existence by any external symptom. The patient has none of the ordinary symptoms of inflammation of the pleura: he has neither stitch in the side, cough, nor dyspnœa: and when the physician discovers the existence of the affection, it is only from the physical signs furnished by auscultation and percussion.

A masked disease [*maladie larvée*] far from concealing, very distinctly manifests, its existence; but in manifesting itself, it puts on the mask [*induit larvam*] of another disease having little or no real similarity with it.

Thus, as I have already said, pernicious fever, in its delirious form, sometimes simulates brain fever, sometimes apoplexy or epilepsy, sometimes pneumonia or pleurisy, and sometimes cholera or dysentery. However, in borrowing from these diseases some of their principal phenomena, it retains other characteristics by which it can be recognized: the former, moreover, want many essential characters which belong exclusively to the affections, the names of which, for lack of better, are employed to designate the different kinds of pernicious fevers.

Let me explain myself: The pernicious epileptiform intermittent borrows from epilepsy its convulsions; but then, the convulsions differ from those of epilepsy. In the pernicious pneumonic intermittent, the crepitant râles and sanguinolent expectoration remind one of the symptoms observed in certain forms of pneumonic catarrh; but without taking into account the appearance of the sputa, there are notable differences between the thoracic symptoms of the two affections. Pernicious dysenteric intermittent has bloody stools as a character in common with dysentery; but the tenesmus and glairy excretions, of so much diagnostic value in the latter, are wanting in the former. Observation of the progress and features of the disease enables one unhesitatingly to recognize its nature.

Though most pernicious fevers are masked fevers, it does not necessarily follow that masked fevers are pernicious: thus, the paludal diathesis very commonly masks itself as neuralgia, and under the form of certain neuroses. A woman, who lies to-day in bed 17 of our St. Bernard Ward, presents one of the most common types. This patient, aged twenty-six, tells us, that about four years ago, six months after the birth of her first child, whom she suckled, she was attacked with facial neuralgia: it particularly affected the eye, which, with each paroxysm of pain, became the seat of very acute congestion, and the source of a profuse flow of tears. The paroxysms recurred regularly every three days: they were announced by rigors, during which the pain supervened; it went on increasing during the hot stage, diminished during the sweating, and completely ceased after some hours.

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* MAILLOT: Op. cit. † HASPEL: Maladies de l'Algérie. Paris, 1850.

‡ FRERICHS: Op. cit., p. 503.

This attack of intermittent neuralgia resisted the most energetic treatment for many months. The patient was ultimately cured.

Three months afterwards, similar symptoms recurred under similar circumstances, that is, after childbirth and during lactation; but with this difference, that the neuralgia in place of being suborbital, as in the first attack, was occipital.

Though to-day we only find pain in this case, the three elements characteristic of masked intermittent, pain, fluxion, and flux, were very manifest in the first attack.

The pain was seated in one of the branches of the fifth pair of nerves: the fluxion, seated in the mucous membrane of the eye, attained a degree of congestion sufficiently violent to simulate formidable ophthalmia: the flux was a profuse secretion of tears.

The nerves of the fifth pair are those usually affected in masked fevers. The neuralgia returns at regular intervals, assuming the different types of intermittent fever, the quotidian, tertian, double tertian, quartan, double quartan, and triple quartan. Whatever be the seat of the neuralgia, whether the neuralgia be sciatica, gastralgia, or enteralgia, it follows this rule.

There are also, as I have just stated, certain neuroses which constitute masked intermittents. Among them, is *spasmodic cough*, which in some subjects exposed to marsh misasmata recurs in paroxysms daily at the same hour, without being accompanied by expectoration or any morbid pulmonary symptom, and which yields in a remarkable manner to the treatment generally efficacious in cases of a similar nature.

There is also a species of *asthma*, which likewise recurs at regular intervals, and yields to the same therapeutic indications.

Let me also mention, that these remarks are applicable to certain cases of *megrim* and *hiccough*.

There are cases in which *insomnia* recurs every two or three nights, unaccompanied by fever, and not preceded by rigors, which are evidently of the same nature, and are cured by similar treatment.

The same may be said of *periodical fluxes*, more or less profuse, from the nasal fossæ, uterus, and intestines, sometimes simply mucous, but at other times sanguinolent, unattended by any other morbid symptom, and yielding to medication similar to that which is useful in marsh fevers.

You are now in a position, gentlemen, to understand more easily the mechanism of masked pernicious fevers. You have seen the presence of pain, fluxion, and flux in neuralgia. Transfer these phenomena to another nervous apparatus, to the ganglionic nerves, or to the mixed nerves, as, for example, to the pneumogastric; and, bearing in mind the beautiful experiments of Claude Bernard,* you will at once understand the great functional disturbance which may be induced in these nerves, by morbid changes taking place in them. Suppose that there existed in the lungs or intestines, phenomena similar to those you have just seen in the eye, you would at once understand the dyspnœa, crepitant râles, and expectoration, or the pain in the intestines, the excessive flux from the gastro-intestinal mucous membrane and glands which discharge their secretions upon it. Transfer to the brain or spinal marrow the element of pain and fluxion which we have just been considering in the suborbital nerve, and see whether you will not have nervous phenomena depending upon the pain itself, or upon the fluxion, such as delirium, convulsions, stupor, and coma.

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* BERNARD (Claude): Leçons sur la Physiologie et la Physiologie du Système Nerveux. Paris, 1858.

We may in this way pretty easily explain the very varied symptoms of both simple or pernicious masked fevers: in this way, confirmation is given to an opinion already held by others, and in which I completely concur; viz., that intermittent fevers, under whatever forms they may present themselves, ought to be classed with the neuroses.

There are numerous reasons, gentlemen, for adopting this view. The suddenness of the invasion of the disease, and its rapid disappearance—the violence and likewise the fleeting character of the symptoms which characterize it—the terrible disorder which supervenes throughout the whole economy, a disorder which, in pernicious fevers, cannot recur without greatly endangering the life of the patient. Then, there is the deceitful feeling of security during the interval between even the most violent paroxysms, the wonderful facility in mastering a disease manifesting itself in such frightful forms. Do not all these circumstances, gentlemen, lead us to adopt the idea that it is a neurosis? Then, again, when we see the most common pernicious masked fever generally accompanied by symptoms which even the most incredulous are forced to regard as nervous. When in these same pernicious fevers, it is so easy to take account of the symptoms from the disorder which they occasion in the nervous centres or ganglionic nerves, we are compelled to regard all forms of intermittent fever as so many different forms of neurotic disease.

When a neuralgia is intermittent, we must not conclude that it is necessarily a masked fever. In summer, for example, we not unfrequently meet with cases of violent neuralgia in which the attack begins every morning and ceases every evening, recurring in this manner for six, seven, or eight days in succession. This kind of neuralgia—*solar neuralgia*—is met with in the most salubrious countries, wholly irrespective of any influence of paludal miasmata.

When intermittence is the predominant character of a masked fever, it is important, with a view to the diagnosis, to inquire into the antecedents of the patient. Intermittence is very common in localities where marsh fevers are endemic: elsewhere, it is of rare occurrence; and in Paris, it is very seldom met with, except in individuals who have lived for some time in marshy countries, whether they have or have not had regular intermittent fevers.

The fact that the patient has sojourned at some former period, more or less distant from the appearance of the symptoms, in a marshy country, renders it probable that the disease is intermittent fever; and this presumptive diagnosis becomes almost a certainty, if there exist engorgement of the spleen, along with that peculiar tint of skin which indicates a state of cachexia. The nature of the disease does not admit of any doubt when it yields very readily to the treatment which is successful in paludal affections.

Treatment by Cinchona (according to the Roman, English, and French systems) and by Arsenic, and the Method of Dr. Boudin.

Gentlemen: I have still to speak to you on the subject of treatment. I need not tell you that the basis of the treatment consists in administering cinchona, and its derivatives, quinine and the sulphate of quinine. Every one knows, that by means of these precious remedies, intermittent fevers are cut short. But every one does not know, that to cut and to cure an intermittent fever are not synonymous terms: this is a fact with which many physicians even do not seem to be acquainted.

It is necessary to administer cinchona according to a plan, so as to obtain the expected results. In my opinion, the best system to follow is that which has been lauded by Bretonneau, and is called the *French method*—a method which I have endeavored to improve; and which is a happy combination of the methods of Torti and Sydenham.

Torti's method, likewise called the *Roman method* (from its having been first adopted by the Jesuits of Rome, who derived it from their brethren of Lima), consists in giving the cinchona, immediately before the paroxysm, in one large dose.

This method has inconveniences which were fully pointed out by Sydenham. When administered immediately before the paroxysm, it is often vomited: this inconvenience was recognized by Torti himself, who, to avoid it, sometimes gave the medicine after the paroxysm. On the other hand, as has been shown by the experiments of Bretonneau, the impending paroxysm which it is intended to stop, is often rendered more violent and painful by the cinchona.

To avoid these inconveniences, Sydenham and Morton directed the cinchona to be given at the longest possible interval before the anticipated paroxysm, commencing the remedy, consequently, as soon as possible after the termination of the preceding paroxysm. The following is Sydenham's formula:

Take of powder of cinchona, 32 grammes;
Syrup of roses and pinks, q. s.

This electuary is divided into twelve doses, one of which the patient is directed to take every four hours, commencing at the close of the paroxysm.

He was also in the habit of prescribing a wine of cinchona containing thirty-two grammes of the powder in two pounds of common red wine.

He ordered the patient to take, in a manner similar to that now described, from eight to nine tablespoonfuls of this preparation.

You have seen me on several occasions, and recently in a case of obstinate quartan fever, have recourse to an electuary very similar to that of Sydenham, the only difference being, that, in place of the syrup of roses and pinks, I used the conserve of roses, or the syrup of bitter oranges, which latter I prefer, as it has the advantage of masking the disagreeable taste of the cinchona.

It is undoubtedly an important rule to give the cinchona at a period as long as possible before the coming paroxysm. The reason is very simple. The active principle of the remedy being neither volatile nor diffusible, is absorbed slowly, and requires a certain time to produce an effect upon the economy. When the quantity administered is not more than an ordinary dose, this time is at least eighteen or twenty-four hours: large doses take effect in from six to twelve hours.

Hence it follows, that the best method of treatment consists in employing a large dose of cinchona divided into three or four portions, giving these portions at very short intervals, and commencing the administration as long as possible before the coming paroxysm is due.

This, which is Bretonneau's method, is a happy combination, as I have already remarked to you, of the methods of Sydenham and Torti; and is that which you have seen me employ. By following it, you will obtain results much more complete and sure, from far smaller quantities of cinchona than would otherwise be required. Fifteen grammes of cinchona administered at once are generally sufficient to suppress a paroxysm of regular intermittent fever; but thirty grammes, that is to say, twice the quantity, given within five or six days in the apyrexial intervals, will fail to produce that effect. We must not, however, adopt to the letter the precept of Torti and Bretonneau. By a single dose [*une seule dose*] we must understand that the entire quantity of cinchona prescribed is to be taken within a very short space of time, within one, two, or three hours at most; for some patients cannot easily tolerate at once fifteen grammes of cinchona, or even eight grammes, the dose of the powder recommended by Torti,* and that which I usually prescribe. This remark is equally applicable to the sulphate of quinine.

In simple legitimate intermittents, of which alone I am now speaking, I prefer powder of cinchona to the sulphate of quinine.

It has not been proved to my satisfaction that quinine (still less its salts, such as the sulphate) possess all the virtues of the bark of cinchona: on the contrary, experience has taught me that, although the bark acts more slowly, it acts more surely and more thoroughly; if I may so express the fact, that the salutary effects are more lasting. Does this arise from the bark containing in addition to that which we regard as its active principle, other principles with which we are not acquainted? Does this depend upon the crude drug yielding slowly its quinine, so as to admit of its being assimilated, while this is not the case with the sulphate of quinine, a part, the greater part of which, perhaps, is eliminated with the urine? These are questions which I cannot answer. Nevertheless, experience has proved the correctness of the statement I have now made.

Moreover, for economical reasons, which may perhaps appear trivial to some savans, one superiority of the bark will be appreciated by physicians, particularly by those whose practice lies in the country among the comparatively poor, to whom greater cheapness will render the bark very preferable to the sulphate of quinine.

When it is necessary to give daily as much as a gramme of the sulphate of quinine to suppress a paroxysm of intermittent fever, similar results may be obtained, and that, too, with more certainty, from eight grammes of cinchona, that is to say, from a dose only one-fifth the price of the equivalent dose of sulphate of quinine. In a word, then, when I wish to cut short a regular intermittent fever, I give, immediately after the termination of the paroxysm, eight grammes [124 grains] of the powder of yellow cinchona, either at once, or in two portions at the interval of half an hour, in a cup of tea or coffee.

This dose rarely fails to prevent the recurrence of the impending paroxysm. But although the disease is arrested, it is not cured, the arrest being only temporary, and the patient retaining some slight reminders of his malady, such as an increase of temperature with feelings of general discomfort, or—and this is more usual—profuse sweating recurring on the days upon which the paroxysm was due. If the medication be abruptly discontinued, if the patient do not return to the use of the quinine, the symptoms soon reappear, first feebly, and in a less decided form, but ere long with all their precise and positive characteristics. It is, therefore, important, even though there be no immediate return of the symptoms, to continue to give the febrifuge in the same doses, at the determinate intervals, for a period prolonged more or less according to the duration of the malady, according to its type, and particularly according to the patient being or not being resident where he has contracted the fever.

Torti, in simple intermittent fevers, gave eight grammes of the powder of cinchona immediately before the paroxysm was expected to begin: and he continued the administration of the medicine till the patient found himself quite well: day by day, however, he diminished the dose. This method of administration, adopted by Torti from the Roman Jesuits, and for that reason designated the Roman method or the method of the fathers, is often excellent in the case of patients who have ceased to reside in the localities where intermittent fever is endemic, and who have not been long subject to recurrence of the paroxysms. It saves the patient from attacks of fever for ten or fifteen days, or even, sometimes, for a longer period; but it has not the power to prevent relapses.

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* TORTI: Therapeutice Specialis ad Febres Periodicas Perniciosas.

Sydenham's method, called the English method, has a much greater power. Sydenham, as I have already stated, begun by giving immediately after the termination of the paroxysm thirty-two grammes [496 grains] of the powder of cinchona, which he gave in doses distributed at nearly equal intervals between the past and coming paroxysms. Eight or fourteen days later, according to the type of the fever, he repeated the same dose: he likewise recurred several times to the same medication, particularly if the patient had long had the fever, and had suffered in constitution from paludal influence. This method is much more efficacious than that of Torti: it is much more protective from relapses: but it has its inconveniences, which I must now state.

Many patients cannot take so large a quantity of powder of cinchona without having vomiting, and still less without diarrhœa being induced. Then again, patients who have been long subject to the fever, have a recurrence of the paroxysm, after the lapse of some days, and before the sort of sacramental epoch fixed by Sydenham for the readministration of cinchona. One may, it is true, ward off the first of these inconveniences by giving a small quantity of opium along with the febrifuge powder; but the objection to the method remains undiminished when the patient is deeply imbued with paludal cachexia.

Gentlemen, recall to your recollection the woman who came from Guadaloupe with tertian fever of more than six months' duration. Sydenham's doses, to which were always added a little laudanum, cut short the malady and gave the patient a respite from attacks of fever for five or six days; but between the sixth to the seventh day, a new paroxysm announced to us, that it was the paroxysm and not the patient that had been cured. It then became necessary to resort to the *method of Bretonneau*, the *French method*, which I have modified to a certain extent, and in a manner which my illustrious master has himself adopted.

Bretonneau found that Sydenham's daily quantities were too large: that when there was involved in the question of dose the cost of a remedy so expensive as cinchona, or, and still more, the sulphate of quinine—and also, when it was remembered that intermittent fever was specially a scourge of the poorer classes,—the money value of the dose was a matter of great importance. He had learned from experience that Torti's dose of eight grammes was sufficient, provided it were administered at the longest possible interval prior to the coming attack, either all at once, or divided into two portions taken with an interval between the times of not more than some hours. Experience had likewise taught him, that by daily administering decreasing doses, according to Torti's plan, relapse was almost inevitable. In respect of dose, therefore, he adopted the Roman method: in respect of mode of administration, he adopted the method of Sydenham, that is to say, he administered the febrifuge immediately after the paroxysm, renewing the administration, *always in similar doses*, at longer or shorter intervals, and extending over a considerable period of time, according to the rule of Sydenham.

Bretonneau's method, or the *French method*, as it is called, consists in giving eight grammes of the powder of yellow cinchona bark or one gramme

[15½ grains] of the sulphate of quinine, in a single dose, or in two doses with a very short interval between them, as long as possible before the coming, that is to say, immediately after the past paroxysm. Five days of rest are allowed to pass, when the same dose of the remedy is again prescribed; and then, it is repeated once in eight days for a month.

When the fever has been of very long standing, it is necessary to continue the medicine for a very long time, augmenting the dose if necessary, as the treatment proceeds. From the second month of the treatment, the cinchona is taken at successive intervals of ten, fifteen, twenty-five, or thirty days; and according to such a plan as will secure the patient against relapses, a result obtained with much less certainty when Sydenham's method is rigorously followed.

During three years which I passed at the hospital of Tours, following Bretonneau's clinic, I only saw once this method fail to cure intermittent fever. However, since I have been at the head of a service in a Parisian hospital, I have repeatedly failed to cut short perfectly regular intermittent fevers, though I employed exactly the formulæ of my illustrious master. The first paroxysm following the administration of the cinchona was postponed, much diminished in severity, or sometimes even suppressed, but the second, or at least the third paroxysm, reappeared in a more or less modified form: here then was a sufficiently serious inconvenience. I avoided it by adopting the method which I have pointed out to you.

Immediately after the termination of the paroxysm, I cause the patient to take eight grammes of the yellow bark, or one gramme of good sulphate of quinine, either in one dose, or in two doses with an interval of one or two hours between them. For a day, I leave the patient without medicine; and on the third day, I give him the third dose of the medicine, administering it either all at once, or at twice, the taking of the second rapidly following the taking of the first portion of the dose. I then allow an interval of three days, four, five, six, seven, or eight days, and finally, for one or two months, I recur every eight days to the same medication, *never diminishing the dose*. Let me add—and the fact is important—that the medicine ought always to be given at a meal.

Bretonneau's method and my own are in reality only modifications of the method of Sydenham.

Gentlemen, you have sometimes witnessed in our wards, the failure of a treatment of which I have just now been proclaiming the infallibility.

You can recall to your recollection the young men who returned to France, re-entering civil life after having made the Crimean, African, and Italian campaigns, who when admitted to our wards, suffered from dreadful cachexia, enormous engorgement of the spleen, and infiltration of the extremities. They refused to take preparations of cinchona, because, as they said, cinchona had done them harm, and had not cured them. I have frequently called your attention to these patients, requesting you to mark the happy influence of the medication which I was about to institute. These are the circumstances under which Sydenhamian doses ought to be unhesitatingly recommended: these are the cases in which the powder of cinchona resumes its old claim to superiority, and leaves far behind it the sulphate of quinine: these are the cases in which my method, scrupulously followed, and aided by the use of ferruginous medicines, produces results so evident as to astonish and convert even the most incredulous.

Very recently, you have seen me treat in a similar manner a man who occupied bed 9 in St. Agnes Ward. In that case, I began by giving an emetic and the purgative decoction of cinchona (of which I shall have forthwith to speak to you): next day, between the two paroxysms, I made him take thirty grammes of Sydenham's electuary (the formula for which I have given you), taking care to administer a drop of laudanum with each dose, so as to prevent vomiting and particularly diarrhœa. I recurred to the same dose, in the first instance after one day, then after three, four, five, six, seven, or eight days of interval, prescribing always thirty grammes of cinchona, with also, at each meal, a tablespoonful of the syrup of the ammonio-citrate of iron. You have seen that during the six weeks of the patient's residence in hospital, he has not had the slightest feeling of a return of the fever, that the spleen has rapidly diminished in size, that his color has been natural, and the digestive functions have been re-established in their normal state: you have also seen that this man, so exceedingly dispirited, so distrustful of the curative powers of the preparations of cinchona, obtained a rapid cure, which was due as much, or perhaps more, to the method of employing the remedy as to the remedy itself.

If the most intelligent administration of cinchona be abruptly abandoned after some days, the fever returns: under such circumstances, it is necessary to begin the treatment exactly as it was undertaken in the first instance.

When, in accordance with other methods of administration, a small dose of cinchona or sulphate of quinine is given daily, the fever is modified and is sometimes cured; but the cure is accomplished with more difficulty, and less certainty: acute gastric pains soon supervene, under whatever form the remedy is administered. If under such circumstances, the fever should reappear, it is no longer possible to cure it. If large doses are repeated daily for a long period, there is manifested (besides the pains in the stomach), a peculiar kind of fever, described by Bretonneau, and which assumes an intermittent type when the cinchona is given in an intermittent manner. This fever forms a sort of vicious circle within which inexperienced physicians revolve: ignorant of the action of the cinchona, they redouble their doses, and throw the patient into a state which may be one of great gravity.

There is another inconvenience; it results from habit [*accoutumance*] if I may be allowed to employ that old term. The patients, from constantly taking cinchona, become at last insensible to its action, and the fever is renewed notwithstanding the daily administration of the remedy.

The methods of Sydenham, and Bretonneau, as well as my own method, are not liable to these inconveniences.

Though the accidents which I have now been pointing out to you may be imputed to cinchona, we cannot blame it for causing engorgement of the spleen and the consecutive dropsical affections, though, in the period immediately subsequent to the discovery of the Peruvian bark, these evil effects were attributed to it. Notwithstanding the renewal in our day of this old controversy, the matter is now definitively settled, it being agreed, that this valuable remedy has no part in producing the organic lesions in question, which are the result of a poisoned saturation of the system.

I am no believer, however, in the wonderful and sudden manifestations of the virtues of sulphate of quinine, which, according to some persons, reduces the volume of the spleen within a few minutes of its administration. I have shown you a young woman stricken by paludal fever contracted in Guadaloupe, in whom the spleen was of enormous size, and in whom the abdominal walls were so attenuated that the entire outline of the engorged viscus could be felt. This case presented a very valuable opportunity to try the sudden action on the spleen of the preparations of cinchona: it did not necessitate recourse to the artifices of percussion, which only deceive beginners. Now, in this woman, the spleen not only did not instantaneously diminish under the influence of the remedy, but *for the first* *twelve hours it increased in volume*, rapidly diminishing during the following days. You all had an opportunity of observing this fact.

By what channels is cinchona introduced into the system? Generally, it is administered by the mouth; but there are circumstances in which this mode has to be relinquished.

Some patients have an absolute repugnance to swallow cinchona, whatever means may be taken to mask its bitter taste: there are others, particularly young children, in whom it produces vomiting as soon as it is swallowed. Administration by the mouth is also ineligible in those cases in which a gastritis or a severe gastralgia has been induced by the long-continued use of potions and powders containing preparations of cinchona. Then, again, in certain pernicious fevers—in which, as I shall immediately explain to you, it is important to act promptly—in certain pernicious fevers, in cardialgia, and in cholera, the vomiting characteristic of the disease makes it sometimes impossible to administer by the mouth the smallest dose of cinchona or sulphate of quinine.

Under such circumstances, it becomes necessary to introduce the remedy by some other way; and the rectum, affording the greatest facility, is the way chosen. The doses of cinchona given by enema ought to be a little smaller than those prescribed to be taken in potions, because absorption takes place more rapidly and better in the large intestine than in the stomach. Should it be found that the rectum retains the cinchona badly, the doses must be repeated in such a manner as to insure absorption of the requisite quantity. Should the intestine ultimately become too irritable, the *endermic method* must be employed. The simplest proceeding which can be recommended consists in employing poultices made with wine and powder of cinchona. These poultices ought to be very large, and must be kept applied to the abdomen for eight or ten hours. I should not be inclined to rely on their efficacy.

To render the cutaneous absorption of the remedy more active, M. Lembert has proposed to make the application to the skin after having removed the epidermis. Crude cinchona ought hardly ever to be employed in this way; but there is no objection to applying the sulphate of quinine to the denuded dermis; and it has been said, that by its application in this manner, intermittent fever may be cured with as much certainty as if the medicine were administered by the mouth or rectum. Certain precautions have to be taken. Dr. Briquet's experiments* show, that a solution of sulphate of quinine applied to a blistered surface only produces very slight pricking and a little local irritation, while in the state of powder, it occasions acute smarting, or pain more or less intense; and that if the powder be applied several days, in succession, it may act as a caustic, causing the formation of a slough, and consecutive ulceration. This I mentioned long ago in my Traité de Thérapeutique.

At the commencement of the treatment, particularly in the intermittent fevers of spring and autumn, I generally give an emetic, and often even a purgative, combined with cinchona according to the following formula:

> Boil fifteen grammes of yellow cinchona bark in three hundred grammes of water, so as to reduce the quantity two hundred and fifty grammes: in this, dissolve twenty-five grammes of the sulphate of soda.

The patient is ordered to take this potion in two draughts, leaving an interval of half an hour between them.

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* BRIQUET: Recherches Expérimentales sur les Propriétés de Quinquina et de ses Composés 2me édition, 1855.

I have now, gentlemen, explained at great length the regular method of treating legitimate intermittent fevers; and it now remains for me to tell you how to combat *pernicious fevers.*

We are indebted to Morton for having formulated the beneficial effect of cinchona in these fevers. He did not, however, point out the method by which we may almost always triumph; and Torti was the first to lay down sure rules for the treatment of these formidable diseases. Abandoning Morton's plan of giving four grammes of the cinchona every three or four hours (in every respect a faulty proceeding, at least when we have to do with a pernicious quartan, which has a long apyrexial period), Torti shows, that in pernicious subintrants, or in those which are really remittents, it is necessary to gain time by giving the cinchona in doses thrice as large as those administered in simple intermittent fevers.

Consequently, he gave the patient, in one dose, from fifteen to twenty-four grammes of cinchona, taking care that the medicine was taken as long as possible before the next paroxysm. He did not give the remedy during the intermission (because there is seldom a complete intermission in pernicious fevers), but at the time when the previous paroxysm begins somewhat to subside—in a word at the beginning of the period of remission.

This method, though infinitely superior to that of Morton, is by no means faultless. It cannot be denied that in fevers which are tertian, pernicious, or subintrant, the interval between the remission from the preceding and the onset of the following paroxysm is often too short to allow the cinchona to be sufficiently absorbed to act usefully.

Bretonneau, impressed with the weight of this objection, modified Torti's method of treating pernicious fevers, in the same way that he had modified Torti's method of treating simple intermittent fevers. He prescribed that *the administration of the cinchona should commence the moment that the pernicious character of the fever was ascertained, even though it should be in the middle of a paroxysm.* By pursuing this plan, we can manage to obtain at least from twenty-four to thirty-six hours before the following attack begins, and so be able to prevent it. In thus giving the cinchona during the paroxysm, we must not be afraid of augmenting its intensity, for the medicine does not act till some hours after it has been administered, and, therefore, not till the remission is about to commence. Inasmuch as we have before us a space of time relatively pretty long, we are not obliged to give all at once so large a dose as that advised by Torti. Bretonneau recommends twelve grammes to be given at once, and the same quantity to be repeated every three hours till the patient has taken thirty-five grammes of the powder of cinchona.

This method is certainly superior to that of Torti; but while I adopt Torti's rule as to the time of administering the remedy, I think that here the sulphate of quinine is very much to be preferred to the powder of cinchona. In a disease, in fact, in which the most terrible accidents are imminent, in which it is often a question of life or death not to allow them to gain upon us, it is necessary, with all possible speed, to put the patient into a condition to sustain the shock and resist its effects. Now, as I have already stated, the cinchona yields its active principles too slowly, whereas the sulphate, particularly the bisulphate, of quinine is rapidly absorbed.

When you have to treat a case of pernicious intermittent fever, you must, therefore, administer to the patient, with the least possible delay, a large dose of the salt, say two or three grammes, in potion; or, if the stomach will not tolerate the potion, give it in enema: this dose must be repeated for five or six consecutive days, till the symptoms are arrested.

As soon as the danger is quite passed, it ceases to be necessary to give the remedy in such high doses. You then, resuming the medication suitable in simple intermittent fever, according to the method which I have pointed out, give cinchona in preference to sulphate of quinine, and continue it for one or two months—eight grammes every morning.

Simple *masked fevers*—neuralgic affections and neuroses masking marsh fever—demand a special treatment, in this sense, that the disease being out of its usual ways, is more difficult to suppress. Though the indications are the same in these cases as in ordinary intermittent fevers, in respect of the medication to be employed, that medication must be more active, and it is specially necessary to continue it for a longer period. Thus, the doses of cinchona or sulphate of quinine, which must be administered immediately after the paroxysm, require to be stronger than those employed to cut short a regular intermittent; and it will often be necessary to continue them for five or six days in succession so as to put an end to the symptoms. When they have been got rid of, it will still be requisite to persevere for a long time in the use of the same means, observing always the rules which I have laid down for your guidance.

Hitherto I have only spoken of cinchona and the sulphate of quinine; but there are other substances derived from the bark, and one in particular, which merit special notice. The substances to which I refer are *cinchonine* and its salts, particularly the *sulphate of cinchonine;* they unquestionably possess the febrifuge properties of the preparations of quinine, but in a very inferior degree. To attain the same results from their use, it is necessary to give them in doses twice as large. There is also *quinium*, or the alcoholic extract of cinchona (prepared by the lime process),* a medicine recently introduced into the materia medica by MM. Delondre and Labarraque, a substance which differs little from crude quinine, to which I must now briefly call your attention.

Crude quinine [*quinine brute*], as much a febrifuge as sulphate of quinine, has certain advantages over it, with which it is important that you should be made acquainted.

Its tastelessness renders it peculiarly valuable in the treatment of children, as it can with the greatest ease be administered without its being perceived, while it is impossible to disguise the bitterness of the sulphate of quinine. It is of resinous consistence, and softens with the heat of the fingers in such a way that it can be reduced into the form of exceedingly minute pills, which can be easily swallowed in soup or preserves. Its dose is the same as that of sulphate of quinine.

I have not said anything to you regarding the *succedanea of cinchona;* nor should I now refer to the subject had I only to mention alleged specifics, such as salicine, olive, alkekenge, and sea-salt, for they do not merit detailed notice; but *arsenic* is a remedy which does not belong to this category. It was long ago employed in the treatment of intermittent fevers; and has been reinstated in its place of honor by my lamented confrère, Dr. Ch. M. Boudin,† whose successes have been confirmed by the successes of many other practitioners, who have been zealous in making them known through the medical press.

Such of you as wish for information regarding the history of this method of treatment, I refer to a chapter which Dr. Pidoux and I have dedicated to that subject in our treatise "On Therapeutics."‡ For the present, I shall confine myself to a statement of the rules which Dr. Boudin has laid down for the administration of his lauded remedy.

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* See the Bulletin de l'Académie de Médecine. Paris, 1857, t. xxii, p. 1008.

† BOUDIN: Traité des Fièvres Intermittentes, Rémittentes, et Continues des pays chauds et des contrées marécagneuses, et de leur traitement par les préparations arsénicales. Paris, 1852.

‡ Second volume, p. 366, of the seventh edition.

The following is the substance of his rules: To begin the treatment by prescribing an emetic consisting of one gramme of ipecacuan, and ten centigrammes of tartar emetic, with a view to combat the concomitant gastric disturbance, and the absence or diminution of appetite; then to give the arsenious acid in frequent small doses, taking care that the last of them is administered two hours at least before the time at which the paroxysm is expected; to proportion the dose to the special character of the fever, which varies with the locality, the season, and the individual.

The dose is a milligramme, or half a milligramme, of arsenious acid, that is, one gramme or half a gramme of the following solution:

Arsenious acid, ...	1 gramme.
Distilled water, ...	1000 grammes.

As a precaution, it is indispensable to boil this solution for a quarter of an hour.

Fifty grammes of this solution represent five centigrammes of arsenious acid. The desired dose is mixed with an equal quantity of wine, infusion of coffee, or water.

This medicine may be administered equally well during the apyrexial day and the day of the paroxysm; the medication ought to be continued during a period proportionate to the time the disease has existed, and the greater or less degree in which it has resisted anterior treatment. In fevers of the first invasion, it ought to be continued for at least eight days after entire cessation of the paroxysms. In old and obstinate fevers, it is often necessary to prolong the use of the arsenious acid for thirty, forty, or fifty days, or even for a longer period.

The tolerance for the remedy is very variable in different individuals, and it likewise varies in the same person, diminishing for a time, and then becoming re-established as before. A particular patient, who at the beginning of the treatment bore very well five centigrammes of arsenious acid, is unable to tolerate this dose two or three days afterwards, when the paroxysm is cut. The signs of this intolerance are nausea, headache, and impaired appetite; or the manifestations of intolerance may occur in a higher degree—in vomiting and diarrhœa.

It is necessary to follow attentively these oscillations of tolerance, so as to diminish doses with diminution of tolerance. It often happens that we are obliged to give the medicine by the rectum, by which mode of administration, the patient supports five, ten, or even twenty centigrammes of arsenious acid, when unable to bear one centigramme by the stomach.

From diversity of idiosyncrasy, it is very difficult to determine beforehand the doses which will be required to effect a cure. Dr. Boudin has often cut short a fever by one milligramme: in other cases, he has found it necessary to raise the dose to five centigrammes or more within the twenty-four hours.

One of the essential conditions of this treatment, is to keep the patient upon a diet as substantial and abundant as possible, the only limits being his appetite and digestive power.

You perceive that Dr. Boudin's treatment does not consist in substituting arsenic for cinchona, but in carrying out a complex medication, in which arsenic sets up an arsenical in opposition to the paludal diathesis, and is seconded by two powerful therapeutic agencies, viz., emetics given to combat the gastric disturbance, and accelerate a restoration of appetite, and an alimentation which will shorten the period of convalescence, combat the tendency to relapses, and prevent many *consecutive accidents apparently connected with an impoverished state of the blood.*

In the treatment by cinchona, the accidents are combated by the cinchona, with which we alternate ferruginous preparations. In cases in which there is paludal cachexia, certain mineral waters, particularly those of Pougues, contribute usefully to the cure.

Under the influence of the arsenical treatment, as also under the influence of the other medications which I have brought under your notice—the preparations of iron, cinchona, and alkaline ferruginous waters—engorgements of the spleen, and other visceral engorgements disappear. Moreover, experience convinced Dr. Boudin that relapses occur much less frequently in individuals who have been treated by arsenic, than in those treated by sulphate of quinine. It remains to be ascertained whether the treatment by sulphate of quinine was carried out according to the good method which I have been teaching you, in the cases thus brought into comparison by Dr. Boudin.

Arsenic, like quinine, will be found an excellent prophylactic. As such, Dr. Boudin recommends it to be given in very small doses daily of about one milligramme.

Let me remind you, gentlemen, in conclusion, that in recent times alcoholic liquors have been announced by Dr. J. Guyon as excellent succedanea for cinchona. I can give you no decided opinion as to the value of this kind of medication, which I have never had occasion to try. I may state, however, that one of my hospital colleagues, Dr. Hérard, physician to the Lariboisière hospital, tells me, that he has obtained wonderful results from it, particularly in a man suffering from African fever. A glass of pure rum, administered at the beginning of the paroxysm, immediately subdued the shivering and cut short the symptoms. In a woman, however, who had contracted intermittent fever in one of the departments of France where such fevers are endemic, a similar experiment completely failed.

LECTURE LXXXVI.

RICKETS.

History.—Age at which Rickets usually shows itself.—General Appearance of the Patient.—The Disproportion between the Size of the Head and the Smallness of the Stature must not be confounded with what is seen in Hydrocephalic Persons.—Rachitic Deformities.—Order in which they occur.—Mechanism of their Production.—Fractures.—Anatomy and Physiological Pathology of Osseous Lesions.—Three Periods: Period of Fluxion and Effusion; Period of Softening and Transformation; Period of Reconstitution and Consolidation.—A Fourth, Consumption, may replace the Third Period.

GENTLEMEN: Towards the middle of the 17th century, in the year 1630, the English mortuary tables make mention, for the first time, of a disease which the oldest inhabitants and the oldest practitioners of England did not remember to have seen prior to that date. It was called by non-professional persons "the rickets," a term probably derived from "*riquets*," a word in which the Norman idiom of that period was applied to persons humpbacked or otherwise deformed.

Immediately afterwards, cases presented themselves in such numbers as to attract general attention. Several physicians who had had the best opportunity of studying them, met to communicate to one another the results of their observations; subsequently three of the number, Glisson, Bate, and Regemorter, were intrusted with the classification of the documents which had been collected. Afterwards, to give greater unity to the editing of the reports, Glisson undertook the task of writing the history of this singular disease, regarding which no trace could be found in the authors of the time, by the most attentive and erudite research. I cannot give the date of the first edition of Glisson's book; but I know that the second edition appeared in 1650. The work, which is written in Latin, is entitled "De Rachitide;" and *rachitis* is the translation into French of the name of the disease.

It matters little whether the name has or has not any claim to a scientific origin, whether Glisson, as has been said, struck with the deformity of the vertebral column in the majority of subjects, went to the Greek in search of his etymology—νόσος τῆς ῥάχεως (disease of the rachis) out of which he made ῥαχέτις, just as from νόσος τῶν πλευρῶν (disease of the pleuræ) is formed πλευρέτις pleurisy—or whether the term was adopted simply to recall the vulgar appellation, *rickets*. The name is good, and ought to be preserved; for, according to the judicious remark of Van Swieten, "*Satis distinctum est abaliorum symptomatum et morborum nominibus, simulque satis facile pronunciatu est, nec difficulter memoria retinendum.*"

It has long been, and is now a question of discussion, whether this disease which all at once made so much noise in England was really as new as was supposed by Glisson and his fellow-workers. Van Swieten—who ought always to be consulted when we have to clear up an obscure point—adopts the views of the English physicians, devoting a long commentary to the controversy. A few years ago Dr. Beylard, one of my old *chefs de clinique*, reviewing numerous works prior to his own, comes to an entirely different conclusion. Without at all questioning the merit which pertains to Glisson, he says that the disease was well known before that physician's day; and he goes back to Hippocrates and Galen (without whom it appears a history of rickets would be incomplete) for allusions to rickets. Then, skipping over ages, and coming down to the seventeenth century, Dr. Beylard cites Whistler, a German, as the author of a memoir on the same subject, and of earlier date than Glisson's work. It is true that this memoir was printed at Leyden in 1645, five years consequently before the second edition of the English physician's treatise; but it is allowable to suppose that his first edition was published anterior to the memoir of the German, and might even have been known to him.

Gentlemen, as you can understand, it is not easy to exhaust discussions of this kind. If rickets have always existed, it is astonishing that a disease so strikingly evident, and so well deserving of the attention of physicians, should not have been the subject of more detailed notices than those accidentally met with in the writings of past centuries. However, once the alert was sounded, rickets, or the *morbus anglicus*, as it was called on this side of the Channel, was soon pointed out in the different countries of Europe. In Germany it was described under the name of *articuli duplicati*: in France it was vulgarly called *chartre*, an old French word synonymous with *prison* (Lat. *castrum*), and which conveys the idea of the patients being imprisoned in an affection depriving them of the liberty of movement:

it is also called *nouure des jointures;* speaking of rachitic subjects, they are said to be tied up [*noués*], an expression still current.

Whether rickets was or was not a new disease in the time of Glisson, whether he was or was not the first to speak of it, he had not the less the undoubted merit of having described it with such accuracy as to leave very little to be added to complete his picture.

Since Glisson wrote his work on rickets, numerous works on it have appeared. You will not ask me to give you a list of them. I shall only mention the authors of the more remarkable: among the older authors, are Duverney, J. L. Petit, and Levacher de la Feutrie: among the modern, are Rufz, Jules Guérin, and finally Dr. Beylard, who has, in his thesis, given the most complete analysis of the subject which we possess.*

Having established these preliminary points, I now proceed to the history of rickets, examples of which occur in several children now in St. Bernard Ward.

Besides these living examples, I place before you plates taken from Dr. Beylard's thesis, which are faithful representatives of individuals, almost all of whom I saw. I will also show you the skeleton of a young rachitic girl, who died in my wards of the Hôpital des Enfans, so that you may be able to form as exact a conception as possible of the effects produced by the disease, and that you may the more easily fix in your minds the views which I shall now endeavor to present to you.

Rickets is a disease of early infancy: it generally supervenes at the epoch of dentition, that is to say, towards the end of the first year of life, or during the first six months of the second year. All authors agree on this point; but all likewise concur in admitting that, in exceptional cases, it shows itself both before and after the period now indicated, and that instances occur in which it is congenital. Without at present speaking of osteomalacia, the rickets of adults and aged persons, to which I shall devote some special paragraphs, the two cases, pictures of which I now show you, are examples of rickets supervening in the second period of childhood. Such cases are of very rare occurrence; for in 346 cases collected by Dr. Jules Guérin, he found only five in which the disease was developed in persons between five and twelve years of age. Make particular note, gentlemen, that the only question is as to the epoch at which the disease declares itself; for when rickets is arrested in its progress after having acquired a certain intensity, the lesions which it has produced often remain, and are incurable, continuing for life, so that the unfortunate sufferers retain horrible deformities, the marks of the malady from which they have been long free, just as a person who has had Pott's vertebral disease, will retain an irremediable gibbosity, although the caries which destroyed the vertebræ had been arrested for a long time, and the bones had even become partially restored.

Upon looking at a young rachitic child, one is struck with its attitude, its physiognomy, and the disproportion between its stature and the size of its head.

While a child of the same age, whether in its cradle or in its mother's arms, sits up willingly, and likes to move about its little limbs, the behavior of the rachitic child is very different. It always keeps the recumbent position, whether in its bed or whether it be held in the arms. Whenever an attempt is made to change this position, should it even be to give it the breast when it is very hungry, it utters plaintive cries, as if it dreaded the pains which the pressure of the hands was about to occasion, and of which the experience of the past is remembered. The child suffers; and is not pacified till gently and fittingly held in the nurse's arms. When one attempts to make it stand up, it complains even more, and avoids supporting itself on its legs, so great is its debility, and so exceedingly sensitive are all its members.

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* Rufz: Gazette Médicale de Paris, 1834.
Guérin (Jules): Mémoire sur le Rachitis, 1839.
Beylard: Du Rachitis, de la Fragilité des Os, de l'Ostéomalacie. Thèse de Paris, 1852.

Were we to judge by stature, we should suppose it younger than it really is. In its huddled up state, at two years, it hardly looks more than six months old. The little boy, whose picture I now show you, is twelve years of age, and he is not one metre in stature, or, in other words, has not attained the height of a child of three years.

Along with the diminutive height, there is a head of abnormal bulk. The forehead projects. The coronal and parietal protuberances are prominent. The top of the cranium is flattened in consequence of the separation of the parietal from the temporal bones, and the retroversion of the occipital bone. The fontanelles are abnormally open. This fact—*the persistence of the fontanelles*—has, in my opinion, a great significance. When I find this condition in a child at or above two years of age, I look upon it as a feature characteristic of rickets, even though no other sign be present.

As you are aware, gentlemen, at birth, the cranium is still soft, and its ossification very little advanced towards the arch, which presents membranous spaces—*the fontanelles*—which separate the pieces constituting the lateral and superior parts of the cranium. You are also aware, that these membranous spaces are the seat of an ossific process which proceeds more or less quickly, but which, in general, is terminated towards the end of the second year, the cranial vault being then completely closed.

When at birth ossification has already taken place, and when, consequently, no fontanelles exist, or when the process of ossification is accomplished with more than usual rapidity, the fontanelles closing during the first months of extra-uterine life, the individuals sometimes remain microcephalous, and this microcephalism is coincident with idiocy. The impediment presented to the free evolution of the encephalon imprisoned within its unyielding osseous walls, explains, to a certain extent, the arrested development of the intelligence and of all the cerebral functions. When, on the contrary, the fontanelles are late in closing, and the brain can, consequently, become more easily developed, there is greater development of the faculties of which that organ is the seat.

Dr. H. Roger has shown that ossification of the fontanelles takes place between the ages of fifteen months and three years and a half; that is to say, that it is exceedingly rare for it to be seen at fifteen months, and that it is always seen at three and a half years. In a normal state, the occlusion of the anterior fontanelle is completed between the ages of two and three. Rickets and hydrocephalus retard or hinder this ossification—the one by impeding the deposit of osseous molecules in the fibro-cartilaginous tissues, the other by exerting an excentric action on the cranium, the sutures of which are thereby separated.*

The same accomplished physician has shown, that of all the affections of childhood rickets is that in which the blowing sound is most frequently heard over the sutures. In forty-seven rachitic children having open fontanelles, Dr. H. Roger noted the cephalic blowing in thirty, the blowing being intense. In ten cases in which it was absent, the rickets was either found in a slight degree, or did not exist. Consequently, says Dr. Roger, "from its great frequency, cephalic blowing may be regarded as a sign of rickets."* Previously, MM. Rilliet and Barthez, in accordance with Dr. Roger, and in opposition to the assertions of Dr. Fisher of Boston, had stated this blowing sound to be frequent in rickets, and rare in hydrocephalus; not being, however, always wanting in the latter, Dr. Roger remarks, that its absence or presence cannot be looked upon as an absolute differential sign.

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* **Roger**: **Bulletin de la Société Médicale des Hôpitaux, t. iv, 1860.**

Gentlemen, you must beware of mistaking that which occurs in rachitic, with that which occurs in hydrocephalic subjects, in whom the head has a size disproportionately large to the rest of the body. In hydrocephalic subjects, on the contrary, the anomalous size of the cranium is coincident with atrophy, and not with development, of the brain. The intraventricular serous effusion, the cause of the increased dimensions of the head, before producing separation of the osseous parietes and enlargement of the fontanelles, has begun to compress, from within outwards, the cerebral mass, effacing the convolutions in such a way, that when the effused fluid has been evacuated, microcephalism is found.

These are very important distinctions, because they explain the differences in development of the intellectual faculties in hydrocephalic and rachitic individuals. Whilst the former—*hebetes et obliviosi*—present all the characters of idiocy, the rachitic, these little suffering creatures, who are unable to move without assistance, generally possess a greater intelligence than other children of the same age: *præmatura ingenii, acumine et sensuum sincero exercitio a coætanis distinguuntur.*

Their physiognomy, so often stamped with sadness and suffering, their expression of countenance, their way of speaking, all denote an advanced development of the intellectual faculties.

But that which still more imparts to the physiognomy a peculiar stamp, that which makes the rachitic subject appear older than the small stature indicates, older indeed than the real age, is the conformation of the maxillæ, which, like the rest of the skeleton, are subject to the evil influence of the malady, which produces a great effect on the process of dentition.

I have already pointed out to you in a previous lecture† the influence of rickets on dentition: I must now revert to that subject.

When dentition has not commenced before the disclosure of the malady —which is very unusual—the evolution of the teeth is almost indefinitely retarded, and when it has commenced, it is interrupted. When the teeth have come forth, they become carious and black, as was pointed out by Glisson; they likewise become loose, and are ejected from their sockets.

This statement you can verify in the cases of the children now under your observation in the wards.

One of them, a year old, has not as yet got one tooth: another, sixteen months old, has only two: a third, twenty months old, has only eight, though rachitic in a slight degree and late in becoming rachitic, not being affected with the malady till less than four months ago. If you had an opportunity of observing the future of these children, you would see their teeth fall out ere they attained three or four years of age.

I have stated the general rule; but there are cases in which the teeth remain till the period of the second dentition.

I consider this arrest in the progress of dentition as a phenomenon of so much importance, that when an infant of a year old suffers from teething without any teeth appearing, I suspect that rickets is threatened.

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* ROGER: Recherches Cliniques sur l'Auscultation de la Tête. 1860.

† Volume II, p. 472.

When the disease has attained a certain degree of advancement, we find, on stripping the patient, that there is *deformity of the trunk and limbs.*

The *chest* presents a very extraordinary conformation. Flattened laterally below the axillæ, it projects anteriorly, presenting in front the appearance of the keel of a vessel, the patient being, as is vulgarly said, chicken-breasted: *instar carinæ navis aut pectoris gallinæ.* In place of being convex throughout, the chest is convex in front, but sunk in and hollow at the sides: if you apply a straight ruler to the lateral parietes, and let it fall from a perpendicular line to the bottom of the concavity, it will be found to measure three, four, or five centimetres in depth. At the junction of the ribs with the sternal cartilages, there are small nodulated projections, which, from their globular form and mode of arrangement, have been compared to the beads of a chaplet. This is what has been termed the rachitic chaplet, the knots of which are formed by swellings of the sternal extremities of the ribs. The appearance of this chaplet is one of the earliest phenomena of rickets.

The flattening of the chest, and its consequent contraction, are only conspicuous from the third to the ninth rib. Although the upper part of the chest may seem to be equally contracted, in reality it has not lost capacity in that situation, as is easily perceived by looking at the skeleton of a rachitic person. The appearance which is so deceitful in the living subject, arises from the shoulders being approximated to one another by shortening of the *clavicles*, which bones instead of being like an elongated letter **S** (as in the healthy subject) are like a flattened **S**.

From the ninth or tenth rib, the thoracic cage at once enlarges, so as to resemble an inverted pelvis in which are lodged the abdominal viscera.

Posteriorly, the natural concavity of the cervical region of the vertebral column is exceedingly increased, while the convexity of the dorsal region makes an anomalous projection—a gibbosity of a more or less decided character.

This deformity, this diminution in the capacity of the chest, produces, as you may suppose, embarrassed respiration, and by a mechanism which it is interesting to study.

During inspiration, the diaphragm descends, and the ribs rise, so as to produce a tendency to a vacuum in the cavity of the chest. But at the same time, the inspired air passing through the trachea and bronchial tubes into the pulmonary cells, distends them to such a degree that the lungs are constantly in contact with the costal walls. There is, therefore, pressure exerted from within outwards by the inspired air, but pressure, insufficient to bring to an equilibrium the opposed pressure of the atmosphere: in the normal state, the want of equilibrium is compensated for by the resistance to the external atmospheric pressure presented by the osseous and muscular cage in which the lungs are inclosed. The history of penetrating wounds of the chest shows us what happens when the external air can enter the pleural cavity through an opening in the parietes of the chest: the lung is squeezed up on itself, the contained air being unable to contend against that which is pressing upon the outer surface of the lung. Suppose that the unperforated thoracic wall does not offer a sufficient resistance, that which happens in rickets will happen. The pressure of the atmosphere acting with equal energy on all parts of the chest, but posteriorly, the ribs, whilst they are supported by the vertebral column, resisting this pressure, and anteriorly, where they are supported by the sternum, the ribs, soft and flexible, bend inward at their middle. Hence results that subaxillary flattening of which I have just been speaking, whilst the sternum is projected forwards, and the dorsal portion of the vertebral column, projected backwards, forms that gibbosity with a very long diameter observed in the rachitic, a gibbosity very different from that met with in Pott's disease. It follows, at the same time, that while that transverse diameter of the thorax diminishes, its antero-posterior diameter increases.

Though the flattening is limited to the lateral parts of the thorax, while the upper part retains nearly its normal aptitude, and the lower part is dilated, it is at that upper part, the ribs, particularly the first, are stronger than those which follow: this arises, in the first place, from their being more protected in that situation than elsewhere by thick muscles, and secondly, from the clavicles being placed, like two buttresses, between the sternum and scapula.

Though the base of the chest is dilated, that arises from its being supported by the liver on one side and the spleen on the other, which viscera are themselves supported by the mass of the intestines, generally much developed in rachitic persons: the diaphragm also here plays its part. Gentlemen, you know that the beautiful electro-physiological experiments of Dr. Duchenne (de Boulogne)* have explained the action of the diaphragm in respiration. During the inspiratory movements, it rests on the organs contained in the abdomen, and in contracting, it raises and throws outwards the lower ribs upon those to which it is attached. Now, from the time when the superior costal respiration becomes embarrassed by the contraction of the chest towards its middle, the increased diaphragmatic breathing becomes supplementary: from an abnormal increase in the frequency of the movements of the diaphragm, there results a dilatation—a persistent dilatation—of the base of the chest.

This abdominal respiration is a phenomenon to which I wish to call your special attention. Should you observe it in a child otherwise well, and free from fever, the probable existence of rickets ought to suggest itself to your minds.

The embarrassment of the respiratory functions, which leads to embarrassment of the circulation (particularly of the venous circulation), explains to us the *enlargement of the subcutaneous veins*, particularly those of the head, which Glisson pointed out as usually occurring in rachitic persons.

The embarrassment in the circulation perhaps also explains the *profuse sweating*, so common an epiphenomenon in the disease of which I am now speaking.

The base of the chest is confounded in appearance with the abdomen, the volume of which is much greater than natural in individuals of the same age: this abdominal enlargement often misleads not only the family of the patient, but also inexperienced physicians, leading them to believe in the existence of the affection known under the name of tabes mesenterica [*carreau*], constituted by tuberculous adenitis of the mesentery, or tuberculous peritonitis, both of which affections are essentially different from rickets.

On palpation of the abdominal viscera, we find that the liver and spleen pass far beyond the margin of the false ribs under which they lie. There is increased resonance, on percussion, over the whole abdomen, due to great flatulent distension of the intestines. Sometimes, however, there is dulness in the lower and lateral parts of the abdomen: this dulness occurs along with fluctuation, and reveals a certain amount of effusion into the peritoneum. Cases of this kind are rare; and a great amount of ascites is never met with. This is a fact which was noticed by Glisson, by whom none of the symptoms of the disease seem to have been overlooked.

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* DUCHENNE: De l'Electrisation Localisée et de son application à la pathologie et à la thérapeutique: 2me édition: Paris, 1861.

The liver and spleen are not enlarged, though they pass beyond the margins of the false ribs. Their prominence in the abdominal cavity depends upon their being pushed down by the contraction of the thoracic cage. It is, therefore, a mistake to believe that there is hypertrophy of the liver in rickets: nevertheless, this erroneous belief is so common that large-livered (*enfant au gros foie—magno-hépate*) is a synonymous term with a rachitic child. I myself long participated in this error, and, twenty years ago, when I published my views on rickets, deceived by appearances, I inscribed hypertrophy of the liver and spleen among common incidents of the disease. Later researches, and more minute study, convinced me that this hypertrophy was, on the contrary, an exceptional occurrence, and that the organs in question were only displaced in rickets.

The increased bulk of the belly in rachitic subjects is therefore due to the pushing down of the viscera contained in the abdomen—which viscera in a normal state are concealed under the ribs. It is due to that cause, but, also, even more, to the gaseous distension of the intestines: it also arises from the digestive apparatus itself being much more developed in these young persons than is natural in subjects of similar age, a fact which Glisson explains by rachitic persons being usually very large eaters, the same result taking place in them as in those animals which eat daily a great bulk of food.

This voluminous belly has as its base a pelvis which has undergone modifications, the development of which, as well as of the abdominal viscera, it partly explains. Thus, while the cavity of the true pelvis is contracted, the large pelvis has, on the contrary, a greater capacity. The bones which enter into its formation being flattened and turned outwards, the iliac fossæ are consequently widened. This depends upon softening of the bones of the pelvis, in common with all the bones of the body, as part of the disease. The iliac bones yield to the pressure of the intestines, which, although they do not press strongly, exert a continuous pressure on these bones, which is augmented almost every moment by the respiratory movements. In this way, the greater pelvis becomes spread out, and this spreading out contributes to the contraction of the lesser pelvis, the ischia being approximated by the see-saw motion, which gives them an inward bias; while, at the same time, the iliac portion of the greater pelvis is pushed outwards. This approximation of the ischia, which is promoted by the pressure of the femurs, leads to a change in the relative position of the coxo-femoral articulations; the cotyloidal cavities, which necessarily follow the ischia, approximate in the median line, their openings being directed downwards and a little inwards, in place of outwards and forwards as in their normal state.

The anomalous development of the abdomen, coincident with narrowing of the upper part of the chest, and a great increase in the size of the head, give the rachitic body a gourd-like form, a sort of figure **8** shape, if you will allow me to make such a comparison. To this deformed body are attached *members* the deformities of which are not less remarkable. Of course, I am now speaking of cases of rickets in which the disease has considerably advanced.

The thighs, separated from one another, form two arches, their concavity being directed inwards and a little backwards, the convexity being outwards and forwards. The legs have also lost their natural straightness. But this deformity almost invariably present in the thighs, where it is an exaggeration of the natural curve of the femurs, varies in the legs, not only in different individuals, but even in the same individual. In general, for example, before a child has begun to walk, the concavity of the curvature of the legs is directed forwards and inwards, in such a way, that the knees are widely separated from one another, and that the lower extremities, curved in their whole length, form, as the saying is, a parenthesis. When the children have walked, the deformity is constituted in the reverse manner, the concavity of the tibia and fibula being directed from without backwards, so that the knees approximate and the individuals are crook-kneed. In other cases, one of the two legs is bent outwards, and the other inwards, so that they fit into each other, so to speak; or they describe angles, arches, and Z shapes.

The same occurs in the upper extremities, with this difference, that the deformity is nearly always the same in the inferior, and varies much in the superior segment, the opposite of that which occurs in the lower extremities, as those just pointed out. Thus, almost invariably, the curved forearms have their concavity directed towards the palmar aspect of the hand, their convexity corresponding with the dorsal surface, whilst, although the arm is usually curved in the same direction, that is to say, concave forwards and inwards, convex backwards and outwards, it is not uncommon to observe an opposite arrangement, the humerus having its concavity backwards and outwards.

As I have already said, and as we shall see by and by, these deformities are due to softening of the bones: so great is the softening, that when one tries to bend the limbs in their continuity, very great flexion is the result. We must take care in this bending of the bones, to use great caution, otherwise we may produce fractures.

The articulations have undergone great changes. In the wrists and ankles, for instance, where these alterations are more manifest than elsewhere, there is visible at a glance, a considerable swelling of the articular extremities of the bones. It seems as if at the joints, a little above or a little below them, the limbs have been strongly bound round, so as to place them in the condition of trees the trunk and branches of which are strangled in such a way as to cause the formation of knots. This articular swelling is so constant, and at the same time so significant a condition, that the disease derives its vulgar name—*nouure*—from this peculiarity. This condition, which often appears at the commencement of rickets, prior to any other deformity, is the result both of this swelling of the ends of the bones and of the relaxation of the ligaments, the latter being sometimes so great as to permit the articular surfaces to separate from one another under the influence of even moderate traction, and that on fixing, for example, the forearm or leg, one can bend the hand or foot so as to cause them to describe a semicircle on their axis, the palmar aspect of the hand being brought into a line with the ulnar margin of the forearm, and to turn the foot from one side to another, the dorsal aspect being directed either inwards or outwards.

The relaxation of the ligaments, which exists alike in the vertebral articulations, in the sacro-lumbar and sacro-pelvic articulations, combined with the softening and extreme sensibility of the bones, explains why rachitic children are so long in being able to walk, and why they are obliged to maintain the recumbent position.

Such, gentlemen, is a picture of the characteristic deformities of rickets, a picture which you will find has an infinity of varieties: in some subjects, it will be as complete as I have presented it to you, and in others, some of the details which I have delineated will be wanting.

The deformities are the more striking, that they coexist with great emaciation. The muscles lack that firmness which they possess in healthy persons: both their bulk and power is diminished: *torositas musculorum minuitur ultra quam credi potest.* The cellular tissue which surrounds them is not filled with that hard thick fat which gives roundness to the form in healthy young children. The skin, shrivelled, flabby, and sallow, like a half-empty bag, covers the atrophied muscles, which form the only separation between it and the stunted, twisted bones.

Pursuing this subject still further, let us now study *the order of succession in which the deformities of the bones are produced:* let us endeavor to discover their *causes*, and explain their *mechanism*. We shall not, for the present, enter upon the pathological anatomy of rickets, but confine ourselves to what takes place in the living patient.

Dr. Jules Guérin, to whom science is indebted for remarkable and important researches connected with the subject now before us, has established the law, that rachitic deformities are always produced from below upwards, that is to say, that their first seat is in the lower extremities, that they appear first in the legs, then, in succession, gain the thighs, pelvis, vertebral column, and thorax, affecting last of all the upper extremities. So decided is his opinion on this subject, that he goes the length of asserting, that "all isolated deformities in the upper parts of the skeleton, in the vertebral column, for example, when there is no deformity in the lower parts, are not due to rickets." This proposition is much too absolute. It is true, as a general statement, in respect of rickets in adults, although many exceptions occur: it is true, to a certain point, in respect of children who have begun to walk: but it is false, entirely false, when applied to very young children. In the latter, the deformities supervene in an inverse order to that indicated by Dr. Guérin. In them, they begin in the chest, trunk, and upper extremities.

These differences in the order of succession in which rachitic deformities occur, according to whether the individuals have or have not begun to walk at the epoch of the invasion of the disease, are explained by the causes themselves which produce them. These causes are pressure exerted continuously upon the softened bones, whether it be on the chest, in the manner I have explained to you, by the column of atmospheric air which surrounds it, or whether it be on the limbs or vertebral column by the weight which they have to support: this pressure, and the incessant action of the muscles inserted into the different points of the levers constituted by these bones, cause the deformities.

These causes acting together produce the double effect, at least upon the limbs, of producing a tendency to settling most marked in the short bones, a tendency to increase the natural curvatures specially marked in the long bones, and more decided in one direction than in another according to the disposition of the muscular apparatus in relation to the lever upon which this power acts, and according to the power and continuity of muscular action.

Let me explain myself. The muscular masses applied directly to the femur—for example, the biceps and crural triceps which envelop it—tend, by contracting in a line parallel to the great axis of that bone, to become shortened, to be huddled up on itself, when owing to the softening from which the patient has suffered, it no longer offers a sufficient resistance. On the other hand, the abductor muscles of the thigh, and those which flex the thigh on the pelvis, much more powerful than the abductors and extensors obliquely inserted at different points in the lower part of the thigh, tend to bend it, by approximating its upper and lower ends to one another, thus acting like the string of a bow. The result is an increase of the natural curve, which, in a normal state, is directed inwards and a little backwards. In the forearm, the mechanism is similar: the most powerful muscles, those situated on the anterior part of the member, and obliquely inserted in the lower parts of the radius and ulna, tend to bend these bones towards the palm of the hand, while the deepseated muscles, acting in a direction parallel to the axis of these bones, tend to huddle them together. Here, on the contrary, where, as in the legs and arms, the opposing muscles nearly antagonizing one another, the deformities of the bones do not admit of being reduced to rule, and are completely subordinate, either to the pressure on the bones from without, or to the pressure exerted by the parts which they support.

This effect of the contraction of the muscles, which, combined with the effect of pressure, may be such as to break the bones, is very decided in the clavicles, which yield to the action of those muscles of the chest and back which are inserted in the clavicle and humerus and draw the shoulders towards the median line.

These, gentlemen, are facts which, to be understood, require only to be stated. You can also understand, without its being necessary for me to enlarge on the point, the manner in which pressure acts. I say nothing of what takes place in the chest, having already treated that part of the subject at sufficient length. I shall only allude to what takes place in the limbs and vertebral column: the latter is shortened from the squeezing up of the vertebræ, and is, at the same time, bent on itself in the direction of its natural curves, which, consequently, are increased.

You can understand from what I have said, why the osseous deformities are produced in so different an order of succession according to the age at which the individuals become influenced by the disease.

A child of fifteen or sixteen months is seized with rickets, some time after it has begun to walk. Its bones undergo progressive softening: those of the lower part of the body, which, when the individual is standing, support the entire weight of the body, are necessarily the first to become deformed. In these cases, the deformities take place in accordance with the law laid down by Dr. Guérin: the legs are affected in the first instance, then the thighs, pelvis, and vertebral column.

But in a child which has not yet walked, matters proceed differently. As it is always lying, its inferior extremities are not subjected to the pressure under which they yield in the other case. The only pressure which the body has to support is that of the surrounding atmosphere, a powerful and constant pressure which the thorax cannot resist, its osseous framework being softened. Hence it is, that in very young children, the first observed deformities characteristic of rickets are those of the chest.

Now comes the question, How and why is it, that in a very young child the upper extremities become deformed before the lower? The mother of one of our little patients has explained this to you: "My child," she said, "cannot stand; and to enable him to sit down, he requires to rest on his hands." In this statement, you can at once perceive an answer to the question: we have, in fact, something very similar to that which I have just been telling you takes place in the walking child. The bones of the arm and forearm, which serve as a point of resistance, as the supporting basis of the whole of the upper part of the body, yield, in consequence of their softened state, under the weight they have to sustain.

I have been telling you, that under the pressure exerted on them, combined with muscular action, the bones of rachitic subjects may be broken; I may add, that an abrupt movement is sometimes sufficient to occasion such fractures. This is a not unfrequent complication of rickets. It is not unusual to find numerous fractures in the same patient—several bones broken, or the same bone broken in several places. When I go into details relative to the pathological anatomy of our subject, I shall have to examine the mechanism of these fractures. For the present, bear in mind, that they often remain unknown to the relations, and even to the physician. On the one hand, the slight cause which often produces them, the usual presence of pain in the limbs, and its small increase at the time of the accident, make its occurrence pass unperceived: on the other hand, when we do suspect the occurrence of the accident, the diagnosis remains very obscure, for a reason which I am now going to state. The deformities due to displacement of the fragments, when there is fracture, are difficult to distinguish from those which depend on increased curvature or deviations of the bones depending on rickets: generally, moreover, there is no displacement of fragments, the integrity of the thickened periosteum maintaining them in position, sometimes in so solid a manner as even to allow the limbs to be raised in one piece. From this point of view, rachitic fractures resemble false joints. Finally, crepitation, the sign of so much value in the diagnosis of fractures, fails us in rachitic fractures, from the softened surfaces in contact not being hard enough to produce crepitation.

We must not suppose, however, that the deformities of rickets are due exclusively to the action of the muscles, or to pressure, as they may be produced during intra-uterine life. The fœtus is very seldom affected with rickets: but cases are sometimes met with; and a very remarkable one has been seen recently by Dr. Peter at the Hospice des Enfans Assistés. The infant in question was eight days old when deposited at the hospital. It was rachitic in a very high degree: there existed great curvature of the upper and lower extremities, deformity of the thorax, and continued fever. But the most interesting feature in the case was the existence of callus, and indications of a consolidated fracture of the ulna, and of a consolidated fracture of the femur. The infant died on the second day after admission to the hospital. At the autopsy, Dr. Peter found the bones of the limbs in an abnormally flexible state, but not spongy. Callus was distinctly visible in the situations where its presence was diagnosed during life. This very interesting case not only proves the possibility of intra-uterine rickets, but it also demonstrates, Dr. Peter says, that the curvature of the limbs is produced in accordance with a law peculiar to the malady, rather than under the influence of weight or muscular contraction: and the existence of well-formed callus shows, that rachitic fractures may take place within the uterus, and that the state of rickets has long existed, as the fractures have had time to be produced, and time to be consolidated.

Dr. Peter failed to obtain any information from the relatives; and could not ascertain whether the precocious rickets depended on hereditary causes, or on other causes belonging to the individual's relationship.

Gentlemen, the study of the *pathological anatomy of rickets* is closely connected with the history of the deformities caused by the disease, and forms the complement of my remarks on its mechanism.

Our knowledge of this subject is of recent date. Glisson and the authors of last century were necessarily imperfectly informed on this part of the subject: they were quite aware that the softening of the bones constituted a leading characteristic of the disease: they also knew that there succeeded to this softening, at a given moment, consolidation of the bones which had been affected by it: but that was the extent of their knowledge. It is true that Duverney, in his "Treatise on Diseases of the Bones," published in 1751, had entered into some details regarding the state of the bones in rachitic subjects. He had noted their rarefaction, their greater lightness, the roughness of their surfaces, due to the presence of layers of osseous matter *formed by the extravasation of the nutritive juices*. So entirely had these summary indications been forgotten, that, in reality, our knowledge of the subject dates only from our own day.

The first researches undertaken in this direction were those published in 1834 by Dr. Rufz.* Dr. Jules Guérin's long and substantial memoir appeared in 1839.†

A first period—*a period of incubation or effusion*—is characterized by a sanguinolent effusion into all the interstices of the osseous system, which seems as if infiltrated with blood less viscid and less consistent than that contained in the bloodvessels.

A second period—*a period of deformity*—is characterized by the development of a very fine, spongy tissue in the epiphyses and interstices of the long bones, and also between the periosteum of the bone, which latter becomes soft and bent.

A third period—*a period of absorption and consolidation*—is characterized by the transformation of this spongy tissue into a compact tissue denser and closer than the normal tissue of the bone, a transformation which gives it an extraordinary degree of solidity, converting it into a true ivory.

To these three, Dr. Guérin adds a fourth period, which is observed in persons who have long suffered from the disease, and in whom the rachitic cachexia is developed: this is the period of *rachitic consumption*.

I accept these divisions; I only change some of Dr. Guérin's terms, substituting for them others which seem to me to be more in harmony with the conditions they represent. I admit, then, a first period of *fluxion and effusion*, a second of *softening and transformation*, a third of *reconstitution and consolidation*, and a fourth of *consumption*. These periods I shall now describe to you.

In the *first period*, the bones are tumefied; and this tumefaction is specially very manifest in epiphyses of the long bones, in the short bones (with which the epiphyses have a great analogy), in the flat bones, of which the external and internal tables are attenuated, their diplöe being as if inflated.

The bodies of the long bones generally retain their curves and natural shapes; because the softening which the osseous tissue undergoes, and which advances to its highest degree in the succeeding period, is not yet sufficiently advanced to yield to the influences of pressure and muscular contraction, the active causes, as I have told you, of rachitic deformities.

This softening, however, is now sufficient to deprive the bones of their consistence, and impart to them a certain amount of elasticity. If we make pressure with the finger, or only with the nail, on a point in the diaphysis of a long bone, we depress it, we crush it: if we squeeze somewhat vigorously a portion of an epiphysis, short bone, or flat bone, we readily cause flattening: if we endeavor to cut one of these bones with the scalpel, we succeed without any difficulty, and this we can do only in the skeleton of a person who has died of rickets.

On examining the incision, we see that the tumefaction is due to the areolæ of the spongy tissue of the short bones and to the diplöe of the flat bones having experienced incipient dilatation, and partly to the concentric lamellæ forming the diaphyses of the long bones being more or less separated from one another. These areolæ and interlamellar spaces are filled with a fatty, gelatiniform, sanguinolent matter, which, in consistence and color, resembles pale-red currant jelly. This matter, which exudes from all the pores of the divided bone, a real matrix of new tissue which we see developed at a later period, likewise fills the medullary canal of the long bones, and is found interposed between their external surface and their periosteum.

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* RUFZ: Recherches sur le Rachitisme chez les Enfants. [*Gazette Médicale de Paris*, 1834, p. 65.]

† GUERIN (Jules): Mémoire sur les Caractères Généraux du Rachitisme. [*Gazette Médicale de Paris*, 1839, pp. 443, 449, 481.]

The periosteum itself has undergone changes. More vascular than in the natural state, and injected with blood, it presents a bright pink color: thicker than it usually is, it adheres intimately to the bone, which is very vascular and has lost its smoothness of surface.

The cartilaginous plate, which in young subjects separates the epiphyses from the diaphyses of the long bones, has a softened, bluish, and semitransparent tint, which disappears after some days of maceration in water; so that the epiphyses become completely detached from the bodies of the bones.

When dry, these tumefied inflated-like bones have lost density and weight: their spongy tissue is formed of cellules much larger than those which exist in healthy bone, some being enlarged, and others constituted by the union of several, which have had their walls destroyed or torn: their compact tissue is perforated by small holes, formed partly by interstitial absorption, and partly by disappearance of the vessels of the new formation which traversed them in the recent state.

In *the second period* of softening and transformation, the swelling of the epiphyses of the long bones is much increased, and towards the end of that period it is also very great: in the short bones (of which the external plate of compact tissue becomes porous, whilst the areolæ of their spongy tissue enlarge), and in the flat bones (the attenuated external and internal tables of which are soft and elastic, and in a state to be indented by pressure with the fingers, whilst their dilated diplöe is constituted by large cellules of a slightly resisting texture containing a violet-colored medullary juice, visible through the transparent laminæ of the compact tissue).

The diaphyses of the long bones swell out, or seem to do so, in consequence of a very peculiar disposition of the medullary membrane and still more of the periosteum, according as the normal direction remains or is modified by deformities.

Thus, the periosteum is much more vascular than in the first period, and the gelatiniform effusion between it and the body of the bone is also much greater: this infiltration of gelatiniform matter has assumed, moreover, enormous proportions wherever it has been effused. It is also transformed into a tissue which is reddish, but less red than the liquid from which it has been constituted. Very elastic, resembling a very fine sponge, this new spongy tissue is easily distinguishable from the old spongy tissue, by its fibres being more dense, more compact, and paler.

It is then that the bones have undergone a notable softening. On grasping them with the fingers, you will be able to bend them with wonderful facility. In the living rachitic subject, we have already found how easy it is to curve the limbs. It is at this period that you can cut them across as you would slice a carrot, a root, or a soft branch; or, you can cut them into longitudinal strips, which may be bent and rebent without being broken.

There is, therefore, in this second period of the malady, a very manifest softening of the old tissue of the bone, the lamellæ of which have become less numerous, more detached from one another, and more pliable than in the preceding period: at the same time, the formation of a new bony tissue, constituted by the gelatiniform matter which began to be effused in the first period, now augments in quantity, and assumes a more and more marked cartilaginous consistence. There is, in a word, disorganization of the old osseous tissue, in which the concentric lamellæ of the long bones, the plates constituting the spongy tissues of the epiphyses and of the short bones are still more separated from each other, and are also less complete than during the preceding period. So great is the destruction of the osseous lamellæ, that when the bones are dry, on injecting water into that which constitutes the compact tissue of a long bone, it is found to traverse the tissue from one end to another, gaining in succession the different layers; and on placing a short bone before the mouth, we find that we can breathe through it, so great is its porosity. But simultaneously with this disorganization of the old osseous tissue, reconstitution of a new bony tissue is taking place.

When this process of reconstitution is very active, the entire bone seems to consist of the gelatinous matter of new formation, in which there is found the substance of the old tissue softened, and the most solid plates of which are those next the medullary membrane, which is very vascular and very thick, as is also the periosteum.

A minute ago, I was telling you that the modifications of the medullary membrane and the periosteum vary according to the presence or absence of deformity. I am now going to describe these modifications, which are coincident with those of the bone itself, and its medullary canal.

If you examine the diaphysis of a long bone which has nearly retained its normal straightness, its periosteum will present neither unusual density nor unusual thickness. Sometimes, the periosteum does not adhere closely to the surface which it covers; but at other times, it adheres to it so firmly, that an attempt to separate it will tear out along with it the most external layer of the bone. In that case, the external surface of the one, and the internal surface of the other, are rugose, covered with small osseous points, which communicate to the finger, when passed over it, the sensation produced by feeling a fine file.

If you examine an abnormally curved bone, you ascertain its different dispositions in the concavity and convexity of its curves. In the concavity, the periosteum, red, and greatly injected, is more or less thickened. It is also more adherent to the bone, from which often it cannot be detached without tearing away a part of the osseous tissue itself, or rather the newly formed tissue exuded upon its internal surface.

This new tissue, which in that situation is found in great quantity, looks like cartilage, or bone, softened by acid. Its most internal layers are constituted by the old tissue, the primitive layers of which are separated from one another by the effusion; its more external layers (those which form the greater part of the whole) are evidently new products of periosteal secretion. The formative process is exactly similar to that by which the formation of callus takes place in fractures. The analogy is the more complete, that the new tissue is about to be transformed into an osseous tissue much more solid, and much more dense, than the old tissue of the bone: this change is absolutely similar to that which takes place in respect of callus.

In the convexity of the deformed bones the periosteum has, on the contrary, lost its normal thickness, which is accounted for by the compression of, and friction upon, that part exerted by the surrounding muscles, and perhaps also by other causes of which I am ignorant.

The medullary canal, filled, as I have said, by the matter everywhere effused, has lost its normal calibre. This contraction, produced by thickening of its internal membrane, and the effusion of matter into the canal, is greatest where the curvature of the bone is greatest. When the curvature exists in a high degree, the medullary canal terminates abruptly at the convexity, and opens under the periosteum, meeting, at an obtuse angle, the other portion from which it has been separated by the new tissue formed in the concavity of the curvature.

Let us now see, gentlemen, what takes place in *rachitic fractures.*

You can obtain a very complete idea of the facts which I am going to explain to you by a glance at the preparations now before you; they present numerous examples of the different kinds of rachitic fractures.

These two femurs, both broken in two places, show you the fragments dovetailing, so to speak, the superior and inferior fragments respectively penetrating each other. These fractures are not consolidated; and this is the condition which you will find most frequently in the second period of rickets, when the fractures really give rise to false joints, that is to say, to a state of the parts similar to that which supervenes in ordinary fractures, in which the consolidation has been prevented by one cause or another, and in which the fragments remain united by a sort of elastic fibrous tissue. This fibro-elastic tissue is formed by the products of exudation furnished by the medullary membrane and the periosteum; and in addition, in rickets, by the cartilaginiform matter, about which I have said so much, and which begins to be transformed, I repeat, in the second period.

It is especially in this second period of rickets that the different parts of the skeleton have in the recent, and still more in the dry state, a very low specific gravity. Here you see on this table the entire skeleton of a child of eight years of age, which hardly weighs one kilogramme, though it ought to weigh seven or eight. Observe how spongy the bones are; observe that when they fall, they neither make any noise nor rebound like normal bones. The tissue of the epiphyses and of the short bones and flat bones are exceedingly rarefied. These puffed-out bones resemble bits of sponge-cake, or bread made of gluten. The bones of the cranium are transparent from attenuation of their external table, which is riddled with holes.

In *the third period,* the period of reconstitution and consolidation, a change takes place somewhat analogous to that which takes place in the first period. In the second period, we have seen that the broken bones become the seat of a process similar to the reparative process which takes place in every fracture—fluxion of the periosteum and medullary membrane, effusion of the matter destined to become the matrix of the new tissue—a fluxion and effusion which are particularly manifest in the periosteum. The analogy which I have already pointed out, and to which I again call your attention, between the process of consolidation in ordinary fractures and reconstitution in rachitic bones, takes place in the third period of rickets. In the midst of the fatty gelatiniform matter effused during the first period, which began to be organized, and had assumed the gelatiniform aspect in the second, we now see the development of calcareous, osseous centres—the first rudiments of the new bone which is being formed. In the long bones, it is in the walls of their diaphyses that we must seek for the most remarkable changes tending to the reconstitution of tissue. "The compact dovetailed layers become," says Dr. Beylard, "thicker and denser; the new tissue which was deposited in the concentric spaces between the cylinders likewise acquires some consistence; and it is remarked that between the small osseous bridges which they send forth by constituting cellules of different dimensions, the organization of the effused matter is accomplished by a layer of phosphate of lime finer than that in the normal state. This transformation is accomplished rather slowly when the lamellæ have undergone great separation. It is not till a much later stage that the walls of the whole diaphysis are found to be transformed into the compact homogeneous substance which acquires the hardness of ivory." Consolidation thus is accomplished by a kind of eburnation exactly like that which is formed by the callus of fractures. This at least is what occurs in respect of the fractures of long bones.

In the epiphyses, short bones, and flat bones, consolidation is not produced at the charge of the periosteum, but solely by the amorphous matter effused into the areolæ of the spongy tissue of these bones. In the epiphyseal extremities of the bone, a change takes place of an opposite kind from that which occurs in the diaphysis. Although in this situation the new tissue acquires the density of ivory, eburnation, which is specially distinct at the places where the bones are fractured and in the epiphyseal extremities, has a tendency to be partially absorbed and to form areolæ in such a way as to suggest to the mind the appearance of normal spongy tissue.

This eburnation of rachitic bones is explained when one has seen that great vascularity at the period of softening and effusion, that extensive development of vessels, and that hyperæmic congestion which impart to the bone the appearance of inflamed bone. What takes place in rickets is, I repeat, analogous to the process of consolidation in fractures and in cases of ostitis; thickening of the periosteum, inflammation, or if the term *inflammation* do not state what actually takes place, let me say a pathological process, in virtue of which there is deposited in the cellular tissue, in a sort of matrix, materials destined either to form the solid callus, or to become new bone, callus, or new bone which will become harder than the old bone.

Sometimes the bones, which during the preceding periods presented greatly increased curvatures, commence, at that period of consolidation, to get so straight again that the deformities may at last almost wholly disappear.

Upon weighing the bones thus repaired, one is struck by seeing, that in some parts of the skeleton they have increased, and in others diminished in weight. The weight is increased, in those which have not undergone shortening, such as the bones of the cranium, which have augmented both in density and in thickness. As for the long bones: upon taking the comparative weights of, for example, the femur of a rachitic child and the femur of a non-rachitic child of the same age, you will find, that the former is much heavier in the mass than the latter; but you will also find that certain parts of the former—those which have acquired a greater density and become eburnated as it were—weigh, on the contrary, much more than parts of equal volume and length taken from the non-rachitic subject.

Such, gentlemen, is the history of the ordinary osseous lesions which characterize rickets, when the disease is arrested, and the patient recovers. To complete the picture of the pathological anatomy, I have still to speak to you of that which supervenes in individuals who have fallen into a state of true rachitic cachexia.

In them, the period of consolidation gives place to *the period of consumption*. There is then no attempt at reconstitution of the osseous tissue: the bone remains rarefied: the matter effused into its areolæ, and into its interlamellar spaces, beneath the periosteum and medullary membrane, is not transformed into that cartilaginous tissue which ought itself to be transformed into new osseous tissue. The old osseous tissue is gradually absorbed.

In the recent state, the long bones are exceedingly soft, and also friable. They are reduced to a very thin shell, filled with fatty matter: in some places, they are whitish, or of a more or less red hue, and contain the débris of osseous plates.

In the dry state, these bones are extraordinarily light, friable, and brittle.

The lightness and friability are not less remarkable in the short than in the flat bones, the tables of which become excessively porous; whilst their diplöe, the cellules of which enlarge, assumes an aspect so much like that of paste which has risen, that I cannot make a more appropriate comparison than to say that it is like a macaroon.

I must necessarily rapidly pass on from these questions, the full discussion of which would lead me far beyond the limits of the instruction appropriate for this place. To fill up the outline which I have sketched, I recommend you to read the chapter which Dr. Beylard has devoted to this subject in his work, to which I have several times alluded.

*General Symptoms of Rickets.—Pains.—Loss of Flesh: Muscular Atrophy.—Profuse Sweats.—Embarrassed Respiration.—Progress of Rickets.—Death is in general the result of Thoracic Complications.—Etiology of Rickets.—Influence of Bad Diet.—Rickets must not be confounded with Scrofula.—*OSTEOMALACIA, *or Rickets in Adults.—Treatment of Rickets.*

GENTLEMEN: I have explained to you the physical or organic symptoms of rickets, among which osseous deformities occupy the most important place. I now resume the study of the symptomatology of the disease, and proceed to discuss another order of phenomena, that is to say, functional disorders. They have so great a value, that they often are sufficient in themselves to characterize the disease now under our consideration. They are the *earliest symptoms*, and as they manifest themselves before any appearance of pathognomonic accidents, are often difficult to realize; and they remain unknown to physicians either inexperienced or unaware of the possibility of their occurring.

In the first rank of this class of symptoms, are disturbances of the intellect, or rather, *special modifications of the mental condition.* There is a certain kind of sadness analogous to that observed in cerebral affections, or, still better to indicate its nature, analogous to that gloom which takes possession of children who are hatching—forgive the expression—who are hatching the malady we term cerebral fever. You will understand that this symptom can only have a relative value, and is not one to which too much importance is to be attached, as it is met with not only in rickets, not only in cerebral fever, but likewise in many other diseases which in their commencement, or in their period of incubation, lead to a state of painful discomfort telling much upon the extreme mental mobility of children.

This mental gloom of rachitic patients depends, according to all appearances, upon their exquisite sensibility in every part of the body, a sensibility which I have been anxious to point out to you, which shows itself by eliciting plaintive cries of pain when an attempt is made to raise up the young patient. The unfortunate little creature, who up till then was enchanted by the caresses lavished upon it, appears now to be afraid of them; even an approach to the bed on which it lies, made as if with the object of changing its position, causes its countenance to express anxiety and fear. This change in the child's character, this fear which it shows of having pain roused up by the pressure of a hand, this habitual stamp of sadness on its countenance, differ from anything seen at the commencement of other serious maladies, particularly from the prodromata of cerebral fever. Indeed, in a child stricken by that cruel affection, we can still produce a transient cheerfulness, causing it for the moment to emerge from its habitual melancholy languor. In the rachitic child, this is impossible. The more we try to excite it, or induce it to move, the more will it manifest impatience. It is heedless as to the games of which, formerly, it was fond. This repugnance to the amusements of its age, this habitual sadness in a child, which, with an appetite increased rather than diminished, loses flesh visibly, which always has an acceleration of pulse coincident with profuse sweats, are symptoms, I say, which have a certain meaning; for the child does not cough, and presents no sign which can give rise to a suspicion of the existence of tubercular phthisis.

These phenomena, in proportion as the child begins to walk, become more and more evident; the fever, or at all events, the acceleration of pulse continues; the skin is constantly covered with profuse sweat, whether the patient be sleeping or waking, or whether more or less covered than usual. The excessive perspiration is greatest in the head, and it is on that account necessary to change the child's cap and pillow-case several times a day, so quickly do they become soaked.

MM. Marchand, Otto, Weber, and some others, are of opinion, that the bones of rachitic subjects contain lactic acid and lactates, through the agency of which the phosphate of lime of the bones may be dissolved, and then absorbed. Thus it is, that the inorganic or saline matter goes on decreasing, and the organic or gelatinous matter becomes predominant: the result is softness of the bones. It appears from analyses recently made by M. J. Drivon, a distinguished interne of the hospitals of Lyons, that in osteomalacia the proportion of inorganic matter fell from 64 to 41 in the compact, and to 18 in the spongy substance: and he also found, that bones of rachitic subjects contain lactates, under the influence of which the phosphate of lime may be dissolved, and then absorbed.* My pupil, Dr. Peter, placing these two analysis side by side with the great clinical fact, the profuse sweating in rachitic subjects, asked, whether they were not connected. According to this view, the important and perhaps fundamental fact in rickets is the excessive production of lactic acid: a part of this acid would be eliminated in the sweat, which is known to be rich in lactates, and another portion, not eliminated, would act on the inorganic constituents of the bone and soften them. The sweats would be an emunction, but an emunction insufficient for the lactic acid secreted too abundantly in consequence of a vice in nutrition, the nature of which has not yet been determined.

It is then that the *pains* assume a character as to which no one can entertain any doubt. Then it is, that if an attempt be made to raise up the child in its bed, or, still more, if an attempt be made to lay hold of it, whatever precautions be used, it shows, by its cries, the sufferings which it is enduring. These sufferings are sometimes so great, that some subjects refuse absolutely to allow themselves to be touched, the nurse being obliged, when she suckles, to bend over the infant to enable it to get the nipple into its mouth.

It is then, also, that we begin to perceive that there is *embarrassment of respiration:* this habitual oppression is a constant phenomenon in confirmed rickets, particularly when it occurs during the first two years of life. At a later age, that is to say, in children of three years, and still more in those that are older, it is an exceptional phenomenon. In the very young infant, it exists in an extreme degree. If you bear in mind my remarks on the progress of rickets, you will see the reason of these differences. Bear in mind, that at a very young age, the disease first appears in the chest, which is the seat of the earliest deformities; while in children who have begun to walk, they are first seen in the lower extremities. Bear in mind the degree to which the thoracic deformities proceed, and the extent to which the play of the respiratory organs is embarrassed.

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* DRIVON: L'Union Médicale, for September, 1867.

At this period of rickets, the loss of flesh becomes extreme; and this has engaged your special attention at the bedside of the little patients in our wards: this emaciation goes on increasing up to the time at which the malady is arrested. It is not only emaciation, that is to say, disappearance of the subcutaneous and intermuscular fat, but also, as I have already told you, a true atrophy of the muscles, which in rickets proceeds to an extreme degree, reducing the muscles to mere small fibrinous bands.

Atrophy of the muscles, softness of the bones, and excessive sensibility of every part of the body, are the causes of the modifications which supervene in the functions of locomotion: they are the causes of the laziness to move which the children show, and the repugnance they manifest to remain in any other than the horizontal position.

I am not afraid to return to facts to which I have already called your attention: their importance is an excuse for repetitions. A child, which, up to the invasion of the malady, could perfectly well stand upon its legs, when supported by the arms, or held round the waist, which with energy straightened itself, and threw about its feet on the bed or piece of furniture on which it was placed, no longer does anything of the kind. If taken out of its bed, it crouches up, draws its knees up to its belly, bending the legs on the thighs, and the thighs on the pelvis, absolutely refusing to hold itself straight up. The child which used to sit perfectly well in its nurse's arms can do so no longer. Should it have begun to walk, its walking becomes slower, difficult, and unsteady; and after some weeks, it will drag itself painfully by the side of pieces of furniture which it uses as resisting-points, and is afraid to relinquish: it will then become wholly unable to get up, and will, consequently, remain constantly in bed.

Let us now study what takes place in the great functions of organic life.

First of all, let us consider the *organs of digestion:* the appetite which at the commencement of the disease had retained its regularity, which may even have been notably increased, diminishes proportionably with the advance of the rickets, and is quite lost when the disease has reached its climax. The great secreting organs, the cutaneous and renal systems, are functionally disturbed. The skin is covered with *profuse sweats*, and as a consequence of this increased perspiration, sudoral eruptions supervene. The urine also is generally abundant, though cases occur in which it is scanty: it is always pale, and there is a deposit which chemical analysis shows to consist of calcareous phosphates.

This incessant waste, and the coincident continuous fever, explain the emaciation of the patients. Nutrition goes on the more badly, that in addition to loss of appetite, the digestive functions are seriously disturbed. Obstinate constipation, alternating with a diarrhœa which is rebellious against all treatment, an intestinal flux, contribute to augment the general debility.

The embarrassment of the respiration, which plays so great a part in the disturbance of nutrition by rendering imperfect the process of hæmatosis, makes rachitic more liable than other children to acute pulmonary affections, bronchial catarrhs, catarrhal pneumonias, which, in consequence of the obstacle to free respiration occasioned by the thoracic deformity, assume a very great degree of gravity. Pulmonary tuberculosis is a very unusual complication of rickets: to this remarkable fact, I shall have to direct your very special attention.

This observation suggests the question: How do the rachitic die? But another question has first to be answered: *What is the progress of the disease?*

Under certain circumstances, the progress is rapid. You will see children reach the last period, the period of consumption, in three or four months. In other cases, the softening of the bones does not attain its maximum for a year, eighteen months, or perhaps not for two years. Generally, rickets runs its course in six, eight, ten, or twelve months. Then, after a period of two or three years, consolidation is effected, and the patient is cured, but considerable irremediable deformities remain.

If matters always had this issue, rickets could not be considered as a serious disease, at least not serious in the sense of danger to life. Unfortunately, however, a fatal issue is often caused by the accidents with which it becomes complicated.

Of these accidents, the most important are thoracic; and, in fact, it is by pulmonary affections that the majority of rachitic subjects are carried off. Others die exhausted by the profound disturbance of the functions of nutrition, digestion, hæmatosis, and of the cutaneous secretions, of which I have just been making a rapid review.

I now come, gentlemen, to a great question—*the etiology of rickets*—a question of far greater importance than one might at first be inclined to suppose. Numerous causes of rickets have been enumerated; but I shall confine my remarks to those which are in my opinion of incontestable influence, believing that the mention of the others would be both tedious and fastidious.

Of unimportant alleged causes there is one, however, which deserves to be discussed.

It has been said, and the opinion has been held by the most eminent physicians, that the scrofulous constitution plays a most important part in the production of rickets: confounding the two diseases with one another, it has been alleged, that the difference between rickets and scrofula exists only in form, the latter being only a manifestation of the former, just as are the glandular congestions, and all the alterations of bone observed in scrofulous subjects.

This is an error against which I have long protested. Having been intrusted for fourteen years with the medical charge of a large number of children at the Hôpital Necker, and having been subsequently four years at the head of a children's service in the Hôpital des Enfans Malades, I have had occasion to see and follow out a large number of rachitic subjects. Observing, with the most scrupulous care, the numerous facts which have come under my notice during that long period, and always finding a very great disparity between rickets and scrofula, I have established (as moreover was well said before me by Dr. Rufz) that rickets and scrofula not only are not manifestations of the same diathesis, but that, as a general rule, the one excludes the other. This law (also expressed by Dr. Jules Guérin), is so absolute, that you will have, *à priori*, almost a certainty of not finding tuberculous or scrofulous affections in a rachitic subject, nor rickets in one who is scrofulous or tuberculous.

There are, as I know, exceptions to this general rule. Cases have been published of this exceptional kind, and I could myself cite examples. These exceptions did not escape Glisson, who likewise pointed out the same distinction which I have established between rickets and scrofula: the exceptions are exceedingly rare, and are too few to weaken the law.

Even in their external characters the two diseases present notable differences. Recall to your recollection the picture of rickets which I drew for you, and compare it with that of scrofula: the differences between the two will then strike you as forcibly as they have struck me.

The scrofulous, far from being, like the rachitic subject, smaller than another child of the same age, is often remarkable for being taller. Its limbs, solid and resisting, present no deformities except when white swellings (not unusual in them, it is true) have attacked the joints, or when caries of the bones has led to ulcerations and indelible cicatrices. Except in cases of white swelling, the articulations are not deficient in firmness, are as well knit as in healthy persons, and offer neither the nodules nor irregularities of surface which characterize rickets.

In taking into account the period of life at which scrofula and rickets appear, the parallel will not be less striking. The one, as I have told you, appears in the earliest epoch of infancy, towards the end of the first year, or in the course of the second; for you must remember that I am not speaking at present of osteomalacia, the rickets of adults and aged persons, while scrofula shows itself especially in the second epoch of infancy. Follow an hospital service where the patients are children between birth and two years of age, and it may be long ere you meet with a scrofulous patient, while a month will seldom pass without your seeing rachitic patients.

After death, the scrofulous will almost invariably present organic tuberculous lesions, although they have succumbed to diseases of the bones, abdominal affections, or thoracic affections: you will almost invariably find, if not pulmonary tubercles, at least tuberculous bronchial glands, which, in young children, are the most frequent manifestation of the diathesis: seldom, very seldom, I repeat, will you find traces of tubercle in the rachitic, even in those carried off by chronic pulmonary affections.

I insist on these points, because the confusion against which I wish to put you on your guard is still too common. How many physicians, allowing themselves to be imposed upon by appearances, on looking at two children, the one having an anomalous rachitic curve, the other having a gibbosity produced by the vertebral malady of Pott, believe that both patients have the same disease. How often, when an infant with a protuberant abdomen is presented to them, do they immediately conclude that it has the tuberculous affection of the mesenteric glands and peritoneum known under the name of *carreau*.* It is important for you to be acquainted with *carreau*, which cannot be looked upon as a disease of early infancy. During my long service at the Hôpital Necker, I did not meet within more than four children under two years of age who were affected with it. It hardly ever attacks children before the age of four or five; and very often it attacks adolescents of from eleven to fourteen years. Do not forget this fact; and when consulted regarding children with large abdomen, under two years of age, the idea that the case is probably one of rickets ought first to present itself.

In a word, gentlemen, scrofula plays no part in the etiology of rickets. It is not one of the causes of which I have now to speak.

Climate has an undoubted influence upon the development of rickets. The disease is unquestionably much more common in damp cold countries than elsewhere. There can be no doubt that it is observed more frequently in Holland, in England, and in certain localities in France, than in other regions of Europe. This remark is equally applicable to man and the lower animals. Veterinary practitioners and breeders of stock will tell you that certain animals if shut up in damp places become rachitic, even when they have good alimentation.

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* Tabes mesenterica.

But of the causes of this disease, *insufficient aliment* is the most powerful. In his first works, Dr. Jules Guérin adopted this idea, that insufficient nourishment (and by that expression, vulgar prejudice understood lacteal alimentation, a too prolonged lactation) occasioned rickets and scrofula. From his usual talent of observation, he was not slow in perceiving that in exact opposition to that opinion, the children which become rachitic are not those too long suckled, but, on the contrary, those which have been prematurely weaned. It is quite correct to say the disease appears under the influence of insufficient alimentation; but then a very different meaning must be attached to the term insufficient alimentation from that generally received.

By experiments on the lower animals, the question was completely elucidated. Dr. Guérin sought to determine by experiments on animals whether rickets could be produced at pleasure. He took a certain number of puppies of the same litter; and after allowing them for some time to suck their mother, he abruptly weaned one half of them, which he fed on raw flesh, a kind of nourishment which might at first seem the most suitable for these carnivorous animals. In a short time, however, the puppies, which had continued to take the maternal milk, became strong and vigorous, whilst those which had been weaned, and placed upon an apparently substantial diet, became sad, were subject to attacks of vomiting, then became deformed in the limbs, and at the end of four or five months presented all the symptoms of confirmed rickets. From these experiments we must conclude with Dr. Guérin, that rickets is in great part dependent upon disorder in the function of nutrition, which is again dependent upon faulty feeding. Now, an untimely, is a vicious, alimentation. To feed carnivorous animals with flesh before they have passed the sucking age is vicious feeding; and from experiments made on pigs, it has been found to be equally deleterious to feed herbivorous animals with vegetable aliment, when they ought still to be at their mother's breast.

Similar results will follow similarly faulty feeding in the human subject. In children, as in the young of the lower mammalia, milk is the only aliment which is suitable: it is the only food to which the digestive organs are adapted; and the absence of teeth is itself sufficient proof of this fact. I entered into this question at sufficient length when I spoke to you on the subject of weaning, to prevent the necessity of my again expatiating upon it. In respect of rickets, attentive observation convinces me that it is most common in infants weaned before dentition is sufficiently advanced, and fed on pap, vegetables, and even meat, in place of a milk regimen, which is better adapted to their digestive aptitudes.

From what I have now said to you regarding this vicious alimentation, regarding the influence of residence in unhealthy localities, in damp, and badly ventilated places, you will be able to understand why rickets is observed more frequently among the poor, than among the comfortable classes of society. This, gentlemen, is not because the prejudices relative to weaning do not prevail as much among the rich as among the poor, but because, along with that evil influence, the latter are subjected to other influences even more baneful.

As there can be no doubt as to the causes of rickets, does it follow, that they exist in all cases? Do children, ill-fed, living in deplorably bad hygienical conditions, inevitably become rachitic, and are those who are well-fed and living under the best possible sanatory conditions exempt from the disease? Certainly, in a general manner, it is correct to say that this exemption exists; but there are exceptions to the rule; and it is important that I should point out to you the possibility of their occurrence.

You will see children who have been suckled by excellent nurses, and reared under the most favorable possible conditions, become victims to this disease; whilst some wretched infants, prematurely weaned, or who without ever having been suckled at all, who have lived on the most indigestible aliments, and been destitute of every solace demanded at their tender age, do not become rachitic. I repeat, however, that these exceptional cases are of very rare occurrence; but they are sufficiently numerous to show that, in addition to the causes I have here pointed out, there remains one still to be mentioned—one which dominates all the others—*individual predisposition.*

This predisposition is often hereditary. No one disputes the part which hereditary predisposition plays in the etiology of rickets; not that the offspring of rachitic parents are necessarily rachitic, but that it is a fact established by observation, that such offspring is more liable to rickets than others, because the disease more readily develops itself in them under the influence of immediately exciting causes; and because, in particular, that when once it is developed, it is much more difficult to arrest its progress.

Gentlemen, the influence of hereditary predisposition has always appeared to me to be much more manifest in OSTEOMALACIA, the rickets of adults, regarding which I propose now to say a few words.

Here, a preliminary question presents itself. Ought we to consider, that there is a similarity between osteomalacia and rickets? I say we ought. In my opinion, and in that of many other physicians, they are one and the same; the differences between them belong to the difference of the conditions in which the economy has been surprised. These differences have much less bearing upon the general symptoms than upon the local accidents, that is to say, upon the osseous deformities, their order of succession, and the greater rarity of fractures in the rickets of adults than in the rickets of children. For this there is a physiological reason. Osteomalacia supervenes at a period when the component parts of the skeleton have attained their complete development; and the rickets of children supervenes when the process of ossification is in progress, the result of which is, that the calcareous matter constituting the solid part of the bones, not being secreted after being absorbed under the influence of the morbid action, as it is secreted at an early age, the bones, reduced to their fibrous parts, yield more easily, without breaking, to deformities, just as occurs in bones softened by the action of an acid. As to the order of succession in which these deformities take place, the details into which I entered at the beginning of this lecture will enable you to understand why it is, that in osteomalacia the inferior extremities are first affected, and why, in rickets, the pelvis is the earliest seat of the disease.

You can also understand, that curvatures and osseous deviations will infinitely vary according to the intensity and stage of the disease, and according to the extent to which the patients have used their limbs, these deformities being subordinate to the energy and frequency of the muscular contractions, which largely contribute to produce them.

In the genesis of osteomalacia, as well as in that of the rickets of children, we can trace the influence of bad hygienical conditions, influences, however, much less marked in the development of osteomalacia than in the developments of rickets of children. The conditions which dominate in the rickets of adults, and which specially merit your attention, are peculiar to the individual.

At puberty, that is, at the period of life nearest to the period of childhood, the disease most frequently develops itself. You know the rapidity with which the body grows during the early years of life. Consider the child in the interval which elapses between birth and the end of the third year; it grows so rapidly, during that period, that were its growth to continue in the same ratio during following years, it would attain gigantic stature. From the beginning of the fourth year, however, growth proceeds more slowly up to the age of puberty, when it takes a fresh start; and then it is not unusual to see the individual grow five or six inches in twelve months; then, also, the skeleton undergoes a modification similar to that which takes place in early infancy. When, these conditions existing, under the influence of immediate causes which generally escape notice, osteomalacia supervenes, it presents the greatest analogies to the rickets of children—I say *analogies*, not similitudes.

After puberty, there are other circumstances which may favor the development of the disease. Thus, it is not uncommon to see women attacked with rickets after having had several children, or even, it may be, after a first confinement, the disease beginning immediately after delivery. This influence of pregnancy, particularly of repeated pregnancies, is mentioned by many authors; and Dr. Beylard says, in his thesis,* that in 36 rickety women whose cases he collected, 15 had had children, 5 had never been mothers, and in respect of the other 16, there was no statement as to whether they had or had not had offspring. Dr. Beylard notices as a remarkable fact, that several of these women had become pregnant for the first time about the age of thirty.

You are acquainted with the researches of Dr. Ducrest in relation to the modifications experienced by the entire organism, particularly by the osseous system, during gestation.† It appears from these researches, that the state of pregnancy causes a certain amount of softening of the bones: the secretion of the solid materials which enter into their composition undergoes disturbances and notable deviations. These disturbances are indicated by the appearance of kyestein, a sort of scum which appears on the surface of the urine of pregnant women; and which, according to M. Gubler, is a layer of ammoniaco-magnesian phosphate, on which there grows a cryptogamic vegetation, when the urine is allowed to remain at rest: this phosphate is found in excess in the urine of rickety children. These abnormal states of the secretion of calcareous matter are characterized by the bones, particularly those of the cranium, frequently presenting a remarkable thickness, caused by deposits in the form of osseous stalactites called *osteophytes*, which have been also found by M. Follin on the bones of the pelvis. There exists, therefore, in pregnant women, a certain kind of rickets, of which osteomalacia may be considered as a more advanced stage.

It appears that in a woman about to give birth, there occurs something analogous to that which takes place in plants when they are going to flower and fructify. At that period, plants undergo remarkable changes: thus, for example, the root of beetroot contains a large amount of sugar till the plant is going to blossom and form seed, when the sugar disappears. Here, then, is a remarkable change in the organization of that vegetable. In female animals at the time of rut, modifications of an equally remarkable nature are observed. Both in vegetables and animals, then, the great and important function of reproduction is announced by momentous phenomena. Woman forms no exception to this great law of nature. All her organism is in movement, if I may use the expression: all her systems, the osseous system included, undergo more or less considerable modifications: the modifications, however, are transient, for as soon as pregnancy has terminated, everything returns to its former state. It happens, however, that when these modifications have proceeded to extremes, if one may so speak, a return to the normal state is less speedily accomplished; and, under the influence of particular causes, the movement continues. The modifications to which the mother has been subjected, no longer responding to their original intention, operate injuriously on her: the modalities, even those which are physiological and transient, become pathological, constituting morbid states of greater or less gravity, manifested by symptoms which become more and more characteristic.

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* BEYLARD: Du Rachitis, de la Fragilité des Os, de l'Ostéomalacie. Paris, 1852.

† DUCREST: Archives Générales de Médecine: 4me Série, t. iv.

The case of a patient whom I treated in the Hôpital Necker, in 1848, a woman, aged 48, named Rehbin (which Dr. Beylard has published at full length), may be given as a complete and typical example of osteomalacia.

This woman considered that the malady which induced her to seek aid at the hospital had begun at the time of her first pregnancy. Her pregnancy took place at the age of thirty-two, and immediately after marriage. At the same epoch, her health began to be seriously deteriorated. Previously, however, the patient (who had always lived under very bad hygienical conditions), had suffered from nervous symptoms, connected with chlorosis dating from her twelfth year.

The symptoms which supervened on her becoming pregnant, were attacks of vomiting (to which we must not attach too much importance) and a general debility, accompânied by wandering pains in the back of the neck, the shoulders, the loins, the pelvis, and the lower extremities. These pains increased up to the end of pregnancy, and were characterized by shootings through the pelvis and thighs. However, notwithstanding her debility, the patient continued to walk, and to go out, up to the eighth month of gestation, when œdema of the lower extremities obliged her to keep her room.

Labor, which was natural, lasted twelve hours: the infant was at the full term, and well-formed. The mother left her bed at the end of fifteen days; but was soon attacked by fever, which recurred two or three times in the twenty-four hours: this state of fever, characterized by slight attacks of shivering, followed by moistness of the skin, lasted the whole period of lactation, that is to say for seventeen months.

The pains became more intense, always specially seated in the sacrum, manifesting themselves, likewise, by continual shootings through every part of the body, from the face, cheek-bones, and jaws, down to the feet and hands. Even gentle pressure on the bones increased the pain.

Six months after her confinement, the patient experienced difficulty in walking; and began to stoop. It soon became impossible for her to go about her house, or attend to her child. She hardly ever left home: and at the date of her giving up suckling her infant, she could only painfully drag herself along by leaning on the walls and furniture of her room. If she left the house, she was obliged to have recourse to crutches.

After a fall on the street, she took to her bed, which she kept several months. No fracture, however, was discovered. The pains became more intense, and she lost the power of moving. In changing her position, she required to take the greatest precautions. She remarked, that her knees and feet turned outwards.

The general health continued good, to this extent, that digestion was accomplished in a normal manner, and menstruation became re-established with its accustomed regularity.

Two years after the birth of her first child, the woman Rehbin became again pregnant. At the end of the third month, her sufferings recommenced; and from that time, her pains became constant and general, occupying all the articulations, and becoming intensified on the least movement. She was compelled to remain at absolute rest, either lying down, or seated, supported by pillows, in an arm-chair: she reached her full time. Labor, which was otherwise natural, lasted twenty-four hours, that is, twice as long as on the first occasion. For some months after her confinement, a certain amount of amelioration took place: she was able to walk with the aid of crutches, which it was found necessary to shorten. The organic functions were performed naturally: menstruation was re-established six weeks after delivery. The pains only remained, and they had become more frequent, occupying the whole of the osseous frame, particularly the right side, and in a still more special manner, the pelvis and continuity of the limbs. So they continued, without much increase in intensity, till about fifteen months had elapsed from the birth of the second child.

The patient then became pregnant for the third time. Tingling sensations and feelings of numbness, which were experienced in the limbs towards the end of the first pregnancy, and had manifested themselves from time to time, now lasted for several hours, so as to lead the patient to fear that she was falling into a state of paralysis. These sensations were stronger in the upper extremities than in the legs and feet. The limbs were likewise liable to painful contractions which had occurred from the beginning of the malady, and returned irregularly. Under these circumstances, the woman reached the full term of her third pregnancy: she was then thirty-eight years of age. Labor, as on the two former occasions, terminated naturally; but this time it lasted seventy-two hours. The child, though strong and well-formed, died when two days old.

After that confinement, the disease became greatly aggravated: the pains were constant: they became intolerable with any movement or on the slightest pressure: to such an extent was this the case, that the patient could hardly bear the weight of her clothes or blankets. During the night, the pains were more severe than at any other time, when they extorted cries from the unhappy sufferer.

Her figure had been crooked for a long time; but from the date of this third confinement, the deformities became great.

The face was shortened: the cheek-bones had acquired increased prominence: the superior maxilla projected so much as not to correspond with the lower jaw: from this cause, and the loss of several teeth leaving a gap, mastication was difficult, particularly when the food offered resistance to chewing.

The head, regarded as a whole, was, however, not deformed; although the cranium was exceedingly sensitive.

The cervical region was shortened, in consequence of increase in its curve forwards, and the squeezing together of the vertebræ.

The thorax was flattened from before backwards, and shortened from above downwards, being inclined towards the pubes. The ribs were imbricated, lying over one other in such a way as almost wholly to efface the intercostal spaces.

The pelvis was flattened in the same way as the thorax, that is to say, in its antero-posterior diameter. The iliac bones were turned outwards in a very obvious manner.

The thighs were shortened and curved, the hollow of the curve being directed backwards and inwards.

Over this curvature, the integuments were hard, and formed very prominent folds.

The legs and upper extremities presented no change, although for a long time the patient could only half supinate the hand.

The malady grew worse; but, nevertheless, the functions of organic life were not disturbed: there was no loss of appetite, digestion was easily performed, and the bowels were regularly opened every day. Her diet was wholesome, being composed of flesh and vegetables, with wine as the usual beverage.

This state of matters had continued several years, when a slough over the sacrum complicated the case. The patient was then admitted to my wards in the Hôpital Necker. As I have already told you, that occurred in 1848. The malady had then existed sixteen years; and during the latter half of that period had made great progress.

I was at once struck with the deformities of which I have now given you a condensed account. I was struck with the patient's diminutive stature: she told us that her height was formerly $1^{m\cdot}$ 78, but was now reduced to one metre. So great was the deformity of the pelvis, that when I wished to make a digital examination, with a view to determine its internal dimensions, I found it impossible to pass my finger into the vagina, and was only able to introduce a pretty large sound.

Well, gentlemen! this woman recovered: she recovered under the influence of cod-liver oil, which I prescribed for her from the time she came into the hospital, and which she continued to take long after she returned to her own home.

You understand that this recovery took place as recoveries take place in rickets. I was fortunate enough to arrest the progress of the disease, so as to procure consolidation of the bones: but I had no ground for hoping that I could remedy the deformities, which were necessarily persistent. Dr. Beylard, however, who saw the patient in 1851, ascertained that she had slightly regained her stature: on measuring her, he found that she had regained 43 centimetres: at that date, her general health was excellent, and she had become quite plump: except menstruation, which had not recurred for two years, all her functions were performed with perfect regularity. She complained, however, of some wandering pains, and of shooting pains supervening on sudden atmospheric changes. Although this case is full of interest in all its aspects, I must refer you for farther details to Dr. Beylard's thesis.

In another case, which occurred in my private practice, I likewise saw rickets supervene, consequent to, but several years after, a first confinement. The lady was married at twenty, and was delivered in 1831 of the only child she ever had; and which, in the following year, when enjoying perfect health, was carried off by an attack of cholera of a few hours' duration. This event occasioned profound grief to the mother, which time was unable to moderate.

A year afterwards, she fell into bad health. She had attacks of menorrhagia, recurring at short intervals; and she complained of incessant pains in the loins and pelvis. These pains soon extended to the back; and in 1835, an obvious rachitic deformity was perceived, the spine being curved forwards. Some months later, the curvature was more marked, and was likewise lateral: one shoulder also projected more than the other. These deformities went on increasing: in 1840, five years after they had commenced, seven years after the beginning of her malady, and nine years, consequently, after her confinement, they were exceedingly conspicuous. The projection of the shoulder was great: the stature was much diminished: there was collapse of the thorax from above downwards, to such an extent that the ribs, as it were, overlapped one another. At this period, the pains were such as to disable the patient from walking.

Now, for the first time, the nature of the malady was recognized. Forthwith, she began to take cod-liver oil. The anticipated success was obtained from that medication. In two months, the pains were sufficiently mitigated to enable her to walk, and after two years of perseverance in the treatment, it being only temporarily discontinued from time to time, so as to avoid irritation of the digestive canal, the cure was complete, with the exception of the irremediable deformities.

In June, 1863, you may have seen, in bed 3, St. Bernard Ward, a very rare case of rickets, or acute osteomalacia, in an old woman of seventy years of age, who came into the hospital with cough, dyspnœa, and high fever. This patient complained of suffering horribly in certain parts of the sternum, and throughout nearly the entire extent of the ribs. At first, I supposed that she was exaggerating her sufferings. She shrunk from the hand when any attempt was made to examine her by palpation or percussion. This was something quite unusual: the pain was as intense as in the most violent neuralgia; but it was not circumscribed as in neuralgia. On the contrary, it was diffuse and bilateral, which is unusual, intercostal neuralgia being generally seated in the left side. Then, again, there was fever. On percussing the chest, with a view to discover whether there existed fulness indicative of possible pleuritic effusion, I thought I perceived slight crepitation, while, at the same time, the ribs seemed to bend under the pleximeter. To make myself quite sure on this point, I pressed somewhat strongly on the convexity of the ribs; and there was then no doubt that the bones bent and crepitated under pressure: very acute pain was at the same time excited. Light now began to dawn; and I thought it probable, notwithstanding the rarity of such an affection, that it was rachitic softening of the ribs. In that case, the pain ought to be most acute at the junction of the ribs with their cartilages: and it was so. In these situations, there was tumefaction. Pressure produced acute suffering, and at the same time enabled one to perceive an abnormal mobility: to a certain extent, the cartilage could be made to ride upon the rib. Pursuing my investigations, I also made out that the sternal pain was not uniformly distributed, but was located at the junction of the first and second pieces of the sternum, and at the union of the second piece with the xiphoid appendix.

I therefore found myself confronted with painful softening of the ribs and sternum, and pain seated in the favorite seats of pain in rickets, that is to say, in the articulation and epiphyses. Could any doubt have remained, it would have disappeared on a more complete examination of the woman, for she presented a very strong gibbosity. Her vertebral column was curved in the form of the arc of a circle of great diameter; and was not bent in an acute angle as in Pott's disease. The articular apophyses of the ribs were bulky, and the long bones were slightly deformed. Finally the patient stated that she had been rickety [*nouée*] in childhood.

She had dyspnœa, due in part to the pain occasioned by the respiratory movements, which caused the softened ribs to ride on one another, and in part also to a slight bronchitis, from which she had suffered for some days. She suffered habitually from palpitation, due perhaps to displacement of the heart, which was not enlarged.

The thoracic pains, which had set in six weeks previously, commenced with febrile excitement. The fever was constant, tolerably moderate during the day, liable to exacerbations in the evening and at night. Profuse sweating occurred towards morning.

I placed the patient on a cod-liver oil regimen, and tried to subdue the fever by administering the tincture of digitalis and the alcolate of aconite.

Fourteen days after admission, that is to say, on the 7th July, she had violent shivering, and on the following day were perceived dulness, crepitation, and an obscure blowing in the right axillary region. Next day this poor woman succumbed under an advancing pneumonia.

At the autopsy, we found a very altered state of the ribs and sternum. There remained only one very thin plate of compact tissue; and the superabundant spongy tissue was gorged with blackish blood. So extensive was the softening of the ribs that they could be bent in all directions and cut by a knife with the greatest ease. The sternum was in a similar state of softening.

It is a remarkable fact that the long bones of the limbs presented no similar alteration; therefore, notwithstanding this woman's advanced age, the rachitic disease had, as in the infant, begun in the chest.

The whole of the upper half of the right lung was hepatized, and all the bronchial tubes were inflamed.

This case, in my opinion, decisively demonstrates the identity of rickets and osteomalacia; the lesions were certainly those of rickets, but the patient's age was that at which osteomalacia is usually observed. Moreover, she died of inflammation of the lungs, as generally happens in the severe form of rickets, as if the impediment to the respiration, by causing passive congestion, produced inflammation of the pulmonary parenchyma.

Gentlemen, I am also strongly led to believe that rickets and osteomalacia are the same disease by the fact that both are wonderfully combated by the same medication.

This medication may be considered as really heroic in the *treatment of rickets;* it consists in giving cod-liver oil and, in a more general way, fish-oil. Though employed from time immemorial by the people in England, in Holland, in Westphalia, and on the northern coasts of Germany, it was never till recently prescribed by scientific practitioners. At the commencement of this century, two physicians on the other side of the Rhine, Schenck and Fehr, published some very interesting observations in relation to its use; but the facts to which I refer remained unknown in France till 1827, when Bretonneau, who, like everybody else, was ignorant of them, was led, in the manner I am now going to relate, to try cod-liver oil in rickets.

At that date, there was a Dutch family at Tours, under the medical care of the eminent practitioner. One of the children, fifteen months old, became rachitic in an extreme degree. For four or five months, Bretonneau fruitlessly contended with the malady, exhausting the entire series of medications then recommended, when the child's father told him that his eldest son had been cured, in Holland, of the same malady by fish-oil, a popular remedy. Bretonneau advised the same medicine to be given to his young patient; the success was so incredibly rapid, that my illustrious master was quite struck by it.

Encouraged by the result in this first trial, he repeated the experiment on other rachitic subjects; and it was when thus occupied with researches into the action of cod-liver oil, of the good effects of which he had satisfied himself, that he had the pleasure of finding that similar good results had been already obtained by the German physicians whose names I have just mentioned.

Bretonneau informed Dr. Guersant, Professor Jules Cloquet, and me of his curious observations; and then, we, in our turn, administered the remedy to the rachitic children we had to treat. The results were as completely satisfactory as those of which we had been informed. The use of cod-liver oil soon became general; and at the present day, there is not a physician who has not recourse to it under similar circumstances.

How does this medicine act? Is it by specific anti-rachitic virtues such as mercury and iodide of potassium possess in syphilis? I do not believe that it is. Its virtues essentially depend upon fish-oil being an analeptic tonic of a superior kind; that is to say, that it acts as a fatty body, and perhaps by being a fatty body combined with different substances which possess the properties of exciting tonics, such as iodine and phosphorus, and combined in the proportions and according to certain modes which chemical analysis might be able to disclose, but which, nevertheless, synthesis cannot reproduce. It resembles, in this respect, all the compound medicaments which we find prepared by the hand of nature: and thus, as I have had occasion to tell you in my lecture on dyspepsia, when speaking of the natural mineral waters, the pretensions of those who wish to substitute pharmaceutical medicines for them is as senseless, in my opinion, as it would be to combine the component parts of wine with a view to produce a substitute for natural wine, even for the worst natural wine. Cod-liver oil, besides being an aliment, is a stimulant perfectly suited to the more or less deteriorated state of the organism.

It does not, however, enjoy a monopoly of these renovating powers. We may perfectly well substitute for it ray-oil, herring-oil, and the fish-oil of commerce. For my part, when I have to treat the children of families whose means require to be considered, I prescribe the oil used by shoemakers, which is much less expensive than either ray or cod oil. However repugnant this oil may appear, the little patients generally accommodate themselves to it as well as to the other oils. I would add in respect of cod oil, that that which is not purified—the brown oil—is far preferable to all the pale kinds, the advertised prospectuses of which proclaim their superiority.

Clinical experience testifies in favor of this opinion; and clinical results are corroborated by those obtained in physiological experiments made in relation to the assimilation of those oils by the economy.

You are aware, that while vegetable oils are either not assimilated, or are only assimilated in so small a proportion that after their reception into the stomach they become converted into emulsions, and are to a great extent passed by stool, the animal oils are assimilated. There are, however, differences between the two kinds of oil which must be pointed out. The more impure and rancid that the animal oils are, the more perfectly are they assimilated, the more does the digestive canal become accustomed to digest them: on the other hand, the purified oils, such, for example, as the pale cod-liver oil, after they have been taken for a certain time, cease to be so well assimilated.

In northern countries, on the shores of the Baltic Sea, where it is a common practice with the people to give to weak children and valetudinarian adults whale-oil and fish-oil indifferently, you may be sure that much care is not bestowed on the purification.

Marvellous and unquestionable though the good effects of fish-oils are, there are circumstances in which, from the individuals not being able to bear their use, or from their having an invincible repugnance to them, we are obliged to substitute other substances.

There is one substitute, *butter*, within the reach of all, from which excellent results are obtained; but to obtain such results, it is necessary that a large quantity, sixty grammes at least, be taken in the twenty-four hours.

So as not to shake the confidence of relatives in a medication so simple as butter, I add to it some of the constituents of fish-oil. The following is the formula which I generally prescribe:

Take of—

Fine fresh butter, . . .	300 grammes,
Iodide of potassium, . . .	15 centigrammes,
Bromide of potassium, . .	50 "
Chloride of sodium, . . .	5 grammes,
Phosphorus, ...	1 centigramme.

Mix them *s. artem.*

This quantity, spread on slices of bread, ought to be taken in three days.

When children have a disgust for this preparation, fowl-fat may be used as a substitute for it, taken in the same quantity, and in the same manner; or recourse may be had to an old practice, still adopted in Scotland and England, of giving fried bacon, or the melted fat of ham spread on bread.

Well-smoked ham, eaten raw, is also recommended as a dietetic auxiliary in the treatment of rickets and osteomalacia: its use combined with that of flat beer [*bière non-fermentée*] has been much lauded, particularly in Germany.

The use of fats and animal oils form the basis of the treatment of rickets and osteomalacia. Need I add, that the patients must be placed in the most favorable hygienical conditions?

They must, as much as possible, live in the country, in the open air, and in dry sunny places. Though it be necessary during the period of the increase of the malady to avoid movements, lest fractures be produced, yet at a later period, when the bones have become consolidated, regular exercise is indicated. Saline baths, particularly sea-water baths, will be exceedingly useful.

It is unnecessary to say, that the thing of paramount importance is an aliment essentially tonic and reparatory: but, gentlemen, in respect of aliment, I must impress on you the necessity of its being varied according to the age of the patients. For very young infants, for those in whom the first dentition is not completed, milk, particularly the milk of a good nurse, must constitute the exclusive food. I attach so much importance to this rule, that I do not hesitate to prolong lactation beyond the usual term. By doing so, and without the use of any other means, I have seen the recovery of rachitic children. When, from the child having been prematurely weaned, it is averse to resume taking the breast, and when the milk of the cow or any other animal causes indigestion, leading to the intractable diarrhœa of which I have spoken to you, I resort to medication with raw meat.

In infants, after the first period of infancy, and in adults, the regimen ought to consist of a varied combination of animal and vegetable food, care being taken that the former predominates, and that farinaceous vegetables are abstained from, as they are of much more difficult digestion than fresh herbaceous vegetables.

LECTURE LXXXVII.

TRUE AND FALSE CHLOROSIS.

False Chlorosis, or Tubercular Anæmia.—Ferruginous Remedies must not be prescribed in False Chlorosis.—Iron arouses the Tuberculous Diathesis, and promotes its manifestations.—The Tuberculous Diathesis ought to be treated by Bitters and Arsenic.—When the Tuberculous Diathesis exists, Fistula in Ano and Leucorrhœa ought not to be cured.—False Chlorosis and Syphilitic Anæmia.—The Blowing Sound in Anæmia is Arterial and Simple: in True Chlorosis it is Double, i. e., Arterial and Venous.—Action of the Vaso-motory System on the Production of Vascular Bellows-Murmurs.—True Chlorosis is a Neurosis, Alteration of the Blood being Secondary.—Treatment: Hygienical Conditions.—Iron.—CINCHONA.

GENTLEMEN: You must have been surprised to see me prescribe medications differing so much from one another for several women now in the clinical wards, all of whom may appear to you to be suffering from chlorosis. In point of fact, they are all pale, cachectic, and neuralgic: in nearly all of them, you hear the blowing murmur in the vessels of the neck: and in these seemingly identical cases, I institute very different kinds of treatment.

You perceive, therefore, gentlemen, that I am far from looking on anæmia and chlorosis as the same disease; and although, as I readily grant, chlorosis is accompanied by profound anæmia, it by no means follows that anæmic subjects are chlorotic. Decoloration of the blood, and modifications in the relations of its elements, are observed in many pathological conditions differing much from one another; and I am convinced that the confusion introduced by some of your teachers into the diagnosis of the diseases which have anæmia as an element in common, daily sacrifices victims.

You see in bed 25 of St. Bernard Ward, a woman, twenty-two years of age, who pants for breath on making the slightest movement, is gastralgic, dyspeptic, irregular in her menstruation, and in the vessels of whose neck is to be heard the blowing murmur. At the time of her admission to the hospital, it was difficult not to consider her as chlorotic; but she told me that, some time previously, she had had slight attacks of hæmoptysis, and that she had been frequently troubled by a short cough, coming on generally after meals. On examining the chest by percussion, with the most scrupulous care, nothing abnormal was revealed; but, by attentive auscultation, it was discovered that the pulmonary expansion was less in the right infraspinous fossa than in the left. I heard neither râles nor vocal resonance; and I am convinced that two months earlier these signs, so equivocal and so little marked, were entirely absent. I was, however, on my guard: and to-day, when you can recognize cavernous râles and gurgling, accompanied by emaciation and disturbance of the digestive and uterine functions, you can make no mistake as to the sad reality.

In bed 3, of the same ward, there is (admitted for the third time) a young girl, with all the apparent signs of chlorosis; and yet, during the eighteen months that she has been in my wards, I have persisted in refusing to give her ferruginous preparations, which some of you wished me to prescribe for her. She also has false chlorosis: at the apex of one lung, she has feeble inspiration; a circumstance which occupies my attention, and alarms me: she has been treated by ferruginous medicines, from which she has always experienced bad effects, and has observed that the gastralgia and feelings of discomfort have been invariably aggravated by their use.

The influence of preparations of iron on the health of women reputed to be chlorotic is a subject which demands very serious consideration. Speaking generally, it may be said that iron, prudently and gradually administered, is well borne by true chlorotic patients. But if in a disease which has every appearance of chlorosis, the physician fail after having varied both the choice and doses of the preparations of iron, he ought to suspect that he has made a mistake: and, if he observe attentively, he will almost always discover that some formidable affection has already revealed its existence by evident signs.

I do not say that iron does not sometimes give an appearance of flourishing health to women threatened with tuberculosis; but in such cases, some signs will present themselves which will show the practitioner that he is on the wrong tack. When in a girl apparently chlorotic, languid, and devoid of energy, the iron rapidly rewakes the muscular power and the appetite, while at the same time it notably accelerates the pulse, and gives rise to a sort of feverish excitement somewhat analogous to intoxication, there arises reason to fear that a continuance of the iron will induce an outburst of fever accompanied by local disorders, frightfully rapid in their progress.

When a very young physician, I was called to see the wife of an architect suffering from neuralgia, a pale woman, presenting every appearance of chlorosis: I prescribed large doses of preparations of iron, according to Hutchinson's method of treating neuralgia. In less than a fortnight, there was a complete change: the young woman acquired a ravenous appetite, and an unwonted vivacity: but her gratitude and my delight did not last long. The excitement soon became fever: and the restored color of the cheek became every evening after dinner more ardent than it had been when she was in good health. A short cough supervened; and in less than a month from the commencment of the treatment, there appeared signs of phthisis which nothing could impede.

The first case of galloping consumption which I had to deplore in my practice, occurred under nearly similar circumstances: the patient was a girl of fifteen, who after a mild attack of dothinenteria fell into a state of anæmia and prostration, which I considered chlorosis. I administered ferruginous remedies, which rapidly restored her to florid health: and although there was nothing in the family history to lead me to fear the coming calamity, she was simultaneously seized with hæmoptysis and menorrhagia, and died two months afterwards with the symptoms of phthisis, which had advanced with giant strides.

I do not blame the iron for having caused this calamity; but I do blame myself for having cured the anæmia, a condition, perhaps, favorable to the maintenance of the tuberculous affection in a latent state.

Nevertheless, gentlemen, inasmuch as under the influence of bitters, arsenic, hydrotherapy, and sea-water baths, I obtain equally favorable results in women who seemed to me to have been in conditions quite the same as those which I have just been describing; and, inasmuch as I have not seen produced in them that general excitement, the prelude, cause or effect, of the tuberculous dissolution, I am constrained to impute to iron some of the evil consequences which I had to deplore.

For many years past, I have looked on it as a duty, when I have had to do with persons hereditarily predisposed to tuberculosis, not to push too far a medication under which all the functions seemed to regain a powerful energy; and though in these same persons, physicians less timid than I am, dare to seek and to obtain results which I dread, I avoid their practice, because under it I have often seen diseases show themselves which might otherwise have remained for a long time in the germ.

Gentlemen, a book has been written on the advantages of bad health; and without guaranteeing all that may have been said on that subject, I would wish to appropriate some of it, and tell you that you will avoid distressing occurrences in your practice by allowing patients predisposed to tuberculous disease to retain maladies from which it may seem easy and opportune to deliver them. It has been impressed upon you by your surgical professors that it is dangerous to cure anal fistula in tuberculous subjects: and it is now a long time since I laid down for myself the rule, not to interfere in young women predisposed to tubercle with leucorrhœa, so common in them, sometimes so inconvenient, and so easily cured. In cases in which I have refused my consent to such patients being treated for this indisposition, and in which other practitioners, more venturesome, have acted otherwise, there have soon supervened accidents till then retarded.

In respect of fistula and leucorrhœa, I have no objection to the theory of revulsion being invoked, yet it has seemed to me that the relative debility of the patients was to them a protection against the sudden outbreak of tuberculous accidents. The older I grow in the practice of my art, the more convinced do I become, that in the same family in which the hereditary taint of scrofula exists, the women who are anæmic, or affected by indispositions which maintain them in a state of precarious health, pay their debt to hereditary taint at a later period than those who are seemingly in the enjoyment of the most complete health.

Gentlemen, I am well aware that very few practitioners concur in these views; and still better do I know the extent to which I have been subjected to the criticisms of those who make use of iron so frequently (and as I think) so inopportunely; but my conviction, far from being lessened, is confirmed more and more every day.

Let us now, gentlemen, recall the case of a woman, aged thirty-two, who lay in bed 22 of St. Bernard Ward. On admission, she was in a state of extreme anæmia, having blowing sounds in the vessels, chronic diarrhœa, and an exceedingly profuse leucorrhœa. I fought long but fruitlessly against the abdominal affection. One day I moderated it, the next it reappeared. For four months I used with great perseverance all the therapeutic measures with which I am in the habit of successfully treating diarrhœa. Nevertheless, a temporo-facial neuralgia supervened, an event not at all surprising in a person so exceedingly anæmic; but the neuralgia had this peculiarity, that it returned every evening, increased in severity during the early part of the night, and ceased at daybreak. This nocturnal return of the malady made me suspicious, and led me to fear the existence of constitutional syphilis, with which, notwithstanding her protestations to the contrary, I felt convinced she was affected.

Some time afterwards, a very painful exostosis on the crest of the tibia declared itself. I no longer regarded the denials of the patient. I prescribed Van Swieten's liquor (bichloride of mercury); and you have seen with what rapidity the health of this woman, so seriously compromised, was re-established. You have seen her color return under the influence of mercury, a medicine which so radically alters the crasis of the blood in healthy subjects.

Nearly at the same time that this woman was under treatment, you saw, in bed 16 of our nursery ward, a young woman who was pale, and had all the symptoms of chlorosis. She had no sign of venereal disease: but the child she was nursing had syphilitic symptoms and hypertrophy of the liver; it was even paler than its mother. She had been uselessly treated by ferruginous preparations. Mercurials, followed by iodide of potassium, restored her to the appearance of perfect health. Bear in mind, in relation to this subject, the interesting researches of Dr. Grassi regarding the blood of persons affected by syphilis. This distinguished chemist has demonstrated, by numerous analyses, that in the secondary period of syphilis, there is a diminution in the number of the blood-globules.

I have been anxious to place before you these facts, to let you see that a multiplicity of causes may alter the constitution of the blood in such a manner as to simulate chlorosis; and I have done so for the special purpose of guarding you against the hackneyed treatment by ferruginous medicines, which in the majority of cases is insufficient, is moreover sometimes useless, and more frequently dangerous.

When my honorable colleague, Dr. Bouillaud, first called the attention of practitioners to the murmur which he called the "*bruit de diable*," the "*bruit de souffle musical, et à double courant*,"* he did not suspect that it would become with many practitioners a frequent cause of perilous diagnosis.

Look, gentlemen, at the patient who now occupies bed 29 of St. Bernard Ward, whose malady is true chlorosis. Study in this case the vascular murmurs, and specially study the other phenomena which constitute this curious disease. Let me in the first place speak of what takes place in respect of the vessels.

On applying the stethoscope below the middle of the clavicle, we have a rather dry blowing murmur accompanying the first sound of the heart. But during the ventricular diastole, the murmur assumes a more musical character, louder, and resembling the purring of a cat when it is being caressed, or the noise of a spinning-wheel. Between the first and second sounds of the heart, the murmur never altogether ceases. The name given by Dr. Bouillaud, viz., "sustained blowing-sound" [*bruit de souffle avec renforcement*] is therefore appropriate; but it is important to remark that the continuance of the sound takes place during the cardiac diastole. Along with many other physicians, I believe that the first sound is in the arteries and the second in the veins. On compressing, by the application of a thread, the lateral part of the neck, above the point where the stethoscope is applied, in such a way as to interrupt the current of venous blood, we find that the second sound ceases.

Whatever there may be in this explanation, it appears to me that there are two classes of blowing sounds of the neck; viz., the simple sounds, purely arterial, and the double-current sounds [*bruits à double courant*] so well investigated by Dr. Bouillaud. The first belong to anæmia, whatever may be the cause of the anæmia; the others are peculiar to chlorosis. They are so decidedly chlorotic sounds that they precede or follow the most ordinary manifestations of chlorosis. We have at this very moment, in bed 3 *bis* of St. Bernard Ward, a young woman who is typically chlorotic. You recollect that when she was admitted to the hospital, she presented, with the utmost exactitude, all the symptoms of chlorosis. Ferruginous treatment rapidly re-established her health; the complexion and the mucous membranes regained color; the veins were traced out by blue lines; and all the functions were restored in their integrity. However—as I have often asked you to verify by your own personal observation—the double-current blowing sound still continues in the vessels of the neck, and though it is not so strong as it was two months ago, it is not the less distinctive; on the other hand, in all the anæmic women in the wards, you can only hear the simple murmur accompanying the first sound.

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* **Bouillaud**: Traité Clinique des Maladies du Cœur, t. i. Paris, 1841.

It therefore follows that, in chlorotic subjects, the vaso-motor nervous system is modified in a manner altogether special, and that this modification is, to a certain extent, independent of the constitution of the blood. It bears no relation to the greater or less number of the blood-globules, because, in the first place, anæmic subjects seldom have the double-current blowing murmur, and, secondly, because chlorotic subjects, after the constitution of their blood is restored to its normal state, long continue to present this sign.

When, as the result of clinical observation, I was led to think that differences existed between the anæmic and chlorotic blowing sounds, and that these sounds may continue for a variable period after the cure, I was not acquainted with the conclusions arrived at by Dr. Peter, as stated in an unpublished memoir on this subject.

In this work, Dr. Peter has established, upon a basis of sixty-three cases, that the vascular blowing sounds are not entitled to retain that importance which has been assigned to them in the diagnosis of anæmia and chlorosis. It has indeed been shown by the chemical analyses of MM. Andral and Becquerel, that vascular blowing may be perceived in cases in which the number of blood-globules is not below the numerical physiological average. As, on the other hand, it has been shown by Dr. Peter, that the symptoms of anæmia and chlorosis may exist without there being any vascular blowing, is it not natural to conclude, that its presence or absence is only of secondary importance in the pathological conditions which we are now considering?

Dr. Peter has also shown, that the blowing sounds may disappear and reappear within a few hours, evidently, therefore, without the crasis of the blood having undergone any sensible modification. Since then, vascular blowing may be heard when there is a normal constitution of the blood—when there exists the functional disturbance of anæmia or chlorosis—since vascular blowing may appear and disappear during the same examination without there being any possible alteration in the condition of the blood, we must attribute this sound, in a certain number of cases, to something else than the state of the blood. May not the sound be caused by a peculiar state of the vessels, by contraction of their parietes? This is a view which brings us back to the opinion of Laennec, who attributed the vascular blowing heard in the hypochondriac regions to spasm of their vessels, a spasm which he thought, and which I think, depends upon the action of the vaso-motor system of nerves.

Dr. Peter, with Laennec, believes in vascular spasm: with M. Chauveau, he believes that the phenomenon of the contracted column of blood [*la phénomène de la veine fluide*] is the condition which occasions vascular blowing; or in other words, that the spasm of the vessel produces constriction, which constriction in its turn produces the contracted column. As you know, according to the experiments of Savart, there is always produced a contracted column when a liquid in traversing a tube passes from a constricted to a wider portion.

From the reasoning and observations of Dr. Peter, it follows, that the vascular blowing sounds do not possess the semeiotic value hitherto attributed to them: they are by no means pathognomonic of anæmia, and possess a value which is only relative. For, adds Dr. Peter, as the wall of the vessel derives its nervous supply from the vaso-motor system, nervous people are subject to vascular spasms, and as anæmic people are in a high degree nervous, it follows that it is principally among them that vascular blowing sounds are met with—and here vascular blowing is a very indirect proof of anæmia. But as a person may be anæmic without having these vascular spasms, and have vascular spasms without being anæmic, it equally follows, that vascular blowing cannot be a direct proof of the existence of anæmia.*

In an interesting discussion, raised before the Société Médicale des Hôpitaux by Dr. Parrot, that able clinician also reduced the usually accredited value of vascular blowing as a sign of an altered state of the blood. He also believes, that it is the wall of the vessel, and not the condition of the circulating fluid, which produces the blowing sound. He also observed that these sounds are fugitive, appearing and disappearing during even the same examination, according to the position of the patient: he adds, that they are usual in children, common in aged persons, and almost invariably met with in the nurses at the *grand bureau*, who are generally robust women, recently arrived from the country, and free from all signs of debility or nervousness.

In explaining the production of the vascular blowing sounds, Dr. Parrot attaches very little importance to the condition of the blood: he believes that the sounds are produced in the veins and not in the arteries, and that they are due to insufficiency of the venous valves, an insufficiency which does not admit of any doubt in respect of the valves of the internal jugular vein. The greater frequency of vascular blowing sounds on the right side is attributed by Dr. Parrot to the almost rectangular passage of the vessels on that side, and to the shortness of the passage to the heart.†

Dr. Parrot does not go so far as to allege that vascular blowing is valueless in the diagnosis of anæmia and chlorosis: but he says, that to give it a decided value, it must be very intense and accompanied by a purring sound.

In his beautiful work on anæmia, Professor G. Sée, who locates vascular blowing in the arteries, also attributes it to the nervous influence in operation from the time that the blood has begun to be altered. There is diminution of the tension of the blood consequent upon relaxation of the vessels. The blood flowing more irregularly than by the capillary extremities, the pulse becomes quicker, and a maximum abnormal sound is produced. It is by the fluid column that Dr. Sée, in like manner, explains vascular blowing. He is also of opinion that the material conditions which produce vascular blowing sounds are permanent weakness of the heart's action, feeble tension of the arteries, the brisk and accelerated flow of blood which passes with ease from the arterial trunks into the minute arterial branches. In the fever of the pyrexiæ, there is a constant fatigue of the muscles of the vessels, and their tension is always enfeebled; and then, we hear vascular murmurs: they are never, however, so distinct as in chlorosis, because the blood is not impoverished in globules. Blowing sounds are likewise produced in hysteria and hypochondria: in these affections, they are derived from a direct excitation of the heart, but which is not sustained by any alteration of the blood. Arterial blowing, then, does not possess any absolute diagnostic value, says Dr. Sée, and is in itself insufficient to establish the existence of chlorosis, particularly when the disease does not manifest itself by other signs.*

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* PETER: Gazette des Hôpitaux, 1867. This theory of Dr. Peter was formally stated in 1863, in the second edition of these Clinical Lectures.

† PARROT: Archives Générales de Médecine, t. ix, Juillet, 1867.

Dr. Potain† also is of opinion, that vascular blowing is not a sign, certain and pathognomonic, of anæmia; but he believes that the composition of the blood may have some influence on its production, and that, consequently, it is not without some semeiotic value. By experiments, equally ingenious and delicate, Dr. Potain has shown, that three vascular phenomena are observable in the region of the heart, viz., a movement, a thrill, and a sound. The movement consists of two slight upheavings followed by two more considerable sinkings, succeeded by a slow ascent of the region. These movements are due to the veins; and the application of the sphygmograph shows that the first upheaving immediately precedes the contraction of the ventricle, and corresponds to the systole of the auricle: the second upheaving is due to the systole of the ventricle. Thus, the two successive elevations of the vessels are due to successive contractions of the right auricle and right ventricle. The collapse of the veins corresponds with the diastoles: the first collapse is due to the diastole of the auricle, and the second, to the diastole of the ventricle. Though the thrilling is intermittent, it is produced during the vascular collapse, that is to say, during the cardiac diastole, a fact which harmonizes badly with the theory of Dr. Parrot, who attributes the thrilling, as well as the blowing, to insufficiency of the valves of the veins; for in that case, the thrilling ought to take place at the time of the upheaving of the vessel and to be coincident with the cardiac systole. Dr. Potain, in relation to the vascular murmurs, without rejecting Dr. Peter's spasm-theory, relates experiments which he performed, and from which it appears, that the greater the fluidity of the circulating liquid, the more intense is the blowing which it produces in the tubes wherein it circulates: thus, the blowing sounds are very loud when it is water or serum which circulates, and they cease altogether when these fluids are replaced by blood. But the greater the fluidity of the liquid, the more rapid is its circulation; so that the whole discussion resolves itself into a question of rapidity of circulation. As the blood is less dense, and more fluid, when there is a diminution in the number of red globules, it follows, that the rapidity of the passage of the liquid must be augmented, and the conditions be realized which produce vascular blowing. This is what occurs in chlorosis: it is, therefore, impossible to deny, that the quality of the blood possesses an influence upon the production of the blowing sounds.

The exposition which I have now made shows that we must not, as hitherto, exclusively attribute vascular blowing to the state of the blood. The causes of the phenomenon are much more complex. Fluid blood flows more quickly, and fluidity is a condition favorable to the production of vascular blowing; but fluid blood is less rich in globules, and being less rich in globules, it is less nutritive, and less an excitant of the nervous system. It is at this point that the too much neglected action of the walls of the vessel plays its part. It matters little whether the parietes of the vessel become paralytically relaxed, or spasmodically contracted; the important point is the occurrence of nervous disturbance—of a temporary and fugitive character, like everything which pertains to life—a disturbance which temporarily modifies the circulation in such a way as to produce a contracted column, and, as a consequence of that, a blowing vascular sound. We can thus understand how it is, that the blowing sound may appear and disappear in the course of the same exploration, as observed by Drs. Peter and Parrot. Moreover, the strength of the blowing sound is greater, if, in addition to the nervous state of the vessel, there exist greater fluidity of the blood.*

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* Sée: Du Sang et des Anémies, p. 202 *et passim*. Paris, 1866.

† Potain: Gazette des Hôpitaux.

In his conclusions, which are perhaps too absolute, Dr. Peter has been specially anxious to place in relief the action of the living solid, the wall of the vessel, the contractile wall, supplied by nerves from the great sympathetic, and to oppose its action, modifiable to an infinite degree within a space of time relatively very short, to the action of the blood, which cannot change sufficiently quick to explain by itself the rapidly changing blowing sounds in the vessels. I am ready to admit with Dr. Peter, that in some nervous persons vascular blowing exists irrespective of an alteration of the blood; but that which is only observed in them accidentally is the rule in the hydræmic and chlorotic.

Bear in mind that, in exophthalmic goitre, continuous reinforced vascular blowing is heard, particularly during the paroxysms of that disease. These paroxysms appear suddenly—most frequently, consequent upon strong mental emotion, and although the blood cannot, within a few minutes, undergo extensive changes in its constituent parts, it is during the paroxysms of Graves's disease, as well as in acute or chronic chlorosis, that vascular blowing is the result of a modification of the contractility of the vascular system.

*I think, however, that chlorotic blowing ought always to be carefully sought for; and the reason for my laying before you the minutiæ of the auscultation of the vessels of the neck is because they are of very great importance. While in the majority of cases of anæmia, ferruginous remedies are not generally trustworthy, and are sometimes dangerous, they possess an almost invariably useful and rapid influence upon chlorosis.

From what I have now said, gentlemen, you have no doubt perceived that I am disposed to class chlorosis with nervous diseases.

Let us, for a moment, leave out of consideration the constitution of the blood, and inquire by what other phenomena than paleness of the tissues the disease shows itself. These phenomena have an almost exclusive bearing upon the nervous system. The intelligence, the senses, the muscular movements of animal and organic life, are very much modified. Chlorotic girls generally experience perversions of the understanding, and I am acquainted with many such cases. They become irascible, and odd in their behavior; the mental disturbance sometimes goes so far as to be really insanity. If we examine very carefully into the state of the skin, we find that its sensibility is deficient in many places, while in others it is occasionally morbidly increased. I never examine a chlorotic woman in your presence without interrogating her as to neuralgic pains; and you may have perceived how unusual it is to find a chlorotic patient who does not suffer from them more or less. In these patients, facial is of all forms of neuralgia the most common; very often it alternates with intercostal neuralgia, and with neuralgia of the stomach, liver, intestines, and uterus.

Spasmodic affections of the locomotor system of animal life are very frequent; and you know how often hysterical convulsions are met with in chlorotic women. Palpitations of the heart, and spasms of the stomach, intestines, and uterus, occur in nearly all chlorotic subjects.

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* For information in relation to this interesting discussion, see the Bulletin de la Société Médicale des Hôpitaux. Also, the excellent article, Chlorose, by Dr. Lorain, in the Dictionnaire de Médecine et de Chirurgie Pratiques, t. vii. Paris, 1867.

The disturbance of the nervous functions produces great modifications of the secretions. The secretions of the stomach are altered in their chemical composition; hence we have pyrosis, pica, &c.: the secretions of the liver and kidneys are sometimes suppressed, and sometimes augmented, giving sufficient evidence of the existence of that nervous disturbance of which I have been speaking; and the great ovarian secretion, which constitutes one of the most important secretions of women, is very often suppressed along with menstruation, of which it is the consequence.

But, gentlemen, amenorrhœa is not always the accompaniment of chlorosis. Many years ago I published a work upon *menorrhagic chlorosis*, in which I showed that in virtue of exceptional tendencies, very difficult for me to appreciate, the menstrual flux becomes exceedingly profuse, and augments as the malady progresses. In such cases, ferruginous medication is quite as powerfully remedial as in normal chlorosis or amenorrhœa.

Gentlemen, we are in the habit of regarding chlorosis as a malady which is not serious; and in so doing, it appears to me, we take too little into account a general condition in which we see supervening great disturbance of the economy. I have long looked upon chlorosis as a serious affection. It has the peculiarity of leaving an almost indelible impression; so that when a young woman has been very chlorotic, she remembers it for nearly her whole life. If you interrogate women who have had several attacks of chlorosis, and have reached the catamenial decline, you will find that they present neuropathic phenomena which almost never leave them, however variable may be the forms which they assume. And this is observed when the blood-lesion has long previously been repaired: sometimes, even, plethora may be observed. This is a farther proof, that chlorosis must be regarded as a nervous disease caused by alteration of the blood, rather than as a cachexia causing nervous disorders.

I need not now recall to your recollection experiments recently instituted by the ablest physiologists demonstrating the influence which the different functional disturbances of the nervous system stamp upon the secretions, as well as upon the composition of the blood. It is easy to understand, that when the essential functions of an organ of hæmatosis, such as the lungs, liver, or spleen, are altered, the composition of the blood must undergo great modifications.

This influence of the nervous system sometimes makes itself felt with wonderful rapidity. Recall to your recollection a young woman, in bed 32, St. Bernard Ward, a patient who has already come twice into our wards to be treated for St. Vitus's dance. When in the enjoyment of very good health, she received a sudden fright during the night. Next morning her health was affected; and four days later she came into our wards with all the signs of confirmed chlorosis. In bed 3 *bis* of the same ward we had a girl of eighteen, who also, consequent upon, and some days after, a violent mental emotion, became chlorotic. These cases show you how little importance can be attached to the primary state of the blood; and how important it is to place anæmia only among the secondary causes of chlorosis.

Amenorrhœa, so common in chlorosis, sometimes announces the beginning of the disease. In other cases, a perfectly healthy young woman becomes suddenly chlorotic, when the catamenia, which had commenced, are suppressed, in consequence of a chill or some great mental shock. Certainly, in such a case, the dyscrasia of the blood cannot be considered as the result of a hemorrhage; and as it also occurs in certain cases of anæmia, it can be imputed only to nervous perturbation. On the other hand, we see young girls remain chlorotic for a long period, although the most rational treatment has been employed, and cease to be chlorotic on the appearance of the menstrual hemorrhage, that is, when the anæmia ought to be increased by the loss of blood. The explanation of the case is this: on the cessation of the general nervous disturbance, the normal secretions are re-established, because the cause of the chlorosis itself has ceased.

From the remarks which I have made, you may pretty well conclude that chlorosis is not always so easily cured a disease as has been supposed. In the clinical wards you have seen some young women cured very quickly, whilst in others we have had to wait a long time ere we saw the results of the treatment. In fact, to conduct this malady to a happy issue, a combination of favorable conditions is required, not always easy to attain or meet with in hospitals, nor even in private practice.

I have told you, gentlemen, the part which the nervous system performs in chlorosis: you can understand, therefore, how important it is to withdraw the patient as much as possible from all deleterious mental influences. Though I have endeavored to prove that anæmia is an effect and not an essential element of chlorosis, I am not the less of opinion that the dyscrasia of the blood, under whatever circumstances produced, has a tendency to induce chlorosis, and that it would be impossible, whatever medication were employed, to cure a patient unfavorably placed in respect of regimen and residence. It is vain to administer iron and bitters, if a subject exposed to paludal miasmata lose daily, under the influence of that morbific cause, all that is gained by the ferruginous treatment. It would be the same if (as often happens) the patients refuse to take exercise in the open air, and obstinately remain shut up in obscure apartments where they become etiolated. It would also be the same were profuse menstruation, or frequent attacks of epistaxis, to destroy as much as the remedies restore. Finally, if the supply of food be insufficient, the alteration of the blood will increase; and thus, as you perceive, we shall always be turning round in the same vicious circle.

But it is easy to foresee the difficulties presented by the *circumstances apparently accessory*.

Though we can remove patients from countries where intermittent fever prevails, it is not so easy to contend against paludal infection when once established in the system. It then becomes necessary to treat the intermittent fever with the same energy and perseverance demanded in paludal maladies which have long dominated in the system. And it is only then, when we have mastered the intercurrent disease, that we can beneficially institute our treatment for the chlorosis.

As to the etiolation, the result of deprivation of light, to which, from perverted inclination, the young patients often condemn themselves, it is necessary to arrest it with the least possible delay, and to employ severe measures to accomplish this object should the weakness of relations render them necessary by placing obstacles in our way.

Uterine hemorrhages and epistaxis, as I have told you, are sometimes the effects, rather than the causes, of chlorosis. Although, in general, ferruginous medication may of itself be sufficient treatment, it too often happens that we gain nothing by it, in consequence of the constantly renewed hemorrhages not being compensated for by the reparation derived from the iron. These are the cases in which the powder of cinchona, extolled by Bretonneau, renders services not to be expected from any other remedy; and although I do not know to what principles cinchona owes its potent properties, I can testify not the less confidently, that in menorrhagia and obstinate epistaxis, powder of cinchona, taken internally in doses of from two to four grammes, daily, or two or three times a week, quickly moderates and rapidly cures the hemorrhage.

This therapeutic agent is much superior to rhatany, tannin, and the mineral acids, as I have had frequent opportunities of seeing in my clinical wards.

But if, as sometimes happens, medicines do no good, we must not hesitate to employ surgical measures, however disagreeable that course may be. Plugging the nasal fossæ by the different means with which you are all acquainted will arrest the bleeding, should you have failed to attain that object by the use of astringent injections; but you can understand that injections are useless in menorrhagia, as the medicinal agent does not come in contact with, and indeed cannot reach the mucous surface of the uterus. It is then that plugging becomes indispensable: and however disagreeable it may be to have recourse to such a proceeding in a young girl, and however painful it may be, we must not hesitate to employ it, because it is the only treatment which has any chance of being successful.

I had to treat a young lady of nineteen who was chlorotic to an extreme degree, and likewise subject to menorrhagic attacks which were at first moderate, but ultimately became intractable. The loss of blood, sometimes so great as literally to traverse the mattress and fall on the floor, obliged me to use plugging: it succeeded wonderfully; and I was able by ferruginous medicines to restore this patient to perfect health. She quitted Paris; and had subsequently a recurrence of the symptoms. Upon this occasion, her family shrunk from consenting to a means of treatment to which the young lady had an intense repugnance, and to the carrying out of which a difficulty was perhaps presented by the youth of the attending physician. Death from hemorrhage supervened, as internal remedies failed to arrest the hemorrhage.

My friend Dr. Campbell has often mentioned to me the wonderful successes which he has obtained in formidable hemorrhage supervening after delivery, by administering enormous quantities of spirituous liquors according to the practice of many English practitioners. He has ordered the administration, in tablespoonfuls, of as much as a litre of brandy or rum in the twenty-four hours. He has given, at the same time, sherry, Madeira, and Malaga wines. Strange to say, the women bore the large doses of alcohol without experiencing the slightest inconvenience, so long as they were under the influence of these hemorrhages, whilst as soon as they recovered they were unable to take the smallest quantity without becoming drunk. Professor Dubois and I have seen similar cases.

May not this treatment, however extraordinary it may seem, be as much indicated in the menorrhagia of which I have just now been speaking, as in profuse hemorrhages consequent on delivery?

A few minutes ago, I spoke to you on the subject of regimen: I remarked, how important it was that the patient should have suitable alimentation: but here, an insuperable difficulty often arises. The disease not only engenders dyspepsia, but also a capricious appetite, a disgust for the most substantial fare, and an insensate craving for things generally regarded as very bad. There are some chlorotic girls who would rather die from hunger than eat the usual food of other people.

In such cases, gentlemen, we must not hesitate to submit to one of those therapeutic capitulations so often demanded in the exercise of our art. I generally pay very little attention to the nature of the food, provided it be digested. I sanction without scruple the use of articles reputed to be very indigestible, such as radishes, salad, hardly ripe fruit, strong-tasting cheese, very sour wine, vegetables, highly spiced pork-meats, acid beverages, and spirituous liquors. But if I am thus indulgent, I expect a concession in return, which is, that this strange aliment be varied. In relation to this point, gentlemen, allow me to make a short digression, not altogether irrelevant.

Man and the lower animals are so constituted that they allow themselves to follow the same routine in diet as in other matters; yet there are many things in which a change, even when it is a change for something worse, is accepted by the economy, not only without injury, but sometimes with benefit. The great effect produced by a mere change of residence is astonishing: the person who daily takes suitable exercise, but whose regimen is sometimes not very good, and who breathes a less pure air and takes the same exercise, experiences a sort of transformation and feeling of betterness which are simply the results of a new excitement in the economy produced by impressions to which it has not been accustomed.

The same observation is true in respect of regimen. We know that the stomach is easily tired by a constant repetition of the same articles of food, and that its functions are favorably influenced by change of regimen. On the other hand, though experiment on domestic animals which it is wished to fatten proves that the same amount of equivalents of nutriment produces proportionately better results the more the elements are diversified, yet experiments instituted in the human subject demonstrate that, though in our ordinary meals we become satiated by a determinate quantity of food, which cannot be exceeded without producing some disorder in the digestive functions, we may, at a banquet of numerous and of varied dishes, take with impunity a double quantity of aliment.

Gentlemen, pardon this digression which I believed necessary to enable you to understand fully, that though we may condescend to allow chlorotic women to eat articles reputed indigestible, all objection ceases provided we arrange so that in every meal there is a multiplicity and variety of dishes. It is in this way that we can arouse the digestive powers, and impart to the blood, with even an insufficient alimentation, some of the constituents which are wanting, and prepare the way for therapeutic agents of which I shall forthwith have to speak to you.

The concessions of which I have been speaking must not be regarded as mere acts of complaisance. When we see chlorotic girls easily digesting aliments which they would have rejected some years previously, the question arises, whether, under the influence of the great disturbance of the nervous system which accompanies chlorosis, new aptitudes do not replace those which are lost, in such manner, that the digestive organs find themselves accidentally in functional harmony with the aliments better suited to the organs of other animals?

Marriage is one of the hygienical measures recommended to chlorotic girls, regarding which physicians are often asked to give an opinion. The strange idea, after germinating in the heads of some physicians, has become popularized, that the erotic instincts are more developed in chlorotic than in other women.

I willingly admit that puberty indicates a woman's fitness to conceive; but I deny that this fitness arouses in her instincts similar to those which it arouses in a man. In our order of society, young girls are as remarkable for chastity in thoughts as in actions; and when chlorosis declares itself in married women whose confidences we obtain, we often learn that their sexual appetites have diminished in proportion as the disease has progressed. That, certainly, does not absolutely prove that marriage would be useless, but it at least seems to indicate that the acts which are the usual consequences of matrimony are little necessary, and may be instinctively repugnant to chlorotic women.

I grant that it is not convenient to cause a young woman to defer too long the duties of maternity, for which she was created; but that is a very different thing from adopting the common notion, that marriage is indicated as a means of cure in a multitude of maladies. Suppose that a girl who from childhood has had horrible scrofulous or dartrous sores, is seized with epilepsy, hysteria, or intermittent mania—is not that the doleful dowry which she would bring to a young husband, but which would be carefully excluded from the stipulations of the marriage contract?

I now come to speak of the treatment by the administration of pharmaceutical substances.

Iron occupies nearly as important a place in the treatment of chlorosis as cinchona in the treatment of intermittent fever; but there is great need of young physicians forming correct views as to the doses and modes of administering ferruginous medicines. We know very well that chlorosis is a disease which has a great tendency to recur: and, as I have told you, a woman who has been seriously chlorotic for a long period retains traces of the formidable neurosis as long as she lives. It is, therefore, an essentially chronic disease; and *chronic diseases must have chronic therapeutics.* That is one of the most elementary precepts in medicine; so that the administration of iron ought to be persevered in for a long period, and frequently resumed. The length of the intervals during which the use of the iron is discontinued will be the more prolonged the more perfect the state of health. After achieving a victory, we must not go to sleep. When, after six weeks or two months of treatment, the color and the menstrual functions are restored, we must proceed further than the apparent cure, and recommence after intervals of two or three months, and continue to do so during three consecutive years, should the chlorosis have been of old standing, and have left a deep impression on the economy.

It is rather difficult to state with precision the doses of iron which are necessary for the cure of the malady; and in respect of dose, perhaps greater diversities occur than are observed in other diseases. We all know that in treating tertiary syphilitic affections, we see one individual get rid of violent osteocopic pains in a few days by taking very small doses of the iodide of potassium, while another, who seemed to be precisely similarly affected, will not obtain relief till he has taken doses of the iodide ten times as strong, and during a much longer period than in the other case. The remarks now made are applicable to a multitude of medicines besides iron. Iron is, however, one of those medicines by which similar results are obtained by the greatest diversity of dose. Some chalybeate waters which, like those of Pougues, Spa, and Schwalbach, scarcely contain a few centigrammes of the salts of iron to the litre, sometimes cure chlorosis more quickly than steel filings, black deutoxide of iron [*l'ethiops martial*], or subcarbonate of iron [*les safrans de Mars*], which are given in doses of some grammes daily. Let me state, however, that, in general, ferruginous preparations ought to be administered at each meal in doses of from fifty centigrammes to one gramme; and that the only limit to the dose is the tolerance of the stomach.

Pharmacy has in preparations of iron a luxurious abundance, unfortunately, to some extent, created by commercial speculation. Every inventor of a new salt finds good reasons for celebrating its superiority. But, after long trial of the old and new preparations, both in my private practice and under your observation in the clinical wards, I feel convinced that no preparations are so well suited for administration in the form of pill as the filings and the subcarbonate, which may be made into a mass with the extracts of cinchona, chiccory, wormwood, or sometimes with rhubarb, provided certain special indications are attended to. Extract of wormwood is a useful excipient in the case of women who have amenorrhœa combined with dyspepsia; and the majority of cases are of this nature. When there is obstinate constipation, the addition of a small quantity of the extract of rhubarb, the exact dose of which cannot be specified, may be of great service. With women in whom there are none of the accidents inherent to chlorosis except want of appetite, the soft extract of cinchona generally agrees well.

When I do not give ferruginous preparations in the form of powder or pill, the liquid form which I prefer is the syrup of the ammonio-citrate of iron in the form of fifteen grammes of the salt to five hundred of syrup. From one to four teaspoonfuls may be taken at each meal.

Gentlemen, you have seen me several times prescribe ferruginous preparations to chlorotic patients who were unable to bear them. In general, iron is borne well, at the first start, in chlorosis; and as I remarked at the beginning of this lecture, it is badly tolerated in many cases of anæmia: and we must be on our guard when the economy does not support it. But although the diagnosis has been carefully established, and the indication is exact and precise, iron may be badly supported; and then it is necessary to use artifice to make a useful remedy be tolerated. Iron, as I have already said, like most medicines, is most suitably administered during a meal. Even, however, when this precaution is taken, the stomach seems sometimes to refuse it, gastralgia increases, dyspepsia becomes more serious: diarrhœa occurs sometimes; and, still more frequently, there is invincible constipation. Then it is, when diarrhœa and gastralgia dominate, that minute doses of extract of opium may be very advantageously combined with the iron: then, too, belladonna in very small doses cures the gastralgia, and, with still more certainty, removes the constipation.

The administration of iron ought from time to time to be interrupted, and cinchona wine given in place of it. Sea-water baths, hydrotherapeia, and sulphurous baths are very useful adjuvants.

It often happens, gentlemen, that recovery from chlorosis takes place without any medicinal intervention: and sometimes you have seen me seek to cure a case of this malady by the peroxide of manganese, when I have failed with ferruginous preparations. Under the influence of these different medications, restoration of the normal constitution of the blood takes place, and disappearance of all the symptoms peculiar to pale-faced chlorotic patients.

The remarks which I have just made must suggest to your minds some doubt as to the part which iron plays in the treatment of chlorosis. You ask yourselves whether it really is from the ferruginous remedy that the blood derives the very small quantity of iron which it requires. Animals in a state of health reconstitute all the elements of the blood from their food; and when we consider how small is the quantity of iron contained in the entire mass of blood in the body, we can easily see that there will always be enough of it in the ingesta.

When a fecundated hen's egg receives the influence of heat in incubation, each of the most intimate elements which enter into the composition of the white and the yolk is distributed to the different parts of the animal, in virtue of vital affinities inherent in the organic molecules. The calcareous salts form the bones and the feathers. The albumen, modified in its composition, constitutes the muscles and the blood: the small quantity of iron found in the newly laid egg concretes to form hepatic globules simply in virtue of nutritive activity: that this take place, it is sufficient that the egg be endowed with life, be sound, and be placed in certain conditions.

We can understand, that in the same way, the living organism, in a healthy woman, can derive from the different alimentary substances, the majority of which contain iron, the elements requisite for the constitution of the blood and muscles; and, as I have just been saying, the quantity of iron contained in the economy is so small, that the usual aliment is amply sufficient for the necessary reparation. It is, therefore, not indispensable that the ferruginous therapeutic agent should furnish to the blood its deficit of iron. It is enough that the ferruginous remedy put the organs into that condition of health which imparts the power necessary for the assimilation of the iron in the food, a power similar to that by the exercise of which the organic elements of the chicken assimilate the iron contained in the egg.

You perceive, gentlemen, the point to which we are led by the theory of those who desire to find the principal constituents of the blood in the medicine. In syphilitic anæmia, and in paludal anæmia, we must accord respectively to mercury and cinchona a part similar to that which iron plays in chlorotic anæmia. Now, I have shown you, and, moreover, you know very well, how powerfully mercury and cinchona act in reconstituting the blood, and, consequently, in restoring to it the iron in which it is deficient. Therefore we may, indeed we ought, to consider iron as the specific for chlorosis, just as mercury and cinchona are respectively the specifics for syphilis and marsh fever; and these three medicines administered in three different kinds of anæmia will restore the wanting iron to the blood and muscles, not because they furnish iron to the economy, but because, by restoring the organs to a sound state, they enable them to accomplish their normal functions, in virtue of which they assimilate the iron contained in the food as in the plenitude of health.

Are you not struck by another curious fact? Consequent upon some great mental emotion, or from some other cause, the menses, when they had just commenced, are suddenly suppressed, chlorosis often appearing within a few days. For a long time, the malady continues; then, without our being able to discover the causes, there is a profuse menstrual flux, and, after some days, the health seems to be re-established. Here, the loss of blood, and of its contained iron, is the condition under which the return to health has taken place: in the former case, the loss of blood has been too small, and the iron has disappeared from the blood-globules. You will understand, gentlemen, that the explanation of these facts is not easy for those who would find in the ferruginous remedy prepared by the apothecary the element necessary for the reparation of the blood.

Perhaps we may render an account of these facts by supposing, that in cases of suppressed menses, there is suppression of an eliminatory function, of an emunctory of morbid principles: whilst with the return of the catamenia, there would be elimination of these same morbid principles. But this double hypothesis will not explain the manner in which iron acts on the blood. And if it be admitted that chlorosis is only a nervous state which produces an immediate effect upon the composition of the blood, ought we not perhaps to see in the preparations of iron only agents which present a modifying influence upon the nervous system, which act on chlorotic anæmia in the same way that mercury, the iodide of potassium, and cinchona act upon syphilitic and paludal anæmia?

LECTURE LXXXVIII.

CIRRHOSIS.

Cirrhosis is not a special product: still less is it Atrophy of the Red and Hypertrophy of the Yellow Substance of the Liver.—It is Chronic and generally Consecutive Phlegmasia.—Cirrhosis in Affections of the Heart, in Alcoholism, Syphilis, and Marsh Fevers.—Slow, progressive Atrophy of all the Tissues of the Liver from Strangulation.—Serious Disturbance of the Hepatic Hœmatosis, and its Response in the Organism.—Cholesteremia.—Cirrhosis, which is a Lesion and not a Malady, adds its evil consequences to the evils belonging to the primitive affection in which it originates.

GENTLEMEN: Here are the morbid anatomical preparations taken from a woman, aged 29, who died in the clinical wards from a disease of the heart. Three years previously, she had had a very slight attack of acute articular rheumatism, consisting in pains in the right shoulder, which were of recent date, and unaccompanied by fever. Six months later, she had begun to suffer from palpitations. At the end of a year, her legs became swollen; and at a later date, her abdomen was tumefied.

When admitted to the hospital, this unfortunate woman had most distressing orthopnœa: fine subcrepitant râles were heard in nearly every part of the lungs. No purring thrill was heard over the apex of the heart: there existed, however, a blowing sound, which was not rough, at the apex: this blowing sound, though soft, was intense: its maximum of intensity was at the middle rather than at the apex. There was no venous pulse, and, consequently, no tricuspid insufficiency. I diagnosed contraction of the left auriculo-ventricular orifice and insufficiency of the mitral valve. There was a considerable amount of ascites. The volume of the liver was nearly normal. If, as I suspected, cirrhosis existed, it was but little advanced. There was considerable anasarca: the œdema of the inferior extremities and of the abdominal parietes was enormous. As yet, there was no well-marked dilatation of the subcutaneous veins of the abdomen.

Two days after her admission to the hospital, this woman sank under the rapid progress of asphyxia, against which all our treatment was fruitless.

At the autopsy, we found a small heart, without hypertrophy of the left ventricle, and without contraction or insufficiency of the auriculo-ventricular orifice. The ventricular side of the orifice presented a *cul-de-poule* appearance, and the auricular side had the form of a linear slit. The valves were five times their natural thickness: they had the color and consistence of fibro-cartilage. The aortic orifice was healthy. There was no coagulum in the pulmonary artery.

The lungs, as you see, are remarkably deformed at their summits. In some places, they are shrunk up: one presents two and the other three curious projections, one being inserted within the other like a finger in a glove. One might say, that there is hernia of the lung: in point of fact, there exists a circular depression of the lung caused by retraction of the pleura, which is lined at certain points by false membrane which has become fibrous, and the lung projects at these places where the retraction did not exist. The greater part of the lung is in a state of chronic congestion. The parenchyma is partly carnified, its anatomical condition resembling cirrhosis of the liver.

The liver is normal in volume; and is rather large than small in size: the right lobe is much congested, and very red, having on the red ground, numerous yellowish points as large as the head of a nail, which imparts a granitic appearance, without there being any granulations projecting from the surface. On the contrary, the whole free margin of the organ, and nearly the whole of the small lobe, present a tawny color and granitic aspect, with projecting granulations, which are the more obviously projecting the nearer they are to the sharp margin. On the inferior surface, you can see depressed, whitish lines, the depression being the result of the retraction of the hypertrophied fibrous tissue—the hypertrophy of the fibrous tissue revealing itself by these furrows.

You see here, gentlemen, a case of cirrhosis which is not much advanced. The naturally bulky part of the liver—that is to say, the right lobe—is still bulky. It is much congested, and of a bright red color: the incipient alteration only reveals itself by the yellow color of the acini. Indeed, in situations in which the organ presents a less degree of thickness, and, where, consequently, there is less predominance of fibrous over parenchymatous tissue, the granular condition is developed. Without it being necessary to go at length into the subject, you can understand the mechanism by which is produced a structural alteration of this nature: as the atrophy of the organ is caused by the retraction of the interstitial fibrous tissue, it is evidently in the situations where this tissue most abounds that atrophy ought to commence, and there also it ought to attain its maximum in the cirrhosed liver. Hence, in all descriptions and pictorial representations of cirrhosis, the sharp edge of the organ, and of its small lobe, are described and figured as being greatly shrunk up. Frerichs of Berlin, to whom we are indebted for a remarkable treatise on diseases of the liver, has mentioned the fact without, however, laying much stress upon it; and in the work to which I refer, you will see a case "in which the liver weighed 2200 grammes: the right lobe was much swollen, covered with contracted cicatrices, circumscribing tuberosities the size of a hazel-nut, or even as large as a hen's egg, while the left lobe was transformed into a sort of short, tough, granulated appendix."* Thus, retraction of the interstitial fibrous tissue, strangulation of the parenchyma—and that in the situations where, normally, it is less abundant—were the facts we derived from this autopsy.

But was this really a case of cirrhosis? Is cirrhosis a consequence of cardiac disease? Dr. Frerichs, the justly celebrated German author whose work I have just been quoting, holds that it is not. This is a doctrinal point which we must discuss. Before proceeding to do so, allow me, however, to give you a short history of cirrhosis.

It was Laennec, as you know, who created the name, *cirrhosis*, deriving it from *κέρρος*, *russet-colored*, in allusion to the color of the diseased organ. Dominated by his views in relation to the nature of cancer and tubercle, which he wished to regard as similar in their nature to parasitic organisms, and which he looked on as new products, living their own life at the expense as well as to the great injury of the organism whence they derive their support, and the destruction of which they ultimately induce—dominated, I say, by these ideas, Laennec regarded cirrhosis as a special product developed in the liver, there manifesting a particular manner of evolution, the last stage of which is softening.

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* FRERICHS: Traité des Maladies du Foie; traduit de l'Allemand par les Docteurs Louis Duménil et I. Pellagot, 2me édition, p. 336. Paris, 1866.

But, long before Laennec's time, cirrhosis had been observed and described: only, it had often been confounded with tubercle, from the mammillated aspect which it gives to the liver. Morgagni, who employs the expression "tubercles," refers more to the configuration than to the essential nature of the morbid product. He notices the coincidence of ascites with this alteration of the liver. Baillie, in his first article on the disease, gave a summary description of cirrhosis under the designation of "common tubercle of the liver." His description was, to a certain extent, in advance of Morgagni; for he mentions the greater frequency of the malady in men, "probably," he remarks, "because they drink more than women:" he called attention to the correlation of this affection with drunkenness, and to its coexistence with ascites: he also pointed out the diminution in the volume of the bloodvessels, and, after giving a sufficiently good description of the granulations, he insists upon the diminished volume and increased density of the organ.

Baillie, in a second article—entitled "Liver very hard in its substance"—considers this peculiar condition as probably forming the first degree of "common tubercle of the liver." He hazards a pathogenic theory, to the effect, that a new material ("additional matter") is deposited in the parenchyma, and there produces the nodosities. He denies, moreover, that the morbid change is of the nature of scrofula (tubercle) or cancer. Here, you see, is progress.

I have stated the views of Laennec, from the respect I entertain for so great a man, and because it is from him that we date the study of cirrhosis. I pass, without comment, the opinion that there is "atrophy of the red and hypertrophy of the yellow substance of the liver," or rather "atrophy of only one portion of the gland, and hypertrophy of the rest of the organ."

In 1840, Becquerel, though erroneously holding that an albumino-fibrous material was infiltrated into the yellow substance of the liver, and so produced hypertrophy of that substance, this hypertrophy being derived in the first instance from compression, and afterwards from atrophy of the red substance, advanced many very correct ideas, one being, that by far the most common cause of cirrhosis of the liver is acute and frequently recurring congestion of that organ. The liver, under the influence of that congestion, receives an abnormal quantity of blood, which is more than sufficient for the biliary secretion. The albumen and fibrin of the blood are deposited, and gradually organized, in the weft of the yellow substance of the liver.*

Excepting that it involves a belief in the existence of two substances in the liver, and a belief in the infiltration of the yellow substance, Becquerel's theory is a very near approach to the views now current in medical science.

Since the researches of Rokitansky, Gubler, and particularly of Frerichs, cirrhosis is regarded as the result of the exudation of an organizable blastema into the layers of the interlobular connective tissue. This blastema or plastic substance becomes organized into connective tissue: this newly formed connective tissue becomes fibrous, contracts, and, by its contraction, strangles the secretory parenchyma and the capillary portal vessels. Three consequences follow from this contraction, viz., atrophy of the gland, diminution of the biliary secretion, and ascites. There are many other consequences, but these will come into view as we proceed: at present, I only wish to discuss, so far as necessary, the cause of that exudation. Is inflammation the cause? Is some other morbid process the cause of the exudation?

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* BECQUEREL: Archives Générales de Médecine.

I have no hesitation in saying, with Frerichs, that cirrhosis originates in chronic inflammation. But I hold, in opposition to the opinion of Frerichs, and in accordance with facts, that this chronic inflammation is very often consecutive to a cardiac affection.

Here, there is a very strong analogy. If the affections of the bronchial tubes and lungs which follow diseases of the heart are not inflammatory, what are they? What is their genesis? In some particular part of that wonderful hydraulic apparatus which we call the heart, a dam exists, and then the blood gradually accumulates above the dam in the afferent vessels. Let us suppose, for example, that there is contraction with insufficiency of the left auriculo-ventricular opening—the auricle empties itself with difficulty, and in an incomplete manner, into the subjacent ventricle, a partial reflux into the pulmonary veins taking place: these veins, again, discharge themselves imperfectly: the result is stasis of the blood in the capillaries originating in these veins, and, as a consequence of this mechanical cause, there is induced hyperæmia of the bronchial tubes, and even of the pulmonary parenchyma. But this unintentional [*non-intentionelle*] hyperæmia—pardon me the expression—this hyperæmia which was not originally destined to form the first in a series of morbid changes which has produced inflammation as its ultimate state, is the origin of that inflammation. In the first instance, the surcharged capillaries discharge their excess upon the bronchial mucous membrane: the result is, a flux, or bronchorrhœa, and then, as Laennec says, this phlegmorrhagia becomes chronic bronchitis, so obstinate an affection in persons suffering from disease of the heart. On the other hand, the capillaries allow exudation of the serosity incumbering the posterior lower parts of the lungs: hence we have pulmonary œdema. The blood gradually thickens in consequence of this very exudation, and, whether from the fluid subsequently exuded being increasingly rich in fibrin, or, because in the fluid originally poured out the fibrin predominates in consequence of the evaporation which takes place from the free surface of the bronchial tubes, or finally, whether, because the distended pulmonary tissue becomes more irritable, inflammation always supervenes, and thus we have broncho-pneumonia. The damming up, purely mechanical, has caused, in succession, congestion, flux, dropsy, and, finally, inflammation.

Similar phenomena are observed in the kidneys: mechanical congestion is followed by albuminuria: to that succeed infiltration of the secretory canaliculi, desquamation of the tubuli, and finally, that bastard inflammation to which Bright has given his name.

But why should we seek for examples in deepseated organs? Do we not see with our eyes similar phenomena in dropsical persons, the skin of whose legs is distended by œdema? First of all, there is slight erythema; then, desquamation of the epidermis; and then, the superficial layer of the dermis gives way: vesicles and pustules appear; and sometimes, even more or less of the skin becomes mortified. These are unquestionably phenomena of inflammation, the starting-point of which was a purely mechanical congestion of the cutaneous capillaries of the lower extremities.

Who then can have any difficulty in believing, that what takes place in the bronchial tubes, the lungs, the kidneys, and the skin, may and must occur in the liver?

The principal objection of Frerichs is, that the embarrassment of the cardiac circulation produces dilatation of the vena cava inferior, which gradually extends to the hepatic veins, to the very origin of their roots, the result being atrophy of the cellules. The same author also believes, that the atrophy is the result of compression: the parts supplied by the hepatic veins become flattened, while those supplied by the vena porta protrude in the form of granulations. In other words, the atrophy is limited to those parts of the liver which are next to the dilated capillary vessels. At the same time, the walls of the vessels exposed to abnormal pressure become flattened. In the neighborhood, here and there, upon the envelope of the gland, there is a production of connective tissue which contributes to giving the liver greater consistence.*

Here, you see, is constantly presented the idea of mechanical pressure, never the more dynamic and truer idea of consecutive congestion, exudation, and phlogosis. I cannot too emphatically protest against this view; and my object in quoting the exact text of Frerichs is to show you the point to which one may go, in giving a physical explanation of morbid phenomena. This author has, however, described cirrhosis in a chapter entitled: "Chronic Inflammation of the Liver."

Cirrhosis, then, is rather a chronic inflammation of the liver than anything else; and it frequently occurs as a secondary affection in cases of disease of the heart, as the result of mechanical causes which I have endeavored to explain.

Alcoholism is a not less powerful cause of cirrhosis. Being immediately absorbed by the veins of the stomach, without undergoing decomposition, as Mitscherlich has shown, the alcohol enters direct into the portal system of the liver. In this way, it traverses the entire chylopoietic apparatus before it reaches the lungs; and it even copiously bathes the tissue of the lobules. You can understand without my making any detailed statement, what a disastrous influence this powerful irritant must exert upon the secretory parenchyma. The less the alcohol is diluted, the greater is its irritant effect. Upon this point, authors are agreed: cirrhosed liver is called "gin-drinkers' liver" by the English. Frerichs states that on the northern coasts of Germany and England, where the lower classes drink very strong spirituous liquors in excess, cirrhosis is more common than in the inland districts of these countries, where the use of wine and beer predominates. In support of this statement, the same author says that he has observed cirrhosis more frequently at Kiel on the shores of the Baltic, than at Göttingen and Breslau.

The action of alcohol on the liver may be compared to that of certain irritant substances carried along in the stream of the blood of the vena porta and poured out into the intestines. Budd is disposed to attribute the frequency of cirrhosis in India to the immoderate use of curry and other powerful condiments. With Frerichs, I have very little doubt that the abuse of spices, very strong coffee, and some other similar substances, produce hyperæmia of the liver, at first transitory, but which by being constantly reproduced, may finally determine the production of cirrhosis.

The action of syphilis occupies a place upon a much less prominent platform. Gentlemen, there is syphilitic inflammation of the liver, just as there is syphilitic inflammation of the testicle. The inflammation is superficial, or it is deepseated. Here, let me bring to your recollection what takes place in syphilitic orchitis.

The affection is sometimes periorchitis, and at other times parenchymatous orchitis. But in every case, it is of the essence of the specific inflammation to attack the connective and fibrous tissue of the organ. Thus in

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* FRERICHS: Traité Pratique des Maladies du Foie. 2d édition, p. 300, Paris, 1866.

periorchitis, there is inflammation of the tunica albuginea, which becomes thickened, and contracts adhesions to the tunica vaginalis. In parenchymatous orchitis, the inflammation is propagated from the periphery towards the centre: the free part of the testicle is first attacked, and the tunica albuginea is thickened: the inflammation then extends along the vesiculæ seminales and vascular cones to the rete testis, which, as also the epididymis, it seldom attacks. The morbid action consists in increased production or proliferation of the connective tissue into the interstitial tissue situated between the canaliculi. In the early stage of this morbid action, we see this tissue transformed into a soft reddish mass rich in nuclei, which afterwards becomes thickened, assumes a tendinous appearance, separates the canaliculi from one another, and causes them to atrophy, after having determined fatty degeneration of their epithelium. Finally, the vascular cone thus affected gradually hardens, becomes tendinous, and retracts, a depression resembling a cicatrix being formed in that situation. When the depression is so very extensive as to be almost general, the entire testicle diminishes in volume, and its surface is segmented by depressions resembling cicatrices. This alteration of the testicle is a true cirrhosis; and what takes place frequently and easily in the testicle is accomplished by a similar process in the liver of certain syphilitic persons.

Just as we have periorchitis, so have we perihepatitis, an affection of the capsule of Glisson, determining to the surface of the organ a sort of miliary eruption, resembling very small warts; and in the same situation, there is a hard, almost callous thickening of the fibrous capsule, and also adhesions, of very characteristic thickness and solidity, formed with the neighboring organs. Likewise, as we see in periorchitis, the morbid action propagated to the interior of the organ along the filiform prolongations of the tunica albuginea, so may we also observe the affection advance by slow degrees, following the course of the capsule of Glisson. The result is the formation on the surface of the liver of whitish, radiated, wrinkled depressions; and in the same situation, there exists glandular atrophy from real strangulation. You can understand, gentlemen, that when these depressions (which in respect of tissue are of the nature of cicatrices), penetrate deeply into the organ, and unite with one another, they ultimately strangle the organ by their contraction. This, then, is a special form of cirrhosis. You now see, how syphilis produces this affection of the liver.

Intermittent fever has been mentioned as one of the causes of cirrhosis; and Frerichs has observed three cases in which cirrhosis occurred as the sequel of obstinate intermittent fever. Chronic hyperæmia of the organ appears to me to explain, to a certain extent, the development of granular induration.

Finally, it is necessary to distinctly understand, that there are a certain number of cases in which the causes of cirrhosis are entirely unknown.

Gentlemen, I have endeavored, in this pathogenic grouping which I have now given, to avoid etiological trivialities, by making you be present, as it were, at the genesis of the disease. I have not thought it sufficient to tell you that cirrhosis is observed in cardiac affections, in alcoholism, and in syphilis; but have likewise felt it necessary to inquire with you into the manner in which it is produced.

To sum up: most of the immediate causes of cirrhosis may be grouped under three heads; viz., 1st, chronic hyperæmia of a passive character depending on heart disease or paludal cachexia; 2d, chronic hyperæmia of an active character depending on alcoholic toxæmia; and 3d, chronic active hyperæmia depending on syphilis. In these three groups of cases, the cirrhosis develops itself from the portal system to the parenchyma, proceeding from the centre towards the periphery, and the affection involves the substance of the liver in deepseated situations: in the latter case, the disease advances from the fibrous tissue to the glandular structure, from the periphery towards the centre. The cirrhosis is less complete, less generalized throughout the totality of the organ. Virchow, however, correctly remarked, that "the cicatricial indurations of the liver do not necessarily follow the divisions of the vena porta."

Each of these causes of cirrhosis has a corresponding form of special alteration of the liver.

In cirrhosis originating in disease of the heart, the liver only presents innumerable small disseminated granulations, on its surface, which is usually, to a slight extent, contracted. There exists a kind of compensation between the contractile tendency of cirrhosis and the expansion-force of the congestion arising from vascular stasis. Consequently, the liver is sometimes increased beyond its normal volume from congestion predominating over contraction; sometimes, its volume remains normal from the existence of an equilibrium of the two forces: and finally, there is sometimes a diminution in its bulk: in every case, however, it is minutely granular.

In alcoholism, the granulations are numerous as well as small: there is great diminution in the volume of the liver, the contractility of the new fibrous tissue not being counterbalanced, as in cirrhosis from cardiac disease, by an antagonizing force. In the cirrhosis from alcoholism, then, the liver is very small and finely granular on its surface.

In visceral syphilis, the granulations and the lobules are more voluminous, and less numerous: they are disseminated, sometimes in the form of patches, at other times, they coexist with encysted gummata; and thus we explain Laennec's cirrhosis in hard patches and cirrhosis in cysts [*en plaques et en kystes*]. The contraction is always less than in the cirrhosis consequent upon alcoholism. In syphilitic cirrhosis, the liver is moderately diminished in volume, and is lobular rather than granular.

I trust that I have shown cirrhosis to be a disease which is not always the same, which is not always identical—which is, on the contrary, nearly always secondary, and dependent upon a protopathic affection—that the lesion differs according as the disease is the result of cardiac affection, alcoholism, or syphilis—and that explains the opposing statements of authors, some of whom have described the lesion as atrophy, and others as hypertrophy. The remarks which I have made upon the mechanism of the different forms of cirrhosis must have led you to perceive why it is, that in heart disease, the cirrhosed liver is more frequently hypertrophied, or at least not reduced in volume, and why it is that the cirrhosed liver of alcoholism is atrophied. You must have also seen why the form of the granulations varies according as the cirrhosis is cardiac, alcoholic, or syphilitic.

I shall not enter into long details regarding the hepatic alterations appreciable to the naked eye, as your classical treatises have made you acquainted with them: it will be sufficient to remind you that the serous envelope is nearly always thickened, and of a whitish-gray color, that it has contracted more or less intimate adhesions with the neighboring organs, that the liver has acquired increased density, and is sometimes as resistant to the scalpel as leather, that the cut surface is intersected by white tracts interlacing around the granulations which they strangle, and which the light of the branches of the vena porta and biliary canals has notably augmented.

I prefer to discuss some less known peculiarities, in relation to the state of the hepatic cells and other parts of the structure of the liver—peculiarities which enable me to embrace the consideration of a great many of the phenomena of cirrhosis.

First of all, let us see what becomes of the secreting part of the gland—the hepatic cellule. Here, in a special manner, Frerichs will be our guide. The pressure which is exerted upon the biliary canaliculi and the capillary bloodvessels causes great disturbance in the nutrition and function of the organ. The hepatic cellules become disintegrated, and are, for the most part, transformed into a brownish pigment collected in small granulated heaps. Moreover, the bile secreted by the cellules which still remain valid, being able only imperfectly to circulate in the compressed canaliculi, accumulates in the cellules themselves, in the form of fine orange-colored granules. Everything, even the blood-globules, becomes changed within the obliterated ramifications of the hepatic veins: in consequence of their stagnation, they become decomposed, and transformed into a substance of a dirty red color, which, ultimately, is infiltrated into the acini of the liver. It is from this mixture of pigmentary detritus of altered cells, the infiltration of the rest of the parenchyma with biliary pigment, and the coloring matter of the blood-globules, that the special color of cirrhosed liver is derived—the color whence the disease takes its name. Thus, you must perceive the want of truthfulness in the doctrines which attribute cirrhosis to atrophy of the red and hypertrophy of the yellow substance—substances which are mere creations of the imagination of anatomists, being non-existent in the normal state.

I have now to speak of constriction of the capillary vessels which terminate in the portal vein. This is a topic of prime importance in the history of cirrhosis.

This condition presents à primary and purely physical obstacle to the portal circulation in the interior of the liver, so as gradually to produce dilatation of the branches, and then of the trunk of the vena porta vein. In this way, gentlemen, there is produced slight dilatation of the veins, and particularly of the small veins in the falciform ligament. These small veins emerge from the convex surface of the liver, creep over the inferior surface of the diaphragm, anastomose in the thickness of the abdominal walls with the epigastric and internal mammary veins, and then with the subcutaneous veins; and on the other hand, they enter into the transverse groove of the sinus of the vena porta. Thus, there appears under the skin of the abdomen a network of more or less dilated veins, which establishes that which you will allow me to call the *parietal compensatory circulation*, to distinguish it from another kind of compensating circulation, of which immediately I shall have to speak to you.

It is not, then, as Rokitansky and Bamberger have said, by a restoration of permeability in the umbilical vein, which neither is nor can be more than a permanently impervious fibrous cord, that this compensatory circulation in the abdominal walls is established; but by the dilatation of the small veins normally existing in the thickness of the falciform ligament—veins insignificantly small in the normal state, but which have to perform a part from the time that the intra-hepatic circulation of the vena porta is blocked up. However, to call them, as does M. Charles Robin, the "subperitoneal accessory portal veins," is perhaps to give them too important a name.* "Among the small veins of this group, there is always one," according to M. Robin, "which opens direct into the left branch of the portal sinus, at the point where it is attached to the cord of the umbilical vein. It is specially by this vein, which is destitute of valves, that the blood flows from the liver towards the abdomen, when that organ, being affected by cirrhosis, affords only an insufficient passage. But, as it communicates with the other veins, the fluid which flows into its cavity passes partly into them, so that the entire group shares more or less in its dilatation."*

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* Robin (Charles): Report on a Memoir by M. Sappey upon "Un Point d'Anatomie Pathologique relatif à l'Histoire de la Cirrhose." [*Bulletin de l'Académie de Médecine*, 1859, t. xxii, p. 944.]

In five subjects affected with cirrhosis, which M. Sappey injected, he found in all of them this enlargement of the accessory portal veins. It would appear, therefore, as M. Robin justly remarks, that the collateral circulation, which is carried on in consequence of dilatation of normal veins, is more usual than has hitherto been generally supposed. Only, as the reflux sanguineous current generally flows through the epigastric veins, which are deepseated and anastomose by numerous vessels with the accessory portal veins, it results that the subcutaneous veins dilate more slowly than the deepseated epigastric veins—that is to say, when they are no longer sufficient for the return of the blood carried by the accessory portal veins; and death sometimes supervenes before they have become sufficiently enlarged.

Dilatation of the superficial veins of the abdominal parietes may be considered, therefore, in the great majority of cases, as a symptom of advanced cirrhosis; but we must not adopt M. Sappey's opinion, "that it ought to be regarded as a somewhat favorable sign, from its removing or diminishing the causes of ascites," because the compensatory circulation is very feeble, and, moreover, from its lessening the causes of ascites, it embarrasses the normal circulation in the abdominal parietes, thereby rapidly producing œdema in that situation. My colleague, M. Monneret, has observed that the abdominal wall is rendered œdematous, and that the dermis itself becomes infiltrated, before the quantity of fluid effused into the peritoneum is sufficient to explain that œdema by ascribing it to distension of the abdominal parietes.

These new conditions under which the circulation is carried on in the subcutaneous abdominal veins sometimes determine the appearance of a double phenomenon which I have pointed out to you in a woman who lately occupied bed 2 of St. Bernard Ward. You no doubt remember this woman; her age was about fifty. Veins of great size ramified on her abdominal walls. On applying the finger to any of these veins, a vibratory thrill was perceived in it, exactly similar to that observed in the jugular veins of anæmic subjects; and which is always an indication of the existence of a vascular blowing, of which this thrilling sensation is the correlative phenomenon. I was therefore convinced that we should hear on auscultation a blowing sound in the dilated and vibrating superficial veins. Indeed, along with me you have been able to ascertain that there existed in this woman a soft vascular murmur, perceptible by the stethoscope. It would appear that this fact had been previously pointed out by M. Pégot, and afterwards mentioned by M. Bamberger;† but I was ignorant of their researches when I noted the similar observations I had made. To me it is utterly indifferent who has the merit of priority in this matter; and I have not taken the trouble to connect this unusual development of the abdominal veins with cirrhosis. I asked M. Sappey to have the goodness to come to see this patient, and also to make with me the examination of the body after death. In point of fact, it was in this subject, injected by M. Sappey with his accustomed dexterity, that he discovered the collateral veins of the suspensory ligament; and in this subject also, he was enabled to demonstrate that the compensatory circulation was carried on by the dilated normal veins, and not by the umbilical veins.

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* Robin (Charles): Same Report, p. 945.

† Robin (Charles): Ib., p. 960.

Excuse my giving you these anatomical details, which perhaps I have stated at too much length: and bear in mind, that I had to speak to you of a fact which was new or at least little known—the vibrating thrill and the succurrus in the subcutaneous veins when very much dilated—and that I had likewise to correct the error which attributed this compensatory circulation to the restored patency of the umbilical veins: for these reasons, the details which I have given seemed indispensable. I now revert to the consequences of constriction of the terminal extremities of the portal capillaries.

I have told you that the first of these consequences is the establishment of a parietal compensatory circulation; and the second, the development of a compensatory circulation in the hepatic artery. In the normal state, as you are aware, the terminal capillary branches of the vena porta frequently anastomose with those of the hepatic artery, or in other words, there is anastomosis between the secretory and nutrient circulations of the organ. In the normal condition, this fact can be demonstrated by injections. It can also be shown by injections, that the anastomosis is much greater in the cirrhosed than in the healthy liver. In the cirrhosed liver, from which the hepatic cellules and acini have disappeared, where consequently there are no longer the areolæ which belong to the acini, where, as a result of the morbid action, the cellules and acini have been replaced by connective tissue of recent formation, we find an entirely new distribution of the capillaries, which have their direction modified, and admit of being injected not only by the vein, but also by the hepatic artery. "Thus it is," says Frerichs, "that new channels of communication are formed between the vena porta and hepatic veins, though their number is limited and inadequate, considering the quantity of blood which traverses the vena porta."*

In connection with this fact, gentlemen, I cannot avoid mentioning another which has been pointed out by my colleague, M. Natalis Guillot, in relation to pulmonary tuberculization. This able physician, as you know, has shown, in respect of pulmonary tuberculosis, that in proportion to the degree in which the area of the circulation of the pulmonary artery is diminished, the circulation is augmented in the bronchial arteries. The cirrhosed liver, therefore, like the tuberculosed lung, has a new circulation: there is a diminished capacity for portal blood in the one organ, just as, in the other, there is a diminished capacity for the blood of the pulmonary artery: inversely, the capacity for the blood of the hepatic artery, and for the blood of the bronchial arteries, is respectively increased. To express the same fact in different language: both diseased organs receive less blood for functional, and more for nutritive purposes. Unfortunately, the nutrition is morbid.

The new circulation in the tuberculosed lung is useless for pulmonary hæmatosis, just as is the compensatory circulation in cirrhosed liver for hepatic hæmatosis. When the pulmonary vesicles no longer exist, there is a final cessation of the exchange of gases: and when the hepatic cellules have disappeared, secretion of bile and formation of glucose are impossible. There is, in both cases, an arrest of hæmatopoiesis.

A third consequence of the constriction of the terminal capillaries of the vena porta is a reflux of blood into the spleen, and consequent congestion of that vascular organ. As the spleen undoubtedly plays an active part in the formation of the blood, be it in forming the red globules or the white globules, or in destroying the red globules, we can understand that hæmatopoiesis is disturbed by stasis and embarrassment in the splenic circulation.

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* FRERICHS: Traité Pratique des Maladies du Foie, p. 297, 2me édition. Paris, 1866.

A fourth consequence of the constriction of the portal distributive capillaries is reflux and stasis throughout the originating portal capillaries, that is to say, throughout the entire mucous membrane of the digestive canal. Thus it is, that intestinal absorption is diminished and then suspended : the nutritive materials, the nitrogenous substances, are no longer drawn by these capillaries into the intestinal villi, poured from them into the vena porta, conveyed from them into the liver, to be there elaborated and modified, and then delivered by the vena cava to the heart, to be sent by it to the deepest-seated and most remote parts of the organism. You can understand, gentlemen, that the cessation of this succession of phenomena very directly and very seriously compromises the function of hæmatopoiesis.

A fifth consequence of embarrassment in the portal circulation of the liver—a consequence more rapid, more easily seen, and consequently better known—is the effect produced on the peritoneum. Vascular stasis in that situation causes dropsy. The ascites is directly proportionate to the duration and gravity of the cirrhosed state of the liver. It is a means provided for the relief of the engorged radicles of the vena porta.

A final consequence, a natural and rationally inferred result of embarrassed portal circulation in the liver is hyperæmia of the mucous membrane of the stomach and intestines—with, as a result of this hyperæmia, hemorrhages and fluxes. But here, observation and induction are not in accord. Gastro-enterorrhagia is of rare occurrence, as Frerichs himself is obliged to admit: in a certain number of cases, however, it happens, that increased pressure in the capillaries leads to their being torn, so giving a sanguineous coating to the mucous membrane. Fluxes, however, which may occur in the form of gastrorrhœa or enterorrhœa, are equally rare: here, consequently, I repeat, observation and induction not being in harmony, we are obliged to combat assertions of a rather hypothetical character by an appeal to facts.

Having stated the consequences of obstruction to the portal circulation in the interior of the liver, I have now to speak of the organic derangement and functional disturbance which take place consequent upon alteration of the biliary passages and hepatic cellules.

The same cause which produces strangulation of the capillaries of the vena porta, also, in part, strangles and atrophies the radical biliary canals. The principal branches of these canals sometimes participate in the general inflammatory irritation of the gland, presenting, at the same time, increased development of their walls and thickening of their mucous membrane. Sometimes, also, the walls of the gall-bladder are hypertrophied; and, like the liver itself, have contracted adhesions with the neighboring organs. The liquid contained in the gall-bladder is tenuous, flowing, of a pale yellow or orange color, and is evidently a mixture of bile and mucus.

We have seen that many of the hepatic cellules are destroyed: the consequence of this is a great diminution, and sometimes an almost total suppression of the secretion of bile. Thus, the bile ceases to play its part, whatever that may be, in intestinal digestion, which is necessarily very much disturbed. This disturbance is probably the cause of an excessive production of gas in the intestines. At all events, we can see in the disturbance of digestion, in the imperfect elaboration of the alimentary substances intrusted to the intestine, a new and powerful cause of wasting and anæmia. Thus, in cirrhosis, everything seems to concur to produce an altered state of the blood and destruction of the organism.

The remarks I made to you on the etiology of cirrhosis must have led you to the conclusion that the *symptomatology* of the disease is exceedingly complex and obscure. In fact, of all the symptoms directly referable to it, the most prominent and characteristic is ascites.

The progress of the local affection is generally slow and insidious, no well-marked symptom appearing at its commencement; or, at most, in some very rare cases, we have, as indications of the inflammation, dull pains in the right hypochondrium, and slight increase in the volume of the liver, causing it to extend beyond the false ribs: at the same time, the tongue is loaded, there is anorexia, sometimes accompanied by nausea, vomiting, and a little jaundice. There is sometimes also slight fever, particularly in the evening. These exceptional occurrences are exceedingly transitory.

Progressive diminution of appetite, increasing slowness and great difficulty in digestion, flatulence and constipation, are generally present. The general nutrition is at fault, the patient loses flesh, and his strength fails: from what I have said to you regarding the pathological physiology of cirrhosis, you can see how this must be so. The skin loses its color, the anæmia becomes more and more evident, and the whole appearance of the patient indicates the existence of that great disturbance of the function of hæmatosis of which I have had to lay before you the many co-operating causes.

Ascites has already shown itself; it increases slowly, advancing in accordance with the progress of degeneration of the liver. Generally, the abdomen acquires a characteristic appearance from the increased development of the subcutaneous veins. The ramifications of these veins can be seen under the skin, which they raise up: they form a vascular network radiating from the umbilicus up towards the epigastrium, and down towards the inguinal region. Such was the case in our patient of bed 2, St. Bernard Ward, in whom the exceedingly dilated veins were the seat of a vibratory thrill, accompanied by a vascular blowing sound perceptible by the stethoscope.

Sometimes, under these circumstances, the abdominal walls and lower extremities become infiltrated. The production of this œdema has been attributed to the compression of the common iliac veins and the vena cava inferior by the effusion into the peritoneal cavity. But is not this œdema partly explained by the disturbance in the circulation in these regions, which is caused by the reflux in the sanguineous current from the vena porta? Opposed to the sanguineous current passing from below upwards by the epigastric veins is the new current proceeding from above downwards from the vena porta. Any obstruction to the circulation in the epigastric veins must be felt in the external iliac and crural veins. A similar cause produces a similar effect upon the venous circulation of the abdominal walls.

Be that as it may, the emaciation of the face, superior extremities, and trunk, forms a contrast to the œdematous tumefaction of the inferior extremities.

I shall not expatiate on the local disorders which are associated with the existence of ascites—such, for example, as embarrassed respiration proportionate in degree to the amount of effusion, and a more marked meteorismus consequent upon derangement of the digestion.

Little by little, the general debility and wasting increase, and death comes as the slow result of the progressive exhaustion, or it may occur more rapidly from an intercurrent affection, commonly called a complication, but which is, in my opinion, merely the consequence of the same morbid state which has caused the cirrhosis. Thus, it is not unusual to observe as the terminal phenomenon pulmonary apoplexy, secondary bastard pneumonia, or pleurisy, when the cirrhosis coexists with cardiac disease. In one addicted to alcoholic drinks, the cirrhosis is coexistent with Bright's disease or delirium tremens. Do not be surprised at the frequency of the coincidence of cirrhosis and Bright's disease; both are produced in drunkards by the action of alcohol, and both are results of prolonged passive congestion in persons suffering from disease of the heart. The fact had often been stated; but without the common etiology of the two affections being pointed out. Occasionally, however, real complications occur, such as pulmonary tuberculosis and carcinoma.

But, gentlemen, perhaps an objection presents itself to your minds similar to that which has often presented itself to my own. Cirrhosis is really chronic atrophy of the liver. You are aware that when the liver ceases to perform its functions, there is a cessation in the metamorphosis of the material, and a consequent accumulation in the blood of substances which, as the result of that metamorphosis, constitute the elements of the bile. It is then that there appears in the urine a number of substances foreign to the organism, such as leucin, tyrosin, and a peculiar extractive matter. Uric acid shows itself only in moderate quantity; and urea, which is the final result of the decompostion of the albuminoid substances, little by little, entirely disappears. How, then, does it happen, that in cases of chronic alcoholia, we have not the disorders of the nervous system, which have been pointed out as occurring in acute atrophy of the liver, that is to say, delirium, convulsions, and coma—or, at least, that their occurrence should be so exceptional as never to have been met with by me, and to have occurred in only two cases mentioned by Frerichs.*

Here, it is impossible not to advert to albuminuria, in which affection we sometimes observe the presence of alarming subacute symptoms, and, at other times, the absence of such symptoms. According to the views of modern chemical physicians, uræmia is caused by the accumulation in the blood of urea, which the diseased kidneys are no longer able to eliminate. But if the symptoms of uræmia depend upon this cause, accumulation of urea in the blood, which is entirely physical, and upon adulteration of the blood from the decomposition of that urea into carbonate of ammonia, a consequence entirely physical, it is both a physical and a chemical necessity that the adulteration of the blood should be greater in proportion to the extent and duration of the morbid state of the kidneys. These are exactly the conditions existing in chronic albuminuria. How is it then, that it is precisely in this form of albuminuria that uræmia is most unusual; and that, on the contrary, it is observed in acute albuminuria, that is to say, in the cases in which poisoning of the blood by urea cannot be complete?

Is this an example of tolerance; and must we class these facts with the facts so commonly observed in acute and chronic phthisis? In chronic phthisis, for example, we see great loss of substance. We reduce the field of hæmatosis to a minimum, and yet the patient continues to live; whereas, in acute phthisis, with much less extensive lesions, and a larger field of hæmatosis, the patient it asphyxiated. Possibly, in the chronic atrophy of the liver called cirrhosis, and the chronic destruction of the lungs called pulmonary phthisis, there exists a parallelism between the deterioration of the organism and that of the organ, so that the requirements of hepatic and pulmonary hæmatosis diminish with the diminishing size of the organ. To such an extent is this the case, that the patient may go on living for a long time, though in a very imperfect manner, as the supply is always less than the demand, till the time arrives when the continuance of life ceases to be compatible with an absolutely inadequate production of blood. On the other hand, in acute atrophy of the liver, as in acute phthisis, the organism is abruptly, not gradually, deprived of the sanguineous supply which is indispensable, the disturbance which ensues being so great that death speedily takes place.

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* FRERICHS: Traité Pratique des Maladies du Foie, p. 285. Paris, 1866.

Again, why is it that cirrhosis, notwithstanding the undoubted atrophy of the liver which it produces, so seldom causes jaundice; whereas, it is one of the principal characteristics of acute atrophy, a characteristic which has conferred on that affection the name of *ictère grave?* Is that explained by the fact that in cirrhosis the atrophy takes place by slow degrees, and that in spite of the destruction of the greater number of hepatic cellules, some of them always remain to secrete the bile; whereas in acute atrophy, the morbid change advances simultaneously and rapidly in all the secreting cellules? Frerichs observed jaundice in seven only of thirty-six cases of cirrhosis, and, except in two of the seven, it was slight. He attributed this slight jaundice to pressure on the radicles of the biliary passages by the connective tissue recently formed around the lobules of the liver. "It is," he says, "the same cause which imparts a yellow color to the liver itself, and which is the origin of the term cirrhosis."

You are also aware that hemorrhage from all the passages is one of the most remarkable symptoms of *ictère grave.* In fact, so common an occurrence is hemorrhage in acute atrophy, that my colleague, M. Monneret, has described the affection under the name of hemorrhagic jaundice [*ictère hémorrhagique*]; and the hemorrhage is attributed to a fundamental change in the constitution of the blood. Now, hemorrhages are far from being frequent in cirrhosis. I have told you that the hemorrhages which may result from stasis of the blood in the mucous membrane of the digestive canal are by no means of frequent occurrence. As to cutaneous hemorrhages in the form of petechiæ, or as to hemorrhages into the pituitary and buccal membranes, or on the surface of serous membranes, or in the form of cerebral or pulmonary apoplexy, I do not know of one example, if I exclude hemorrhages depending on a disease of the heart, of which the cirrhosis itself is an effect. Frerichs states, however, that they are not so very uncommon. Be that as it may, they are infinitely less frequently met with than in acute alcoholia, although in cirrhosis there exists great atrophy of the liver, and great consequent disturbance of the function of hæmatosis.

I have still to lay before you certain opinions of a New York professor of physiology—opinions which have a direct bearing on cirrhosis. Dr. Flint has made some very ingenious experiments, which have led him to conclude that the liver is in respect of cholesterin what the kidneys are in respect of urea—an organ of elimination and not of production. He considers that cholesterin is an *excrementitial* product, formed to a great extent by the *disassimilation* of the brain and nerves; that it is an effete substance [*substance usée*], separated from the blood by the liver; and that it is thrown out into the upper part of the small intestine. In passing along the digestive canal the cholesterin is transformed into *stercorin*, a ternary compound identical with the *séroline* of Boudet: the stercorin is finally expelled with the fæces. It is not cholesterin then, but stercorin which is found in the fecal matter.

Here are the proofs given in support of these doctrines: the blood of the internal jugular vein contains a much larger proportion of cholesterin than is contained in that of the carotid artery, which in fact contains very little:

therefore, the blood which returns from the brain is much richer in cholesterin than that which goes to it: consequently, it must be in the brain that the cholesterin is produced. Again, the blood of the femoral vein contains more cholesterin than the blood of the femoral artery: that is to say, that the nerves of the lower extremities have furnished this predominance of cholesterin in the venous blood. Let me remark, however, that I look upon the conclusion arrived at by Dr. Flint as not strictly logical; for it may be quite well alleged that the excess of cholesterin is derived from the muscles, or from all the tissues of the lower extremities, as well as of the nerves. The experiment in which it has been shown that the venous blood of paraplegic limbs contains less cholesterin than that of healthy limbs only proves that the nerves produce the cholesterin, seeing that in paralyzed muscles nutrition unquestionably suffers, and that in them, consequently, disassimilation must necessarily be languid.

On the other hand, the blood of the hepatic veins is, according to Carter's experiments, much less rich in cholesterin than that of the hepatic artery; that is to say, that the blood which comes from the liver contains much less cholesterin than that which goes to it. To the extent then, that cholesterin is formed by the nervous system, it is taken up by the blood, which throws it off as it traverses the liver.

It is evident that, if this were the case, suspension of the functions of the liver must lead to a cessation of the elimination of the cholesterin, which, by accumulating in the blood, will produce cholesteræmia.

Chemical analysis of the blood of a patient who had been laboring under cirrhosis, and who died in a state of profound stupor, demonstrated to Dr. Flint that there was a large increase in the amount of cholesterin contained in the blood. Becquerel and Rodier also found a large quantity of cholesterin in the blood in an analogous case.

The nervous complications to which Frerichs has directed attention, and of which I have spoken to you, are complications which sometimes supervene at the end of cirrhosis, and which the learned German physician* attributes to alcoholia, that is to say, to suppression of the functions of the liver and a cessation of the production of bile. Dr. Flint attributes the symptoms to cholesteræmia, comparing them to those produced by uræmia, which, as you know, is the result of the accumulation of urea in the blood, consequent upon suspension of the functions of the kidneys.

Dr. Flint says, that the reason why cholesteræmia does not exist in every case of cirrhosis is because the whole of the liver is not disorganized, and that a part of the organ suffices to eliminate the cholesterin, just as, in cases of degeneration or ablation of one kidney, the other continues to eliminate urea.

I must add, that the very interesting experiments of Dr. Oré (of Bordeaux) appear to support the views of the New York professor. In fact, it would appear from the experiments of Dr. Oré, that the bile is secreted from the blood of the hepatic artery, and not from that of the vena porta. We know, on the one hand, that the portal blood contains nitrogenous matters derived from the digestive canal, and very little cholesterin; and on the other we know, from the analyses made by Carter, that the blood of the hepatic artery contains a large quantity of cholesterin.

Gentlemen, I have thought it right to place before you this ingenious theory. It is specially important to take every opportunity of bringing into comparison the results of clinical observation and experimental physiology. In respect of cirrhosis, I have also thought it incumbent on me to inquire, along with you, into the possible mechanism by which this redoubtable lesion of an organ so important as the liver inflicts such irreparable damage on the economy.

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* Robin (Charles): Journal de l'Anatomie et de la Pathologie, for September, 1864.

Were I to wish now to embrace in one general view the entire pathological mechanism of cirrhosis, I should perceive that it consists of a concatenation of phenomena which succeed one another in a necessary order, because they originate the one from the other. In chronological order the phenomena are: exudation of an organizable blastema into the interlobular connective tissue—organization of this blastema, which becomes fibrous—gradual retraction of this fibrous tissue, which both strangles the secretory parenchyma and compresses the divisions of the vena porta. Thence, as a twofold consequence, we have consecutive atrophy of the liver, and gradually increasing embarrassment in the portal circulation.

But we have seen, that as soon as the portal circulation is impeded, the secretion of bile and of glucose becomes less and less; and that in respect of nutrition, the alimentary materials taken up by the radicles of the mesenteric veins ramifying on the mucous membrane of the intestine, enter with increasing difficulty the vena cava, thence to pass into the general circulation.

The organism, therefore, is not only threatened on account of the hæmatosis being incomplete in consequence of the ever diminishing secretion of bile and glucose, but also on account of nutrition being directly interfered with in consequence of the inadequacy of the amount of assimilable materials floated along the vena porta, then along the vena cava, and finally propelled by the heart to the remotest recesses of the organism, to carry thither the elements of reparation.

We have already seen the direct consequences to the economy of a morbid state of the liver. Nor is that all: we have seen that cirrhosis is generally a secondary affection occurring in the course of, and as a consequence of, a previous malady which contributes a large share to the deterioration of the organism.

Thus, in the subject affected by alcoholism, there is diarrhœa with concurrent chronic gastritis of sometimes an ulcerous character, the alcohol exerting its direct deleterious action on the mucous membrane of the stomach prior to its irritating the liver. The dyspepsia resulting from the malady of the stomach disturbs nutrition at its very source, and becomes additional to the dyspepsia proceeding from the cirrhosis. The series of lesions and functional disorders which belong to chronic alcoholism, are also often superadded; to this category belong fatty degeneration of the heart, atheromatous degeneration of the arteries, atrophy of the brain from true cirrhosis, chronic bronchitis, even pulmonary tuberculosis, granular disease of the kidneys, all of which, I need not say, exercise an evil influence on the entire organism.

In one who is the subject of heart disease, the position is also very serious. In such an individual, the vascular stasis which interferes with hepatic hæmatosis has already for a long time compromised pulmonary hæmatosis; in point of fact, he is affected with pulmonary congestion, chronic bronchitis, or bronchorrhœa; and to the debility resulting from the progressive diminution in the pulmonary hæmatosis, there is added that which is the natural consequence of the loss arising from abnormal bronchial secretion. On the other hand, the renal depuration of the blood is generally imperfect, in consequence of the kidneys being usually in a congested state, and often from the albuminuria caused by the congestion, in which case, the situation of the patient is still more aggravated,

In the syphilitic subject, the poisoning is general: when the virus is so deeply rooted, and the affection so inveterate as to have become visceral, there is not only cirrhosis of the liver but also a structural change in the kidneys and spleen—often also in the lungs. The kidneys frequently undergo amyloid degeneration, and the result of this is albuminuria. Here, everything concurs to produce a morbid change in the blood: the liver is an organ both of hæmatosis and of depuration; the kidneys serve only as depurators.

The chances of the clinical wards have not enabled me to show you the lesions which belong to syphilitic cirrhosis. To give you a sufficiently exact idea of the nature and extent of the lesions, I cannot do better than quote, in an abridged form, a typical case from the work of Frerichs.

His thirty-ninth case* is the history of a woman, thirty-six years of age, who had suffered on several occasions from primary and secondary syphilis. Two years before admission to the hospital, she had been treated for albuminuria complicated with anasarca, from which she was easily relieved. She came into hospital with all the symptoms of a profuse pleuritic effusion of the right side. She died fifteen days afterwards, having had frequent nausea and vomiting, white serous stools, and delirium for a short time.

At the autopsy, in addition to the effusion into the right pleura, a multiplicity of lesions were found. The mucous membrane of the small intestine presented an intensely injected appearance; and its solitary glands were prominent. The large intestine was also injected. The pancreas was hard; and the mesenteric glands had in part become calcareous. The spleen was large, hard, and lardaceous; and, from the presence of amyloid granules, had a brilliant aspect. The kidneys were bulky; their parenchyma was in part hard and lardaceous, and in part friable, with yellow infiltration. Everywhere, there were intimate adhesions between the liver and diaphragm: *the left lobe was completely atrophied*, and was almost indistinguishable from the diaphragm: the right lobe presented, on its convex surface, and on its upper margin, deep cicatricial contractions which circumscribed portions resembling lobes of the size of hazel-nuts. The parenchyma was everywhere knotty, very hard, and of a shining reddish-brown appearance. The bile was thick and mucous, nearly gelatinous, and of a dull color: it deposited a large quantity of coloring matter, and was without albumen.

You see that in this case, the patient presented simultaneously amyloid degeneration of the liver, spleen, and kidneys. In other words, there coexisted disturbance of the function which presides over the formation of the blood by the liver and the spleen, and disturbance of the function by the exercise of which the blood is depurated by the kidneys. It appears then equally incorrect and irrational to consider that this woman succumbed solely from her hepatic affection: it is likewise seen, how difficult it would be to describe the cirrhosis, leaving ascites out of view, as a separate isolated disease, and to say, how much of the complex symptoms in this case were positively due to the hepatic lesion. In point of fact, the woman died from visceral syphilis presenting everywhere the numerous lesions and disorders which belong to this disease, and not only from the liver having become cirrhosed.

When you find granular degeneration of the kidneys described in your treatises on pathology among the complications of cirrhosis, you are inclined to say that an unrecognized general disease is being discussed, and that the effect has been mistaken for the cause, or rather, that there has been a failure to detect the disease on which a series of lesions depended. That is the conclusion to which an excess of pathological anatomy must inevitably lead.

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* FRERICHS: Op. cit., p. 333.

I cannot too often repeat, that cirrhosis, in an immense majority of cases, is *not an isolated disease*, is not, in fact, a disease at all, but a secondary lesion, connected by a relation of causality with other lesions of similar origin, such as those arising from cardiac, paludal, syphilitic, and alcoholic affections; and that it is only by adopting an artificial basis of description, unsanctioned by a correct interpretation of facts, that it has come to be described as a distinct disease, with precisely defined characteristics.

Were cirrhosis regarded, as I regard it, from an etiological point of view, and not from an anatomical point of view—were the lesion looked upon, not as made but as being made—were the idea followed of the action of the general morbific cause, we would be present, so to speak, at the genesis of the complications. We would then understand, for example, how, in consequence of the alcoholic poison exerting its irritant action in succession on the stomach, liver, and kidneys, there often occurs simultaneously thickening and ulceration of the gastric mucous membrane, cirrhosis of the liver, and Bright's disease of the kidney—how the syphilitic poison acting on the organs of hæmatopoiesis, produces simultaneously amyloid degeneration of the spleen and kidneys with cirrhosis of the liver—and, finally, how the generality of cause engenders multiplicity of lesion.

The remarks I have now made show you how difficult is the diagnosis of cirrhosis. In the totality of symptoms depending on the general affection, cirrhosis hardly has any other signs which belong to it, except ascites and enlargement of the subcutaneous abdominal veins. For we must beware of supposing that the venous murmur of which I spoke to you is constantly present.

It is with chronic peritonitis, and particularly with tuberculous peritonitis, that the ascites due to cirrhosis may be confounded. But, generally, tuberculous peritonitis coexists, in the adult, with pulmonary tuberculosis; and then, the signs peculiar to the latter affection enable us to recognize the tuberculous origin of the peritoneal effusion. There are, moreover, in tuberculous peritonitis abdominal pains, whereas they do not occur in the ascites arising from cirrhosis. Cases perfectly inexplicable do, however, occur; and the following is an account of one of that description.

A man, forty-seven years of age, was admitted to the clinical wards on the 3d March, 1864. He had suffered from difficulty of breathing for five months; and so much had his health declined, that he was no longer able to do his usual work. In fact there was extensive pleuritic effusion on the right side. The pleurisy, on its first declaring itself had been treated by a blister, which was three times repeated without any obvious beneficial effect. There was absolute dulness extending from the spine of the scapula behind to the second rib in front. In that situation, no respiratory murmur was audible. The liver was squeezed down as low as the umbilicus. From the great extent of this effusion, I resolved that paracentesis of the chest should be performed by Dr. Peter, my *chef de clinique*. By the operation, 2300 grammes of serosity were withdrawn: the dyspnœa ceased quickly, and did not return: the patient, however, remained pale, and recovered his strength slowly. Although he had no cough, and although I could detect no abnormal sound at all attributable to pulmonary tuberculosis, yet as his pleurisy was on the right side, has assumed the latent form, had advanced in a chronic manner, and finally, as the fingers were Hippocratic, I concluded that the man was tuberculous. My suspicions were in part confirmed, when the patient, who had gone out at his own request, asked, on the 25th April, to be readmitted. He was then thinner and weaker than when he left us; and he had considerable effusion into the abdomen. This effusion had been preceded by wandering abdominal pains. I had no hesitation in concluding that the dropsy was caused by tuberculous peritonitis. No conclusion, apparently, could be more rational. The form and progress of the preceding pleurisy, and the pains which had preceded the peritoneal effusion, seemed to support the view of the simultaneous existence of tuberculosis of the pleura and peritoneum. There was, nevertheless, a specialty in the case which caused me to hesitate, and kept my mind in suspense: it was the absence of any pulmonary sounds peculiar to tuberculosis.

This patient remained six weeks in our wards, becoming feebler day by day, having hectic fever, vomiting nearly everything he ate during the three latter weeks of his life, and suffering slightly from diarrhœa. At last, he died in a state of marasmus.

At the autopsy, we found traces of chronic pleurisy of the right side, with close adhesions between lung and pleura. There was about half a litre of lemon-colored serosity in that side of the chest; and in the left side, there was nearly a litre of similar effusion. Thus, there were the signs of chronic bilateral pleurisy: and yet we could not find any tubercular deposit in the lungs. Nor was there any tubercle in the peritoneum, which, throughout its whole extent, was exceedingly injected. The intestinal convolutions were, here and there, lined with false membrane, which was thin and soft. The peritoneal effusion was copious, and of a dirty color. There, therefore, existed chronic peritonitis: there was also a morbid condition which we did not expect to find—cirrhosis of the liver. The liver was small and indurated: it presented, externally, a fine granulated appearance: the granulations were as large as millet-seeds, or, at most, as large as hemp-seeds: white lines seamed the surface of the liver, and in the situation of the majority of the lines, there were slight depressions, some of which were linear, while others were broader: they all corresponded with the trabeculæ of the capsule of Glisson. A section of the liver presented a fine granulated appearance.

That is, I think, the form of cirrhosis which is associated with chronic alcoholism. It looks like a raking of the organ, which does not present any projections of the size of hazel-nuts, such as are found in syphilitic cirrhosis: none of the granulations are larger than hemp-seed: there is an almost general thickening of the capsule of Glisson.

The mucous membrane of the stomach was enormously injected: in some parts, it was of a slaty color, in others, it was bright red; and this latter was the appearance it presented in the neighborhood of the pylorus. The mucous membrane of the small intestine, particularly in its lower portion, was much injected: there were a certain number of oval ulcerations on the valvulæ conniventes, and some circular ulcerations in the situation of the separate follicles. There was no trace of tubercular deposit in any part of the intestinal canal.

We ascertained, as the result of careful inquiry, that the man had been an habitual drinker of alcoholic stimulants. Appearances, notwithstanding, he was not tuberculous; and we must ascribe his gastro-enteritis to alcoholism. The gastro-enteritis probably caused, by propagation, the peritonitis; for the peritoneum presented the greatest appearance of injection in the situation of the intestinal ulcerations and in that of the vascularity of the mucous membrane of the stomach. The peritoneal effusion, therefore, was due both to the peritonitis and the cirrhosis. As to the latter, there was necessarily misconception, deceived as one had previously been by the anterior pleurisy, and signs of peritonitis—pleurisy and peritonitis which it was much more truth-like to ascribe to the tubercular diathesis.

In fact, cirrhosis ought to be suspected when we have ascites appearing in a cachectic drunkard, or in a syphilitic subject who has reached the period when tertiary symptoms occur: we ought also to suspect its existence in a person affected with cardiac disease, when the ascites is to a much greater degree than the œdema of the inferior extremities, particularly if there be dyspepsia with marked wasting of the face and superior extremities. Finally, there is reason to believe that cirrhosis exists when there is ascites without our being able to discover any material obstacle in the course of the vena porta.

Having told you how cirrhosis, a lesion secondary to a more general affection, adds its share of gravity to a condition already very formidable—having explained to you how, by impeding hepatic hæmatosis and drying up the very sources of assimilation, it assails life in a doubly dangerous manner—it would be wasting time to insist upon the gravity of the *prognosis.*

In that gravity, however, there are different degrees, according to the period of the lesion and the nature of the affection on which the cirrhosis depends. Thus, for example, incipient cirrhosis is evidently less grave than cirrhosis in its last stage; and cirrhosis depending on paludal toxæmia is less grave than cirrhosis arising from an affection of the heart, which, again, does not become so rapidily formidable as cirrhosis originating in visceral syphilis or alcoholic poisoning.

What I say in respect of prognosis, is applicable also to *treatment.* That is one of the reasons which led me to enter so fully into the etiology and evolution of cirrhosis. Therapeutics, in fact, avail only when the lesion is incipient: when it is confirmed, treatment is of no use.

The endeavors, therefore, of the physician must be directed to prevent cirrhosis, or to impede its progress. When, therefore, in a person who has an affection of the heart, you ascertain that there is enlargement of the liver, local pain, and a slight icteric color, you must set yourself to subdue this manifest congestion of the liver. But as this congestion is connected with embarrassment of the circulation, the means to be adopted for diminishing the general vascular tension and the consecutive visceral congestion are: regulating the circulation by digitalis, slightly stimulating the system by the use of a little coffee, which acts as a diuretic as well as a stimulant.

Again, when in a drunkard, disturbance of the function of digestion leads you to apprehend the imminence of cirrhosis, you must advise the patient at once to renounce his bad habits, recommending him at the same time to make use of aliments but little stimulating, and easily assimilated.

In cases of syphilitic and paludal cirrhosis, simultaneously combat the specificity of the cause by a specific medication, and the debility of the system by general tonic treatment. You will treat the lesion of the liver, with its immeditate consequences, viz., anorexia, dyspepsia, ascites, &c., by an appropriate medication, which will vary with the varying indications, but which, too often, can only be palliative.

I trust that I have now shown you that the history of cirrhosis is, so to speak, only an episode in the history (in other respects very complex) of cardiac, syphilitic, alcoholic, or paludal cachexia: but then, it is an important episode. It is, let me say, one of the terms in a morbid series embracing all the organs, which commences with congestion, continues by phlogosis, and concludes in cachexia. Once the minute mechanism of cirrhosis is understood, there is little ground for hoping much from therapeutics. Art is necessarily powerless, when the lesion is irremediable. This fact I have already sufficiently impressed upon you.

LECTURE LXXXIX.

ADDISON'S DISEASE.

A Special Disease.—A Peculiar Kind of Anæmia, generally associated with an Affection of the Suprarenal Capsules.—A Few Words regarding the Suprarenal Capsules.—Symptoms of Addison's Disease.—Consequences of the Anæmia.—Peculiar Dingy Color of the Skin.—Difficulty of Diagnosis.—Treatment.

GENTLEMEN: To testify the gratitude which Art and Science owe to eminent men, perhaps also with a view to avoid the creation of new words, physicians—who, speaking generally, are slow to render justice to their brethren—have given to certain diseases the names of their discoverers, or the names of the individuals who first gave a complete description of them. For example, under the name of "Pott's Disease" we know a disease of the vertebral column characterized by caries of the vertebræ, with or without tubercular deposit, giving rise to abscesses from congestion and usually leading to spinal deformity, sometimes to paraplegia. In the same way, by "Bright's Disease," we mean chronic albuminuria, an affection which the celebrated London physician studied and described better than it had been previously studied or described by any one. In the same way, insufficiency of the sigmoid valves of the aorta is still called "Corrigan's Disease." And it would on the same principle be an act of justice to give the name of "Bouillaud's Disease" to endocarditis, an affection almost quite unknown, till the illustrious Professor of the Hôpital de la Charité directed to it the attention of the medical world, by writing a history of it so complete as to leave nothing to be added by others.

In obedience to a similar feeling of equity, I propose to-day to designate the disease of which the individual occupying bed 5 St. Agnes Ward presents a remarkable example, by the name of the English physician who discovered it. The physician to whom I refer is Dr. Addison, the fellow-laborer of Bright, the Dean of the Medical School attached to Guy's Hospital, London, a man who has long been known among us by his valuable scientific works. I propose then to give the name of "Addison's Disease" to that singular cachexia which is specially characterized by the decoloration, or rather the peculiar coloration—the bronzed tint—of the skin, which obtained for the malady the name under which Addison described it, viz., *bronzed disease.*

In the course of his practice, Dr. Addison had long been struck by meeting with certain forms of general anæmia which were neither attributable to excessive antecedent hemorrhages nor to profuse or long-continued intestinal fluxes, and could not by symptoms be connected with any diathesic state or marsh miasmatic affection—forms of general anæmia, in fact, which appeared to supervene under the influence of non-recognizable causes. In studying them, he distinguished from the rest one which was, besides the debility and languor of the patient, characterized by that bronzed hue of the integuments of which I have been speaking, and which is most strikingly apparent on the skin of the hands, penis, scrotum, groin, and axilla.

Dr. Addison having observed that in every case the malady followed the same course, and invariably terminated in death, made very careful autopsies with a view to obtain the greatest possible amount of information regarding the nature of the disease. From the minute manner in which he was in the habit of directing his inquiries into morbid states of the kidney, he discovered that in this singular form of anæmia, the suprarenal capsules were often diseased. The coincidence of a morbid change in an organ, the function assigned to which in the economy had till then been insignificant, the coincidence of an anatomical lesion apparently so insignificant with so serious a general condition of the economy, soon put him on the right track. From the date of his first case, he thought that he had found the explanation he was seeking.

In 1855, Addison published the results of his observations in a monograph containing the history of eleven cases, which, although not all equally decisive, enabled him to conclude, that the *bronzed disease* is associated with a lesion of the suprarenal capsules, without assuming, however, that this lesion (whatever might be its nature) was the cause of the disease, any more than the alteration which takes place in Peyer's glands is the cause of typhoid fever, pustules the cause of small-pox, or engorgement of the spleen the cause of paludal intermittent fever.* In all these examples, the organic lesion is only a characteristic part of the disease, distinguishing it from other morbid species of a similar kind.

The profession having thus been put on the alert in this inquiry, similar cases were, during the same year, recorded by the English medical press; and in France, my friend, Dr. Lasègue, made known Dr. Addison's researches, by publishing an analysis of them.†

With the case under notice in our wards, with that example so to speak, under our eyes, it is my desire to-day to give you an idea of Addison's Disease, following the description of the English physician, as reproduced by my learned colleague of the Hôpital Necker.

The following is a summary of our patient's case. His age is thirty. He was coachman to the Minister of the Interior. His occupation, although certainly an arduous one, was not so relatively, because he was well paid, well fed, and well clothed: he had no drawback, except insufficient sleep; and, in fact, only complained of inability to obtain his necessary amount of rest at night. When I saw him, however, at the date of his admission to the hospital, he was under the impression that he had been losing flesh for the preceding four or five months, in such a way as to alarm both himself and his family. For three months, he had observed that his hands retained a dark hue, however much care he bestowed on washing them; that his face was assuming the bistre, smoked aspect of a mulatto's skin; that the skin of the inside of his lips had a hue reminding one of the interior of the mouth of certain dogs; and finally, that the dark color was making its appearance on different parts of his body, and was in no degree amenable to the prolonged use of baths.

I verified the change of color which I have now described. The patient's nails had that remarkably white appearance met with in anæmic persons, proving that the dark hue of the hands with which they contrasted was not the result of any neglect of cleanliness. The dark hue was found on the skin of the penis, scrotum, groins, and arms. The areola, however, of the nipple, which, in similar cases, often acquires a deep hue, as in pregnant women, had its natural color.

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* ADDISON: On the Constitutional and Local Effects of Disease of the Suprarenal Capsules. London, 1855.

† LASEGUE: Archives Générales de Médecine, for March, 1856.

The man stated that his legs bent under him, and that he had lost so much strength as to be unable to accomplish the small amount of walking required of him. A blowing sound, evidently anæmic, was heard on auscultating the heart and vessels of the neck. On examining the different organs of respiration and digestion, the liver and spleen, no organic lesion was discovered; and the functions of these organs seemed to be performed with perfect regularity. The urine—analyzed on several occasions—contained neither albumen nor glucose. Except great debility, and the peculiar color of the skin, the only morbid symptoms complained of by the patient were pains resembling those of rheumatism, which he said he felt, from time to time, deepseated in the sides. These pains ceased quickly, returned without any appreciable cause, and were not fixed in any one place. I attached all the more importance to this point, as the patient presented the symptoms characteristic of the bronzed disease. I endeavored to discover whether there were any special symptoms indicating an affection of the suprarenal capsules.

Although the patient, who rose every day, walked about the wards, ate and drank very much like a man in health—except that, like chlorotic women and some patients suffering from cancer, he had a disgust for animal food—was in the full enjoyment of his intellectual powers, and, except anæmia, presented nothing abnormal save the bronzing of the skin, from that symptom I at once formed a very unfavorable prognosis.

It certainly was not my personal experience which led me to this conclusion, for the case was only the second of the kind which I had met with. My first case was that of a young man of twenty-seven. Consequent upon the effects of cold, from discontinuing the use of a flannel belt which he had been in the habit of wearing, he was seized with fever and pains in the lumbar region. He soon fell into a state of great debility, the slightest muscular exertion causing breathlessness and palpitation. The skin looked like that of a mulatto, and, in some places, was quite black: the black hue was seen in old cicatrices, and also on the hands, the nails of which had a variegated appearance, if I may use the expression; that is to say, they were longitudinally streaked with brown bands. The mucous membrane of the lips, gums, and tongue presented similar brown streaks, which contrasted in a remarkable manner with the natural color of the rest of the mouth.

I lost sight of this young man; so that the case did not afford me much instruction. Consequently, my very unfavorable prognosis in the case of our patient in St. Agnes Ward was not based on my personal experience, but on the statements of Dr. Addison and the other English physicians who had spoken of the bronzed disease. I knew that of fifteen patients whose cases had been published, all had died. With such recorded mortality before me, there was, it must be admitted, a strong presumption, that no better fate would attend cases occurring in my practice.

The result too truly realized my fears; and the fatal termination of the case occurred even sooner than I expected. The patient was suddenly seized with a profuse diarrhœa, having as many as eight or ten stools in the twenty-four hours; and the whole body became cold, without, however, there being anything to suggest the existence of cholera in the special appearance of the stools, suppression of urine, or extinction of voice. Delirium supervened: debility made rapid progress: and on the fourth day from the commencement of these symptoms, death took place.

On opening the body, we found no lesion of lungs, heart, or intestines, sufficient to explain the cause of death. The kidneys, which were examined by me and Dr. Brown-Séquard, presented no morbid appearances, except slight hypertrophy, some tubercular deposit, and some fibrous filaments. But, in the *suprarenal capsules*, there were numerous masses of tubercle; and the left capsule (which was considerably enlarged) was almost entirely transformed into that heteromorphous product.

In the apex of one of the lungs, there was a small mass of tubercular deposit of the size of a lentil. Neither the bronchial nor mesenteric glands contained any tubercle.

The blood, which was examined by Professor Charles Robin, presented no other alterations than those of ordinary anæmia.

Here, then, is an individual, who when still a young man, is suddenly attacked by a cachectic disease which is inexplicable by existing pathological knowledge. The bronzed color of the skin, and the disseminated black patches were, during life, characteristic of the disease described by Dr. Addison; and after death, the only noteworthy lesions we found were the morbid changes in the suprarenal capsules which he has described.

If you will now bear in mind the symptoms of which our patient complained, and those which we ourselves observed in him, you will find that they present a picture identical with that which Dr. Addison has drawn, and which I cannot do better than reproduce.

But before proceeding to do so, let me say a few words regarding the suprarenal capsules. So little information have anatomy and physiology afforded in relation to these organs, that they have hardly engaged serious attention. It was observed, that they were susceptible, like other organs, of undergoing serious structural changes, apoplectiform hemorrhages, tuberculous or cancerous transformations; and that cysts might be developed in their substance. In 1837, Dr. Rayer published a considerable number of cases of this description; but he concluded his work with the discouraging avowal that up to the date at which he wrote, the study of the morbid changes of the suprarenal capsules offered so little that was interesting that it might, without detriment, be neglected by pathologists; and that it had not thrown any light upon the functions of these organs.*

Nevertheless, their richness in vessels and nerves, and the constancy of the existence of the suprarenal capsules, gave reason for believing that their functions were of some importance—just as we suppose to be the case with the spleen, which likewise plays an unknown part in the enonomy. But there was not an absolute lack of hypotheses: some physiologists looked on the suprarenal capsules as forming part of the urinopoietic system, and others supposed that there was some connection between them and the genital organs.

From the profusion of bloodvessels in the suprarenal capsules, it has been supposed that they perform an unascertained part in hæmatosis, analogous, probably, to the function of the spleen, or thymus. A remark made during last century, to the effect, that the suprarenal capsules are larger in the negro than in the white race, suggested that they were in some way related to the pigmentary secretion. Bergmann, in his inaugural dissertation, held that they were nervous ganglia. He based this hypothesis upon the anatomical structure of the organs, and on the observations of his father, the celebrated alienist of Hildesheim, who maintained, with Jacobson, that they are often found to have undergone morbid changes in diseases of the brain and spinal marrow.

The recent researches of Dr. Brown-Séquard, and the discovery of Dr. Addison, may accelerate the solution of the question.

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* RAYER: Recherches Anatomico-pathologiques sur les Capsules Surrénales. [*L'Expérience* for 1837.]

Let me state the principal facts enunciated by Brown-Séquard. He says, that the suprarenal capsules are very sensitive. In opposition to the hitherto received opinion, that they are very large in the fœtus, and become atrophied after birth, he holds that they increase in weight and volume from birth up to adult age, so that they cannot be looked upon as the remains of an organ of fœtal life.

Extirpation of both suprarenal capsules kills animals as certainly and as rapidly as extirpation of both kidneys. Brown-Séquard performed the experiment upon sixty animals: he states that eleven hours was the average duration of life after the extirpation. When only one suprarenal capsule was removed, the animal did not survive more than seventeen hours. In none of these cases could death be attributed to hemorrhage, peritonitis, lesion of the kidneys, liver, or other important neighboring organ.

It was observed that when both suprarenal capsules were extirpated, pretty nearly the same series of phenomena was produced; viz., in the first instance, acceleration of the breathing, which soon, however, became slow, jerking, and irregular—increased rapidity in the pulsations of the heart—diminished temperature—and, on the approach of death, nervous phenomena, such as vertigo, convulsions, and coma.

This, gentlemen, leads me to ask whether this profuse diarrhœal flux in our patient was a phenomenon of this description.

Extirpation of only one capsule produces the same symptoms, but more slowly, and not till a period has elapsed during which the animal seems to be recovering: when there are convulsions, they occur on the side on which the extirpation has been performed, the animal turning round like a screw on its own axis, as is observed after section of one of the middle peduncles of the cerebellum: the direction of this rotatory movement is from the side operated on to the other side.

In Paris, an epidemic prevails among rabbits, which is characterized by inflammation of the suprarenal capsules—*an inflammation which produces the same effect as extirpation of these organs.* The blood of rabbits infected with this disease, when injected into other rabbits induces affections similar to those which result from the extirpation and inflammation of the suprarenal capsules.

Wounds of the spinal marrow occasion hyperæmia of the suprarenal capsules, a fact established by Brown-Séquard in 1851: from this results hypertrophy or intense inflammation, under which, in a short time, the rabbits succumb.

From the facts now adduced, we must conclude with the physiologist from whom I have taken all the details, that the suprarenal capsules are organs essential to life, that their extirpation, alteration of structure, or destruction, influence the economy, either by arresting the functions of these organs as hæmatopoietic glands, or by inducing irritation of the nervous system.

Brown-Séquard, after demonstrating the analogies and differences between the results of ablation of the suprarenal capsules, the pigmentary disease which he had so frequently observed in rabbits, and Addison's disease, terminates his memoir by a study of the functions of the suprarenal capsules, concluding that their probable function is to modify a certain substance destined to be transformed into pigment, and to modify it in such a manner as to prevent this transformation from taking place.* In relation to that subject he refers to Professor Vulpian's experiments.†

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* BROWN-SÉQUARD: Journal de Physiologie, 1858.

† VULPIAN: Comptes Rendus de l'Académie des Sciences, 1856.

Having said this much regarding the suprarenal capsules—the bearing of which you perceive—we have now to inquire: *What are the symptoms of Addison's disease?*

The malady begins slowly; and its existence is not at first perceived. The patient has difficulty in fixing with precision the date at which he experienced its earliest symptoms. Its first manifestations are general discomfort, an enfeebling of the physical and moral faculties, and a state of real languor. The arterial pulsations are small and feeble; or the pulse is full, soft, and easily compressed. The appetite is capricious, the patient (like the man in St. Agnes Ward), showing repugnance to animal food, or a diminished appetite. At first, digestion proceeds in a normal manner: at a later stage, this function is disturbed by intractable vomiting. This symptom is accompanied by pain, or at least by a painful sensation, in the epigastric region. The patient wastes away; and yet the most minute examination fails to discover any sign of organic change sufficient to account for the great disturbance of health and extreme anæmia which exist. Finally, there is a state of extreme debility, to which, very properly, attention has been directed by a distinguished hospital physican, Dr. Siredey: this condition is made very evident by dynamometric examination.

However, the characteristic color of the skin, to which the patient himself directs the physician's attention, or which has at least attracted the notice of those about him, supplies a pathognomonic element of diagnosis. This altogether special brown color occupies the entire surface of the body, and is nowhere more marked than on the skin of the face, neck, superior extremities (particularly on the hands, where it forms a remarkable contrast to the usually colorless nails), on the scrotum, axilla, and around the umbilicus. In these situations, the color shows itself in dark patches, varying from a clear brown or bistre to a bronze hue: the mucous membranes present similar appearances. Both the man of St. Agnes Ward and the young man whom I saw in my private consulting-room were thus affected: the one had the lips, and the other had the lips, gums, and tongue marbled with black patches, which, to use a comparison I have already employed, recalled the appearance of the inside of the mouth of certain dogs. Very recently, Dr. Hérard caused notice to be taken of a similar coloration of the lips and gums of a woman whom he showed to the Société Médicale des Hôpitaux.* It is not the mucous membranes only which show this remarkable coloration: Dr. Addison states, that in one subject, he observed similar brown patches on the peritoneum.

These patches are constituted by deposits of pigment irregularly accumulated in greater masses in different places. Strips of skin taken from the hands of the dead body of an individual who died of Addison's disease (and whose case is published by M. Second-Ferréol), presented, under the microscope, the characters of the skin of the negro, showing a profusion of pigmentary granules.

Alongside the patches of a more or less decided brown color, Dr. Addison remarked, that others were visible which were not only lighter in shade, but were even of a dull white color, spots of true vitiligo, mingling with the brown patches, just as if the pigmentary matter had deposited itself in some places and had neglected others.

As the disease advances, the bronzed color becomes more decided, while, at the same time, the general symptoms assume increased intensity. Day by day, the debility increases; and the individual, having become wasted to the last degree, dies from exhaustion, unless he be abruptly carried off, like our patient, by an acute intercurrent affection.

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* HÉRARD: Bulletins et Mémoires de la Société Médicale des Hôpitaux de Paris, 1867.

All these general symptoms resolve themselves into those of extreme anæmia. Professor Charles Robin on examining the blood of our patient (a small quantity having been taken by a cupping-glass) found, that it presented nothing abnormal, except a diminution in the red globules, such as is always observed in anæmic subjects.

In two patients, however, observed by Dr. Siredey, the red globules were as numerous as in health, and presented no modification either in form or volume. The number of the white globules was diminished. There was an absence of pigmentary matter. In relation to this latter point, Dr. Siredey justly remarks that it is one of curious inquiry.

Dr. Siredey points out that the two patients, who were absolutely cachectic, did not present the vascular blowing sounds which belong to anæmia, a fact which led him to conclude that the bronzed disease, classed by some among the anæmic affections, must be a peculiar species.*

The same observer has likewise pointed out a fact which demonstrates depression of vital power—that fact being a decrease of temperature. In three patients whom he observed, the temperature, when fasting, was always below 37° C., the normal standard: in one subject, it was 35.7°, in another, 35.4°, and in a third 36°. These phenomena are interesting; they have not often been observed, and demand further investigation.

The urine of our patient, which was examined by M. Robin, contained some pus-globules, but they were so few in number as to possess no pathological importance. In the urine of his patients, Dr. Siredey found traces of glucose and cyanurine, but no appearance of albumen.

Dr. Jaccoud, in a work of remarkable merit, like every work of that distinguished physician, from an analysis of 127 cases of the bronzed disease, thus characterizes it: "An asthenia which goes on increasing up to death, a melanodermia presenting special characters, gastric disturbance, pains in the loins and abdomen—such are the four groups of symptoms which essentially constitute the symptomatology of Addison's disease. The two first are constant, and may continue during the whole course of the disease: the others occur sufficiently often to be characteristic, and to be useful aids to diagnosis."† The gastric derangement consists in obstinate and sometimes quite intractable vomiting: this symptom was present in 74 of the 127 cases analyzed. It was absent in my two patients, and in the three patients of Dr. Siredey. Along with the symptoms now enumerated, Dr. Jaccoud groups others which like them are dependent on perturbation of the nervous system, viz., headache, convulsions, vertigo, delirium, and coma. These symptoms are by no means of frequent occurrence.

Connected with animal life, Dr. Jaccoud observed palpitations and dyspnœa: in three cases, they were expressly pointed out, and in many others, no doubt, existed, but escaped notice. Dr. Jaccoud called attention to the fact, that while the asthenia was so serious as to lead to death, it was not accompanied, as might have been expected, by loss of flesh, albuminuria, leucocytosis, hemorrhages, vascular blowing sounds, and diarrhœa, as are met with in persons affected with cachectic asthenia. When there is loss of flesh, it is attributable to pulmonary phthisis: albuminuria is very unusual, and, as you know, was absent in our patients, as it was likewise in the patients of Dr. Siredey. Leucocytosis is exceedingly seldom met with, and is due to a complication and not to Addison's disease. Hemorrhages were observed in only one patient; and he had disease of the liver. Dr. Siredey has not found vascular blowing sounds except in patients who had become anæmic from anterior or concomitant affections. Diarrhœa, which as you saw, terminated the life of one of our patients, is attributed, in the few cases in which it occurred, to complications independent of the bronzed disease.

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* SIREDEY: Bulletins et Mémoires de la Société Médicale des Hôpitaux, p. 355. Paris, 1867.

† JACCOUD: Dictionnaire de Médecine et de Chirurgie Pratiques, t. v, Article Bronzée (Maladie).

The *prognosis* is shown to be of the most unfavorable possible character by the cases related by Addison and other physicians, by that of our patient, and also by two other cases which I have recently had to treat. When the disease, after pursuing a chronic course, becomes acute, death is the inevitable issue.

This is also what happens in leucæmia, as in other kinds of extreme anæmia of very long duration, and which neither originate in great loss of blood, bad or insufficient food, nor in poisoning by marsh miasmata. Persons affected by the bronzed disease have never been known to recover.

At the *autopsy*, there are found, almost invariably, lesions of the suprarenal capsules; and, in most cases, tubercle, cancer, and fibrous products. It is a remarkable circumstance, that Addison, and those who after him reported cases of the disease which he was the first to describe, never met with the hemorrhages of the capsules, and consecutive capsular dilatation, as pointed out by Rayer in his memoir to which I have alluded. Sometimes, cancerous or tuberculous deposits were found both in the capsules and in other viscera, while at other times, the capsules only were affected. The kidneys were generally healthy, and almost free from any modification of structure.

In all cases of the bronzed disease, the suprarenal capsules are in a morbid condition; but the converse is not a correct statement, for there may be lesions of the suprarenal capsules without bronzed disease. Addison explicitly says so, quoting, with that good faith so conspicuous in his work, a case in which, on making an autopsy, he found small multiple cancers and cancerous products in the suprarenal capsules of a patient who had never had in the least degree the special tint of the skin. Rayer also cites cases which are analogous in this sense, that the suprarenal capsules were found more or less completely destroyed in persons who had never presented symptoms characteristic of Addison's disease.

Here, then, gentlemen, to the already extensive catalogue of chronic diseases, we have to add a new and entirely distinct species of anæmia. Need I say, how great is the service which Addison has rendered to our science and art by establishing this important distinction!

When I began my medical studies forty years ago, the subject of dropsy was an inextricable chaos. Fortunately for practice, this confusion has been unravelled by the beautiful researches of Professor Bouillaud, my colleague, and Dr. Bright. The researches of Virchow and Bennett into the subject of leucæmia, and of Addison into the bronzed disease, have thrown a new light upon the not less complex question of anæmia. Till then, the treatment of anæmical affections was deplorably empirical, it being impossible to distinguish cases in which we might usefully interfere from those in which no treatment could be of any use. In dealing with the former, we might make lucky hits; but in treating the latter, we incurred the risk of seriously complicating already serious cases by employing unsuitable measures.

Nevertheless, gentlemen, the diagnosis of the disease now under our consideration is far from being devoid of obscurity. The special color of the skin, which constitutes its pathognomonic character, does not generally make its appearance till an advanced stage of the disease. Its beginning is announced by a series of phenomena which belong to it in common with other forms of anæmia.

We must beware of mistaking for Addison's disease other cachexiæ in the course of which the skin assumes a dirty hue, considerably resembling that which is observed in the *bronzed disease.* Thus, in pregnant women, and in individuals with profuse and protracted suppurations, the skin acquires a dirty or brown appearance; but this coloration is not distributed in the same localities as in Addison's disease, nor is there ever that bluish color of the mucous membrane of which I spoke.

The remarks which I made on the prognosis render it unnecessary to enlarge on the *treatment.* All the means which have been tried have failed to prevent a fatal issue. Having no specific remedy, we are obliged to direct our measures against the symptoms of anæmia: ferruginous medicines, preparations of cinchona, and a tonic regimen, which you have seen me employ in the case of our patient, are indicated.

My friend, Dr. Duclos, lays down similar therapeutic rules.* My talented colleague of Tours is of opinion that the *bronzed disease* is dependent on a lesion of the suprarenal capsules; that the symptoms, the progressive debility so characteristic, and the fatal issue of Addison's disease, are consequences of a general poisoning of the economy by the pigmentary matter, which, in consequence of the perturbation of the function of the suprarenal capsules, is not destroyed as it is under normal conditions. Dr. Duclos founds his opinion, which he discusses with his usual ability, upon the attentive study of the case which he observed, and the cases recorded by authors: he also founds his opinion upon the results of the physiological experiments of Brown-Séquard and Vulpian—experiments which he looks upon as in accord with the information he had obtained from clinical observation.

I am well aware, and it is my duty to tell you, that this view of the disease has been met by many objections. Dr. Martineau in his inaugural thesis on Addison's disease,† while he regards it as a well-defined morbid entity, disputes the part assigned to the suprarenal capsules. He rests his opinion on cases, derived from different sources, in which the disease had existed, while no trace of lesion in the suprarenal capsules had been found on examination after death.

Dr. Jaccoud has enunciated the following theory of the pathological chain of symptoms in Addison's disease. The rich supply of nerves which the suprarenal capsules possess, and the presence of ganglionic corpuscles in their medullary substance, justify our regarding them as a nervous apparatus dependent upon the abdominal sympathetic system. But the semilunar ganglia constitute the centre of innervation, and consequently of reflex action of the suprarenal plexus: all abnormal excitation then of the one will affect the other by reflex action producing the visceral disturbance which belongs to Addison's disease. On the other hand, the great sympathetic holds under its influence the vaso-motory system: this system presides over calorification and pigmentary secretion. Now, one cannot understand reflex disturbance of the vaso-motors, on the one hand, producing that reduction of temperature pointed out by some observers (and recently by Dr. Siredey); and, on the other hand, that exaggerated secretion of pigment which is the cause of the bronzing. To explain the examples of incontestable lesion of the capsules without bronzing of the skin, and bronzing without lesion of the capsules, Dr. Jaccoud remarks, in relation to the first order of facts, that visceral disturbances are produced more easily and more quickly than the deposit of pigmentary matter, so that they may have been of sufficient intensity to cause death before it was possible for the bronzed color to have been produced: in respect of the second class of facts, he adds, that other alterations of the nervous plexuses might excite the abnormal secretion of pigment, and that thus we might have bronzing without lesion of the suprarenal capsules. Moreover, remarks Dr. Jaccoud, bronzing without progressive asthenia and without visceral disturbance is not Addison's disease.

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* DUCLOS: Bulletin Général de Thérapeutique, for 1863.

† MARTINEAU (L.): De la Maladie d'Addison: avec trois planches coloriées. Thèse de Paris, 23 Décembre, 1863.

Without criticism, I leave this ingenious pathogenesis in your hands.

Professor Sée regards Addison's disease as a cachexia, the seat of which is probably in the hæmatopoietic organs; and which is generally of a tuberculous or cancerous character. The nervous phenomena, and the very great prostration which accompany this cachexia, seem to be explained by the nervous texture of the suprarenal capsules. When there is a lesion of these organs, it is supposed that the alteration is nervous; when there is an absence of material lesion, we are obliged to fall back on neurosis of the capsular nerves. According to Professor Sée, it is most rational to suppose that there is disturbance of function in the vascular glands, particularly in the suprarenal capsules.*

My personal experience is too limited to allow me to come to an absolute opinion upon this interesting question; and I doubt whether others are really better prepared to speak in a fashion more categorical and positive. However, from what I have seen and read, I rather incline—as I have already said in this lecture—to the theory held by Dr. Duclos, and to believe that there is a relation between the lesions of the suprarenal capsules and the bronzed disease.

LECTURE XC.

LEUCOCYTHÆMIA.

A Disease characterized by great and progressive augmentation in the White Globules, or Globulines of the Blood.—In Leucæmia, there is Enlargement of the Spleen, Lymphatic Glands, and Liver.—Etiology entirely unknown.—The only Essential Symptom of the Disease is the Presence in the Blood of a greater number of Leucocytes and Globulines.—Anæmia and Cachexia are consequences of Leucæmia.—Preparations of Cinchona, which have so manifest an action on Engorgements of the Spleen caused by Marsh Miasmata, have no effect on Engorgements of the Spleen in Leucæmia.

GENTLEMEN: Chemistry and the microscope are often of very doubtful utility in their applications to pathology, and still more in their applications to therapeutics. Notwithstanding the progress which is being made day by day, notwithstanding the efforts made by eminent men who specially occupy themselves with these means of investigation, we too frequently discover their worthlessness at the bedside of the patient. Still, let me at once admit, that, under certain circumstances, they have rendered, do render, and will continue to render, signal service; in some cases, indeed, it is absolutely necessary to employ them as means of diagnosis. Was it not by these methods of investigation demonstrating the presence of sugar in the urine, that glucosuria became better known to our generation than it was in former times? And without employing them, when this disease presents itself to our notice, how should we be able to attain a precise knowledge of the case? Similar remarks are applicable to albuminuria: it is chemistry which enables us at any moment to seek for and find albumen.

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* SÉE (G.): Du Sang, et des Anémies. Paris, 1866.

As much may be said of the utility of the microscope. Does it not give us, on very many occasions, information regarding the normal and pathological anatomy of the different tissues of the body? What services is it not destined to render to the study of pathological anatomy, which it has started on a career of true progress! Therefore, while we avoid falling into the excesses of those who believe that everything in pathology is to be achieved by the aid of chemistry and the microscope, who would allow them to dominate medicine, who through them would rationalize therapeutics, it is important that we study them, as means of clinical investigation, so as to be able to utilize them on fitting occasions.

It is to the microscope, and to the happy application made of it by Hughes Bennett and Virchow in the diagnosis of leucocythæmia, that we owe the power we now possess of distinguishing hypertrophy of the spleen which is characteristic of this malady, from hypertrophy of the spleen symptomatic of paludal poisoning, of the paludal diathesis. We required to have recourse to the microscope to establish a precise diagnosis in the case of a man who occupied bed 9, St. Agnes Ward. I hesitated some time before I formed a settled opinion as to the nature of his case. Some of you thought, with one of my most skilful colleagues, that, possibly, this man had renal disease; others, looking to the fact that the patient came from a district where intermittent fever was endemic, believed (although the patient affirmed that he had never had intermittent fever) that there was hypertrophy of the spleen consecutive on paludal poisoning; but the microscope relieved us from the uncertainties of our diagnosis, by demonstrating the existence of that alteration of the blood which is characteristic of leucocythæmia.

When healthy blood is examined by the aid of the microscope, we observe (besides the red globules, which are seen lying one above another like a pile of five-franc pieces), other globules which are white, larger in diameter and much less numerous than the red. We also find isolated nuclei, in a proportion which may be described as insignificant.

In certain physiological conditions, such as during digestion, menstruation, and pregnancy, and also, in certain pathological states, as in inflammatory diseases, in typhoid fever, puerperal fever, cancer, and phthisis, when these maladies are far advanced, there is an increase in the number of the white globules of the blood; but this numerical increase, essentially temporary in the physiological state, essentially accidental in pregnancy, being in all these instances subordinate to causes which are not persistent, does not constitute leucocythæmia, any more than diabetes is constituted by the accidental presence of sugar in the arteries and veins, in the renal arteries, and sometimes even in the urine, during digestion.

This increase of white globules in the diseases I have mentioned, no more constitutes leucocythæmia, than the presence of albumen in the urine in the first stage of cholera, in convulsions, and in inflammatory sore throat, constitutes Bright's disease.

To constitute leucocythæmia, that is to say, the special disease, the dyscrasia, which, from its very beginning, makes incessant progress leading to inevitable death, it is necessary that the excess in the proportion of white globules be greater than in any of the circumstances I have mentioned: the maximum proportion fixed by authors who have treated this subject is 1 to 20.

According to Moleschott, in the normal state of the economy the proportion of white to red globules is 1 in 346. In leucocythæmia, the proportion is at least 1 in 20: between this minimum and the report of 1 to 1 noted by Dr. E. Vidal, my colleague in the hospitals,* cases have been met with in which the proportion was 1 to 19, 1 to 12, 1 to 7, and 2 to 3. But according to Virchow, leucocythæmia does not exist merely because there is a certain increase of the white globules: he holds, that there must also be a simultaneous diminution of the red globules, a substitution of the former for the latter—this substitution being often so great that the blood assumes a more or less white color, as if (says the German author) there was true albinism.†

When we wish to examine the blood of an individual whom we suspect to be stricken by leucocythæmia—and I repeat, it is only by a microscopical examination of the blood that we can arrive at a precise diagnosis—we prick with a needle the end of one of the patient's fingers. The blood, on exuding, has a troubled appearance and a yellowish-red color: on coagulating, it assumes a deeper brown hue. In a case published by Vogel, blood drawn from a vein was in two separate portions. The first was defibrinated: after four hours, a whitish cream was seen floating on the surface, and after twenty-four hours, the defibrinated portion of blood divided into two layers, the upper being of milky whiteness resembling pus, and the lower being of a reddish-brown color. The second portion coagulated like healthy blood: the clot was covered by a whitish granular layer formed by the aggregation of the white globules: the serum was abundant, clear, and limpid. This experiment of Vogel recalls to our recollection the process of separation pointed out in 1844 by Donné.‡ This process consists in defibrinating the blood, and allowing it to settle till the white globules have separated: the white globules, being less dense than the red, float, while the red are precipitated. By proceeding in this way, results are obtained, which though not absolute, at least admit of easy comparison. Dr. E. Vidal has turned to account this method, which is applicable to a very small quantity of blood, for estimating the relative proportions of white globules which exist in a case of leucocythæmia.§

The blood, having been first defibrinated, was poured into a graduated tube. Forty-eight hours elapsed before complete separation had taken place. The mass was then seen to be divided into three well-marked layers differing in thickness.

First, there was an upper layer formed by the serum, which was limpid, lemon-colored, and normal in appearance.

There was then formed a middle layer of grizzly-yellow, slightly inclined to green, and of a color analogous to that of pus, constituted by the aggregation of white globules.

Lastly, the under layer was composed of red globules of the color of wine-lees, marbled towards its upper part, and exhibiting some whitish particles adherent to the sides of the glass.

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* VIDAL (E.): De la Leucocythémie Splénique. [*Gazette Hebdom. de Médecine*, 1856.]

† VIRCHOW (R.): Gesammelte Abhandlungen zur Wissenschaftl. Medezin: Frankfurt, 1856.

‡ DONNÉ: Cours de Microscopie.

§ VIDAL: Bulletins de la Société Anatomique, 1857.

In a first examination, the second layer (white globules) was in relation to the third (red globules) in the proportion of 1 to 2.14. Six months later, a renewed examination was made, when it was found that the layer of white was greater than the layer of red globules, their relative height being in the proportion of 1.25 to 1.

On counting the globules in the field of the microscope, it was found that there was a nearly equal number of white and red, the latter, however, being rather more numerous. To appreciate properly the apparent difference between the results of the process of separation and the numerical estimate, we must bear in mind that the leucocytes are more voluminous than the red globules above which they float; they are less piled up on one another, and there is interposed between them a certain quantity of serum.

Considering that this alteration of the blood, this substitution of leucocytes for red globules, is coincident with the alteration of the solids which we are now about to study, and granting that the disease began with special lesions of the spleen and other vascular glands, lesions which show themselves by the organs becoming hypertrophied before the blood has undergone the characteristic change, Bennett and Virchow have enunciated an essentially different theory of the nature of leucocythæmia.

According to Virchow, the spleen and lymphatic glands are charged with destroying, in a certain manner, the red globules. The more these organs are hypertrophied, the greater, he says, is their activity; and consequently, the number of white globules will be great, and the number of red globules will be diminished.*

Bennett holds that the spleen and lymphatic glands are charged with the formation of the white globules; but he does not admit that these organs destroy red globules. He believes that red globules are really white globules modified and colored in other parts of the circulatory system. The difference between the two theories is, therefore, at once apparent. According to Bennett, there is increased functional activity of the spleen resulting from its hypertrophy, leading to the formation of a number of white globules, which ultimately circulate in so great a quantity that the transformation of all of them into red globules is impossible. According to this theory, there is no substitution of white for red globules, but only an increase in the proportion of the white globules.

Dr. E. Vidal and Professor Magnus Huss discuss both of these theories and consider neither satisfactory, because, on the one hand, they are based on physiological views of a merely theoretical character being demonstrated to be truths, and on the other, because, if leucocythæmia be exclusively dependent on hypertrophy of the spleen, why is it not associated with the hypertrophies consequent upon intermittent fevers, and in some of which the spleen acquires a volume at least as great as in any case of leucocythæmia? You may recollect the young woman who had contracted a quartan fever at Guadaloupe, and came into our wards with a greatly enlarged spleen. Her blood did not contain an excess of leucocytes. It is evident, then, that there must exist, besides the hypertrophy, a hitherto undiscovered special structural and functional lesion of the spleen.

The same objection may be made to the lymphatic leucocythæmia of Virchow. Though this kind of leucocythæmia is characterized, according to the Berlin professor, by hypertrophy of the lymphatic glands, the spleen preserving its normal size and structure, it differs from splenic leucocythæmia, not only in the white globules predominating in the blood, but also in the predominance of globulines identical with those of lymph. Well then, if in this kind of leucocythæmia, hypertrophy of the lymphatic glands were the sole cause of the disease, how are we to explain the fact, that it does not always occur in the many cases in which we find glandular engorgement and hypertrophy without leucocythæmia? We had lately an example of this in a tuberculous patient who died in our wards with great enlargement of the cervical glands. His blood, examined by the aid of the microscope, presented none of the morbid alterations which belong to the disease we are now considering. Moreover, in an early lecture, I will lay before you several cases of generalized glandular hypertrophy, in which neither an excess of white globules nor of globulines had ever been detected by microscopic observation.

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* VIRCHOW: "La Pathologie Cellulaire basée sur l'Etude Physiologique et Pathologique des tissus;" traduit de l'allemand par Paul Picard. Paris, 1861.

To sum up: we may say with Vidal and Magnus Huss that, while we admit leucocythæmia to be a malady *sui generis*, we possess no satisfactory data whereby to determine its essential nature; and that the close relation which may exist between morbid change of the spleen or glands and morbid change of the blood will remain unknown, so long as the formation of the blood, and the functions of the spleen and glands without excretory ducts (such as the thyroid and thymus) remain secrets.

In pointing out the alterations of the blood characteristic of leucocythæmia, I have noticed the most important part of the anatomical history of this cachexia. I have, however, still to speak of the state in which the blood is found on opening the dead body. Its color varies from brick-red to a deep-brown or chocolate hue. Sometimes, it forms clots which do not adhere to the walls of the vessels which they fill to distension; but there is never any alteration in the walls of the vessels. The clots are mingled with yellowish or grayish coagula, which at a first glance may be mistaken for concrete pus. Sometimes, we find the blood fluid, pale, and reddish-yellow, having the appearance of the muddy blood of the spleen, and containing a large quantity of white globules.

The most remarkable organic lesions are found in the spleen, liver, and lymphatic glands.

The increase in the volume of the spleen, which in the great majority of cases of leucocythæmia arrests the attention of the physician in the living patient, exists nearly always, or, I may say, always, in the dead body.

The weight of this organ reaches as much as six [French] pounds; and its dimensions, which generally vary between 30 and 32 centimetres in length, between 16 and 18 in breadth, have been as much as 41 by 20 centimetres, with a thickness of 7 centimetres. Its form is an enlargement of the natural form: in different cases, there is a great diversity in the appearance and consistence of its parenchyma. In the majority of cases, the tissue is hard, frangible, of a uniform deep-brown or reddish color, and exhibiting, on section, a lustrous aspect: in some cases, the color tends towards yellow, presenting red and yellow layers, which give it a marbled appearance. In five autopsies, included in the history of the cases of leucocythæmia which form the basis of Dr. Vidal's excellent monograph, the spleen contained one or more deposits of whitish or yellowish-white matter resembling the fibrinous deposits sometimes met with in its interior in subjects having disease of the heart.* In two cases, it was riddled with small whitish points. Its capsule was thickened, opaque throughout, and adherent by plastic exudation to the diaphragm and peritoneum.

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* VIDAL (E.): De la Leucocythémie Splénique. [*Gazette Hebdom. de Médecine*, 1856.]

Examined under the microscope, its tissue presents important changes; viz., increase in the number and volume of the normal elements, and the substance between the cells of the pulp is more abundant and more condensed than in the healthy state. This modification of texture has been described by Virchow under the name of *hyperplasia with induration*. Vidal and Luys have observed great hypertrophy of the Malpighian tufts. These tufts filled with cells having several nuclei, and with free nuclei, are tripled or quadrupled in size, and assume a whitish appearance, marbling in a certain manner the reddish-brown of the parenchyma of the organ.

The liver, without being altered in structure, is increased in volume. In some cases, it attains three times its normal bulk, and weighs from four to six kilogrammes.

The lymphatic glands, which are often hypertrophied, never present, even in lymphatic leucocythæmia, more than simple augmentation of their normal elements.

Gentlemen, let me call your attention to an anatomical fact recorded by Dr. Lancereaux. While it tends to confirm the remarks of Magnus Huss, E. Vidal, and Virchow, it also shows us how numerous the white globules may be in the capillaries, particularly in those of the brain. The case occurred in M. Marotte's service at the Hôpital de la Pitié. The patient was thirty-two years of age, and had presented the symptoms of leucocythæmia. The liver and spleen were both much enlarged; and an examination of the blood showed that the white were very numerous, and also more numerous than the red globules. At the autopsy, it was found that the spleen extended from the hypochondrium to the symphysis of the pubes. It had a pretty uniform brownish color, which became yellow or bright red on exposure to the air: when cut, there flowed from it a very thick chocolate-colored fluid, composed to a great extent of white globules. A tear of the organ presented a granulated appearance, and enabled one to see the hypertrophied Malpighian bodies, even by the naked eye. The cerebral sinuses and their affluent veins were filled with brownish clots. The surface of the brain presented a fine injection of the veins of the pia mater, a fact to which I wish to direct your special attention. It had the appearance of a mercurial, or, still more, of a purulent injection. The white matter which constituted this capillary injection was entirely composed of white globules. I am not aware, gentlemen, that a similar injection of the capillaries of the brain had been observed in any previous case. The same kind of injection may probably be met with in other organs rich in capillary vessels, such as the lungs and glands.

It has been correctly remarked by Professor Sée, that enlargement of the spleen and lymphatic glands is not sufficient to produce leucocythæmia: he holds, that to produce leucocythæma there must be *hyperplasia* of the proper tissue of the organ, that is to say, an augmentation of its active part. He says: "If the spleen or lymphatic glands undergo a morbid change, without there being hyperplasia, no increase of white globules takes place: we meet with fever patients having enormous enlargement of the spleen, without any trace of leucocythæmia: these engorgements are sanguineous infarctus, or very formidable lesions of the tissue of the spleen." The same able physician also observes, that "leucocythæmia always supposes the formation of new glandular tissue, or of new elements in the normal glands: in the adult, leucocythæmia is *almost* always of splenic origin, while in the child, on the contrary, it is of glandular origin."*

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* SÉE (G.): Du Sang et des Anémies, p. 280. Paris, 1866.

Professor Sée adds that, when there is no primitive hyperplasia of the lymphatic glands, new lymphatic glands make their appearance, being formed everywhere in the pleura, liver, kidneys, and intestines. There is, so to speak, proliferation of the adenoid tissue, the lymphatic elements being increased in number and volume; this is the conclusion derived from the observations of Friedreich, Leudet, Botcher, and Billroth.

Such are the principal anatomical characters of a disease of which we received the first accounts almost simultaneously from Germany and England, where they were published in 1845, at an interval of only a few days, by Virchow[*] of Berlin and Bennett of Edinburgh.[†] In France, however, so far back as 1836, the malady was observed by our able colleague, Dr. Barth, as is mentioned by Dr. Vidal in his work, in the chapter devoted to the history of the subject. In 1852, Dr. Leudet published a case of leucocythæmia, in which the diagnosis was made after death;[‡] and in the following year, Dr. Charcot placed another case on record.[§] Dr. Vidal's monograph, which I have frequently referred to, is based on thirty-two cases. To these, other cases might be added—particularly, the one related in the work of Professor Magnus Huss of Stockholm, the case now in our clinical wards, and the case of a child fifteen months old, admitted to our nursery ward in 1862, of which I am about to speak.

Dr. Vidal has remarked that leucocythæmia must be very rare in early infancy, as he had not found one case of its occurrence at that period of life, although he had made himself acquainted with most of the works which had appeared on the disease up to 1856. Of the thirty-two patients whose cases he analyzes, the youngest was 13½ years of age. This is the proper place to narrate the case of the child of fifteen months, to which I alluded a minute ago. In that case, there was great hypertrophy of the spleen; and microscopic examination showed us numerous large white globules in the blood. To give to this case its full value, let me add, that Dr. Vidal was so good as to examine the blood of our little patient; and, on two occasions, he found that it presented all the characteristics of splenic leucocythæmia. Here are the details of this case.

A child, aged 15 months, suckled by its mother, was admitted, on the 16th February, 1862, to St. Bernard Ward. It seemed to have been ill for a long time: the face was somewhat puffy: the mother stated, that for three weeks the child had had diarrhœa and vomiting: she likewise mentioned that it had had, for eight or ten days, every afternoon, an attack of fever with slight shivering. There were no signs of pulmonary phthisis nor of rickets. The extremities were thin and wasted: and the general wasting was rendered more obvious by contrast with the enormous size of the abdomen. By palpation, a very considerable enlargement of the liver was detected; and in the left hypochondrium, I found great tumefaction of the spleen, that organ descending obliquely as low down as the spine of the ilium, and its inner margin reaching nearly to the umbilicus. There was a small amount of ascites. As I have already stated, the little patient had had diarrhœa for some days: the mother had remarked that occasionally there was blood in the stools, and this I also noticed several times. Here, let me remark, that this child was born at the Hospice des Cliniques, had never been out of Paris, and, in all probability, had never been subjected to the causes which engender paludal fever. Microscopic examination of the blood revealed the presence of a great number of white globules. There was no room for doubt, therefore, as to the nature of the disease: it was certainly a case of leucocythæmia. During the month the child remained in our wards, the diarrhœa and vomiting were kept in check by preparations of chalk and bismuth. The ammonio-citrate of iron seemed to impart a certain amount of color and firmness to the flesh. However, from time to time, the sanguinolent diarrhœa reappeared. Crude quinine, in doses of 15 centigrammes, did not always succeed in cutting short the paroxysms of fever, which returned nearly every day, sometimes with and sometimes without shivering. There was no diminution in the volume of the spleen; and when the mother wished, contrary to my counsels, to leave the hospital, it still extended as low down as the anterior iliac spine; and, on palpation, presented as great a degree of hardness as before.*

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[*] VIRCHOW: Froriep's Notizen, No. 780.

[†] BENNETT (John Hughes): Edinburgh Medical and Surgical Journal, for October, 1845.

[‡] LEUDET: Bulletins de la Société Anatomique, 1852.

[§] CHARCOT: Comptes Rendus des Séances de la Société de Biologie, p. 44, 1re Série, t. v, for the year 1853.

Gentlemen, I have already mentioned incidentally the slight influence which quinine has upon engorgement of the spleen in leucocythæmia. Let me now remind you of the great rapidity with which these engorgements disappear if produced by paludal poisoning, when treated by cinchona in large doses according to Sydenham's method.

At the same time that the child of whom I have been speaking was in our wards, you may remember to have seen, in bed 13 St. Bernard Ward, a young woman of 18, who came in with quartan fever and great engorgement of the spleen. Every time that I gave her cinchona, you saw that intense fever was set up for some hours, while at the same time, we could perceive that there was enlargement of the spleen. But from the following day, the fever entirely ceased, and the progressive diminution of the organ was clearly demonstrated by palpation and percussion. This diminution continued for six, seven, eight, or ten days, that is to say, during the whole of the apyrexial period; but as soon as the fever returned, the spleen was observed to swell anew. This almost experimental observation, which I repeated several times during the patient's residence in hospital, affords additional confirmation of two already known facts, viz., that cinchona has a special action on engorgements of the spleen of paludal origin, and that this result is not obtained without a temporary increase of the fever, accompanied, probably, by hyperæmia of the spleen.

No definite information has as yet been obtained as to the causes which give rise to leucocythæmia. In Dr. Vidal's thirty-two cases, it occurred twice as frequently in men as in women. The age of the patients has varied between fifteen months and sixty-nine years; but the affection is most common among adults. The ill fed and badly lodged, the poor, those addicted to excess in alcoholic drinks, and persons placed under unfavorable hygienical conditions, are those who have chiefly paid tribute to this sad disease.

On inquiring into the antecedents of the patients, we find that four women dated the beginning of their malady from the period of their last pregnancy. Others had previously complained of rheumatism. Some patients had had intermittent fever; but in these cases, no necessary relation could be established between paludal poisoning and the leucocythæmia. For even supposing, with Dr. Magnus Huss, that the splenic engorgement of paludal poisoning was in these exceptional cases an organic cause determining leucocythæmia, we must admit the small importance of such an etiology, when we observe every day splenic engorgements of various origin, which are not accompanied by any augmentation in the number of the white globules of the blood.

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* VIDAL (E.): De la Leucocythémie Splénique. [*Gazette Hebdomadaire de Médecine*, 1856.]

However, as a symptom, engorgement of the spleen has much importance: indeed, the circumstance upon which our patient laid most stress was the greatly swollen state of that organ. It is the phenomenon which when present, enables the physician to make a decisive diagnosis between splenic and lymphatic leucocythæmia, because it is absent in the latter. Enlargement of the spleen is often also the fact to which patients attach most importance: this enlargement may, as I have said, attain great proportions. The abdomen bulges out in the hypochondrium and left side, in consequence of the enlarged spleen invading a great part of the abdominal cavity. The skin in that region is ridged by distended veins. The limits of the organ are easily ascertained by palpation: in our case, we could ascertain its limits by inspection. The tumor, in its upper portion, is fixed, and moves very slightly when the patient moves: it descends somewhat when the patient stands. Palpation and percussion occasion more or less pain; and sometimes pain spontaneously supervenes of so acute a character as to necessitate medical intervention. When patients are simply standing, they only experience a feeling of weight which is increased by walking or by the performance of any work. This feeling, being increased by the pressure of the clothes, obliges patients to slacken the waist-band.

Enlargement of the liver often coexists with hypertrophy of the spleen: but it is chiefly in the second period of the disease that enlargement of the liver is met with. It is accompanied by pain. Hypertrophy of the spleen and liver may give rise to abdominal effusion, and anasarca; but the latter serous effusion may occur at an advanced period of the disease under the influence of the cachexia.

Hypertrophy of the spleen or lymphatic glands, and the pathognomonic alteration of the blood—particularly the latter—are the symptoms which really belong to leucocythæmia: the other symptoms are not peculiar to it, and occur in all cachectic diseases.

In the beginning of the disease, there is debility which sometimes makes rapid progress. There is, at the same time, loss of flesh, paleness of the skin, and all the symptoms of anæmia, such as palpitation, buzzing in the ears, dimness of vision, headache, and sometimes a tendency to faint. Some patients complain of neuralgic pains. The temper becomes irritable, melancholy, and morose. In the latter days of the disease, a tranquil delirium supervenes, which continues till the death of the patient.

Digestion is generally well performed, even in the last stage of the disease; and though at the very end, diarrhœa is the most constant symptom, the stools generally remain regular, except in some individuals in whom there is an alternation of confinement and looseness of the bowels. However, in the case of a Spanish merchant, who consulted me in 1861, the malady began with disturbance of digestion: two or three hours after eating, he experienced acute pain in the stomach. He drank, without benefit, mineral waters of the most varied kinds, for the cure of this gastralgia: in vain, he changed the hours of his meals and the nature of his food—the digestion remained unimproved. It was not till three years had elapsed, that glandular enlargement made its appearance: treatment by preparations of iodine proved of no avail. The abdomen became distended with gas; and cachexia began to show itself. It was in these circumstances that the patient applied to me. I found that he had great enlargement of the spleen and liver, as well as of the lymphatic glands of the neck, axilla, and groin. On percussing the abdomen, slight ascites was discovered: the countenance was pale, thirst was urgent, and urine scanty. The pulse was quick, particularly at night. My accomplished colleague, M. Robin, on examining the blood at my request, found that the white globules were in the proportion of from 20 to 25 in 300, in place of 1 in about 300, the usual proportion in normal blood. The result of this examination, therefore, fully confirmed my diagnosis.

The embarrassed respiration, which is observed in the majority of patients from the beginning of the disease, and which is increased in some persons after dinner, by walking, or by movements of the body, is connected with anæmia, and is also probably dependent on the mechanical obstacle to the free play of the respiratory apparatus caused by the enlarged spleen which pushes up the diaphragm into the cavity of the chest.

This dyspnœa, which increases as the malady progresses, may, in the last stage, become orthopnœa, although no pulmonary lesion can be found at the autopsy. It is sometimes accompanied by an occasional short cough, which is generally dry, though followed in some cases by slight mucous expectoration.

The pulse is weak and compressible; and does not become rapid till hectic fever is lighted up. In the cases in which this fever showed itself at an early period of the disease, it was very different from the paroxysms of intermittent fever. The febrile movement, however, often commenced with rigors, and was followed by profuse sweating, particularly at night, obliging the patient to change his linen: the attacks recurred very irregularly, and were very transitory: they generally came on towards evening, like paroxysms of hectic fever, and unlike paroxysms of paludal fever, which generally declare themselves in the morning or at noon.

I have mentioned anasarca, and have to a great extent attributed its occurrence to the cachectic state. Serous effusions into the cavities of the abdomen and pleura, œdema of the cellular tissue and lungs, are generally observed in the last stage of leucocythæmia; and, in some cases, œdema has been seen to appear and disappear at different times.

It is usual to observe—as was observed in our patient—a tendency to hemorrhages. The most common are nasal hemorrhages, after which come intestinal, inguinal, and subcutaneous hemorrhages: in twenty cases, there were two in which metrorrhagia occurred.

Virchow connects this tendency to hemorrhage with the affection of the spleen. Here, again, apply the remarks I made on the supposed relations between the alterations of the spleen and blood; for, as was observed long ago, great enlargement of the spleen following paludal fevers usually gives rise to hemorrhages. I may also refer to structural change of the liver, it being well established, particularly since so much light was thrown on this subject by Dr. Mönneret, that there is a tendency to hemorrhages when the liver is diseased. But, asks Professor Magnus Huss, may not the hemorrhages in leucocythæmia be more dependent on the excess of white globules? Being of larger diameter than the red globules, and having a tendency to become agglutinated, they will form clots obstructing the passage of the blood through the capillaries, and rupturing them: the result would be hemorrhage, more or less considerable in amount, according to the extent of the rupture of the vessels. I confess that this mechanical explanation does not much commend itself to me.

The urine is normal in the first stage of the disease; but towards the end, it contains ammonia and the urates in increased proportions.

In some cases, under the designation of complications, there have been noted concomitant affections of the lungs, such as tubercular disease, pleuritic effusion, sanguineous congestion, and œdema. In three of the cases detailed by Dr. Vidal, jaundice occurred during the course of the leucocythæmia: in another case, there was cirrhosis of the liver: and in three other cases, the leucocythæmia was complicated with Bright's disease.

Furuncular eruptions, with sloughing over the sacrum, and pemphigus (as in a case of Virchow and in another of Magnus Huss), have been observed to supervene during the last stage of leucocythæmia.

It is rather difficult to state with precision the duration of leucocythæmia, as we never know the exact time at which it began; but, speaking approximately, we may say, that in the cases related by authors, the disease lasted from three months to five years, the average duration being from thirteen to fourteen months. Its progress, therefore, is essentially chronic: its termination is death. On this point, all observers are agreed: in all the cases which have been reported, the account states, either that the patient had died, had not been cured, or remained in an almost hopeless state.

Different methods of treatment have been employed; but they have all proved not only ineffectual as means of cure, but even of no avail in temporarily arresting progress. Nevertheless, I am inclined to think that some relief will result from treatment directed to the anæmic condition; it may not suffice to cure the disease, but it may retard its progress, if it be allowable to draw a conclusion from what we saw in our patient. The preparations of iron have proved very useful; and preparations of cinchona have been of undoubted benefit in arresting hemorrhages. For some time after coming into hospital, our patient had every day rather alarming attacks of epistaxis: on having recourse to the powder of cinchona, the hemorrhages did not occur for some time. Every day, he took, in a little coffee, two grammes of the powder of yellow cinchona; for three weeks, he had no bleeding from the nose. But it recurred; and always yielded anew to the influence of a large dose of the same medicament. Notwithstanding the reality of this amendment, it was too slight to justify a more favorable prognosis.

To give you an idea of the medication which I recommend, let me quote the substance of the written advice which I gave to the Spanish merchant of whom I spoke a little time ago. I prescribed for him the waters of Pougues, recommending him to begin at once, if possible, to take them: I also recommended preparations of iron, and preparations of iodine, in the first instance alternately, and then combined—the successive use of saline, sulphurous, and ferruginous baths—powder of cinchona—wine of cinchona—bitters, such as quassia and nux vomica—and finally, a varied diet. The understanding was that the patient's Spanish physicians should treat him in accordance with this general programme, modifying details as circumstances changed.

LECTURE XCI.

ADENIA.

An Affection characterized by Progressive Hypertrophy of the superficial and deep Lymphatic Glands.—Hypergenesis of Glandular Cellules.—Never any Inflammation of the Glands.—Sometimes concomitant Hypertrophy of the Spleen, Liver, and Intestinal Glands.—The Disease has three Periods, viz., the Latent, the period of Progress and Generalization, and the Cachectic Period.—In the first period, there is no general disturbance of the system: in the second and third periods, there is Anæmia without Leucocythæmia.—Œdema of the Limbs, Ascites, and sometimes Anasarca.—Cough.—Dyspnœa.—Suffocative Attacks from compression of the Bronchiæ.—Duration of the Disease is from eighteen months to two years.—The Termination is almost always Fatal, either by an attack of Suffocation, or by the Cachectic State.

GENTLEMEN: From the earliest times, the attention of physicians has been directed to *engorgements*, or, as they were formerly called, *obstructions* of the viscera. In treating of the cachexiæ, the older writers attached great importance to obstructions of the spleen and liver.

Since the publication of the works of Sydenham, Morton, and Torti on paludal poisoning, there has always existed an anxious desire to ascertain its relation to enlargement of these organs. The anæmia and cachexia which accompany these visceral engorgements had led to the belief that there was an essential alteration in the constitution of the blood. Morbid changes in the constituents of the blood had been suspected, but not proved. You recollect with how much interest the works of Bennett and Virchow on a new disease were received—the disease to which they gave the name of leucæmia, and considered as the consequence of hypertrophy of the spleen, liver, or lymphatic glands.

At first great enthusiasm was created by the discovery of the two foreign savants, and everybody endeavored to adduce confirmatory cases. It was believed that there was a leucocythæmia connected with the spleen, and a leucocythæmia connected with the lymphatic glands. Numerous cases, however, have proved that engorgement of the liver, spleen, and lymphatic glands, may exist without there being any modification of the number of the white globules and globulines; and, moreover, the important researches of Professor Charles Robin have shown that white globules and globulines may be in excess in the blood, although neither visceral nor glandular hypertrophy exist. Moreover, hypergenesis of leucocytes may be met with in morbid states of the economy quite unconnected with any visceral engorgement, and even in physiological conditions.*

Gentlemen, it has been long known that hypergenesis of leucocytes is not a necessary consequence of hypertrophy of the spleen and liver. Clinical observation was not long in establishing that hypertrophy of the lymphatic glands might also exist without any increase in the number of globulines.

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* Journal de Physiologie de BROWN-SÉQUARD, p. 51. Paris, 1859.

The subject to which I wish to direct your attention to-day is Generalized Hypertrophy of the Lymphatic Glands. Since my attention was first directed to this strange affection, I have been struck with never seeing suppuration of those enormous lymphatic tumors, which for a time give rise to no other inconveniences than those arising from the effects of their pressing on organs.

I have also ascertained the incurable character of these tumors, which, though they never become the seat of inflammation, possess insurmountable tenacity, constantly tend to increase in size, and after a period of very indefinite duration, end by producing a most injurious influence on the constitution, sometimes causing death by compressing organs essential to life.

These facts have convinced me that we ought to regard the formation of these glandular tumors as a specific form of disease. Wishing to attach a special name to a special disease, I called it Adenia. This name, no doubt, might be applied to a great many glandular affections, such as cervical and mesenteric tuberculization. Nevertheless, I persist in giving the disease this name, that it may henceforth be an established fact that a new morbid species has been found in the extensive family of glandular diseases.

But, gentlemen, let me first of all beg you to understand that I have made no discovery—that before I described the disease it had been well described by others—in England by Hodgkin;[*] in France by Dr. Bonfils, one of my pupils;[†] and in Switzerland by Dr. Cossy.[‡] I lay claim to no other merit than that of having brought together a large number of cases collected from my own practice and from that of my professional brethren, associating them together by one common description, including them in one special name, and endeavoring to disseminate a correct knowledge of them.

My friend and colleague, M. Nélaton, has frequently addressed his pupils on the disease now under our consideration, and, like me, he has been disposed to regard it as a glandular affection quite special in its character. My colleague Professor Laugier (in whose hospital wards I saw the case reported by Dr. Bonfils), was equally struck with the peculiar characters of the disease. M. Leudet, the honorable and accomplished director of the Rouen school of medicine (with whom I have often discussed the subject), has remarked, with his usual sagacity, that adenia is a disease altogether special.

In 1861 Dr. Cossy published a memoir, in which he recorded three cases of simple general hypertrophy of the glands, accompanied by leucæmia. Finally, I am indebted to Drs. Potain and Laboulbène, my colleagues in the Parisian hospitals, for having been so exceedingly obliging as to communicate to me two cases, one of which occurred at the Hospice des Ménages, and the other at the Hôpital Sainte-Marguerite.

All these cases greatly resemble one another; and, from their attentive study, it appears that adenia consists in simple hypertrophy of the superficial and deepseated lymphatic glands, and in the formation, in different organs, of lymphatic products analogous to those met with in leucocythæmia, but—and the fact is both essential and characteristic—unaccompanied by any augmentation in the white globules of the blood.

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[*] Hodgkin: On some Morbid Appearances of the Absorbent Glands and Spleen. [*Medico-Chirurgical Transactions* for 1832, vol. xvii, p. 168]

[†] Bonfils: Réflexions sur un cas d'Hypertrophie Ganglionnaire Générale. [*Société Médicale d'Observation de Paris*, 1856.]

[‡] Cossy: Mémoire pour servir à l'Histoire de l'Hypertrophie Simple plus ou moins Généralisée des Ganglions Lymphatiques, sans Leucémie. [*Echo Médical*, t. v. Neuchâtel, 1861.]

The hypertrophy of the glands is sometimes accompanied by simple hypertrophy of the liver and spleen. Moreover, as I have just told you, and as was very apparent in Dr. Potain's case, there is sometimes hyperplasia of the aggregate and solitary intestinal glands.

In all the cases, excepting perhaps in one of those communicated to me by Dr. Leudet, there was no increase in leucocytes or globulines.

Adenia, therefore, is distinguished from other glandular affections, and particularly from leucocythæmia, by there being no appreciable alteration in the blood, excepting that which belongs to anæmia, and in the anæmia not showing itself till the second stage of the disease.

The patients generally apply for medical advice during the early months of the malady. They complain of numerous tumors on the surface of the body, and, sometimes, of slight dyspnœa. They state that they are in good health in other respects; there is no loss of appetite, no serious disturbance of the principal functions, and during the first five or six months of the disease, nutrition is not morbidly influenced in any appreciable degree.

Hypertrophy of the glands usually begins in the submaxillary region: soon afterwards, the patients are alarmed by observing the formation of tumors in the sides of the neck, in the axillæ, and in the groins. Less frequently, there is hypertrophy of the epitrochlean and popliteal glands. The enlargement of the submaxillary and cervical glands soon gives a singular appearance to the face: the head, which appears relatively small, rests on a glandular mass which the patients try to hide by adopting toilette artifices. The tumors in the neck are not associated with any change in the color of the skin. They have not contracted adhesions with neighboring parts, and it frequently happens that the individual hypertrophied glands remain quite free from each other. They are movable: they can be touched, squeezed, and even kneaded without causing pain. Those situate in the submaxillary region, joining with those of the opposite side and with the cervical lateral tumors, sometimes permanently assume the form of a band round the neck. The latter are not exclusively superficial. In one case, I have seen the lateral tumors on the sides of the larynx and trachea, and extending probably to the bronchi. They sometimes extend below the clavicle, and become continuous with axillary tumors. The axillary tumors are generally very large, being often the size of the egg of a hen or turkey. Drs. Escalonne and Leblanc had a patient near Fontainebleau, in whom they were as large as mammæ, to which indeed they bore a strong resemblance, both in respect of the color of the skin and the network of veins on their surface. Tumors so considerable in volume not only interfere with the movements of the arms, which they keep far apart from the trunk, but they likewise present an obstacle to the venous circulation, and not unfrequently also produce œdema of the hands and forearms. Sometimes, the subpectoral glands are enlarged.

The inguinal glands also become very large: consequences follow similar to those produced by the greatly enlarged axillary glands—that is to say, a difficulty in moving the arm, and an impediment to the free return of the venous blood. The feet and legs are nearly always œdematous. The inguinal tumors sometimes occupy the whole extent of the triangle of Scarpa; and often, the hand, when applied below Poupart's ligament, can feel similar tumors in the iliac fossæ. By introducing the finger into the vagina and rectum, similar tumors can be detected in the pelvis. In thin persons, by means of abdominal palpation, enlarged glands may be felt at the sacro-vertebral angle and along the vertebral column; but sometimes, the hand is prevented from reaching these deepseated regions by the abdominal fulness consequent upon enlargement of the mesenteric glands. In many cases, it is easy to recognize the existence of ascites. Let me also remark, that at the very beginning of the disease, and prior to the cachectic state, persons have been supposed to be suffering from anasarca, caused by visceral affections, whereas, in reality, they had simply extensive œdema of the extremities, and ascites, caused by embarrassment of the general and portal circulations.

In only three of my eleven cases have I observed hypertrophy of the liver and spleen. There may be great enlargement of the latter organ. In the case of a woman, twenty-three years of age, I saw the spleen occupying the whole of the left side of the abdomen, extending to the umbilicus, and filling the iliac fossa. It is important to note that in this case there was no leucæmia.

I shall very soon revert to the consideration of the manner in which the symptoms of adenia are connected with one another; but before doing so, I wish to describe to you the case by which I have been most struck.

On February 20th, 1863, I was consulted by Madame X., aged twenty-three, affected with general glandular hypertrophy. At that date she looked in good health. Her temperament was not lymphatic: her color was sufficiently good: her eyes were bright: and had attention not been called to her condition by the presence of greatly enlarged submaxillary glands, one would have been far from believing that she was the subject of any serious organic mischief. She had not suffered in childhood from any scrofulous affection, from suppurating glands, nor from chronic coryza: in her later years, she had had neither herpetic sore throat nor persistent sore eyes. This young woman had always menstruated regularly: she married at nineteen, and had two children, who are in good health. There is a deficiency of information as to the antecedents of this patient's family; but we know that her father and mother are both living and in good health.

About nine months ago, that is to say, in the third or fourth month of her last pregnancy, Madame X. observed that she had small tumors in the groins and axillæ: in a fortnight, these glandular swellings acquired considerable volume, and other glands became enlarged, including glands at the angle of the jaw and in the occipito-cervical regions. The attention of Drs. Henrot and Landouzy (of Rheims) was arrested by so general an enlargement of the glands, and one which was so rapid in its progress. They attributed Madame X.'s cough to a similar enlargement of the bronchial glands. She had a favorable confinement. After the birth of her child, large glandular masses were detected in the iliac fossæ, and in the sacro-lumbar region. When the patient first came to me, a general glandular hypertrophy was apparent; the occipital, submaxillary, cervical, inguinal, epitrochlean, popliteal, and axillary glands were as large as eggs of pigeons or hens. On making digital pressure on either side behind the pubic arch, a deep chain of glands was felt. By palpation, the spleen was found to be much increased in size. This young woman never had had intermittent or hectic fever. I beg you to remark particularly that her health always had been, and was when I examined her, unexceptionably good.

It being important to ascertain whether the blood contained an excess of white globules or of globulines, an examination was made by M. Dumont-pallier. The result showed that it contained exceedingly few white globules, which were lost amid a great number of normal red globules: no globulines were found.

I have mentioned that the patient had a small dry cough. She never had had hæmoptysis. On the left side, respiration was natural; but on the right side, during expiration, there was a bellows-sound and moist râles. There was no notable dulness at the apex of the lungs. The heart was healthy. There was no blowing sound in the vessels of the neck. There was no œdema of the extremities. There was no loss of appetite; digestion was easily accomplished; nutrition seemed normal; and there was no appreciable loss of flesh. Sleep was good; there was no depression of spirits. In fact, Madame X. would not have thought of making any complaint, had it not been for the annoyance she experienced from the formation of the glandular tumors.

Here then is the case of a young woman who had always enjoyed good health up to the third month of a pregnancy, when she perceived that a great number of small tumors were forming below the inferior maxilla, in the neck, axillæ, and groins, which tumors soon increased in number, and acquired a great size. She had a good confinement. She was able to nurse her infant for some months, after which her milk dried up; and then a new impetus was given to the development of the tumors. The lady's health, however, appeared satisfactory up to the day on which she consulted me. She had a good complexion, experienced no pains anywhere, digested her food easily, and seemed to be normally nourished by it. The short dry cough by which she became affected during her pregnancy continued; but as I could not associate it with any serious lesion of the lungs, I adopted the opinion of Drs. Henrot and Landouzy; with them I believed that the cough was caused by pressure on the air-passages, occasioned by hypertrophy probably of the glands surrounding the right bronchus. This explanation was founded on the absence of organic lesions of the lungs, and on the generally increased size of the superficial and deepseated lymphatic glands. I did not conceal from the family my uneasy forebodings as to the issue of the disease. There was, in fact, a probability that the patient would succumb to repeated attacks of suffocation, to asphyxia arising from bronchial obstruction, or to the cachectic state progressing so rapidly as to induce marasmus, profuse sweating, or colliquative diarrhœa. My sad forebodings were not long in being verified: the general modifying measures which I recommended were of no avail; and I learned, some months later, that the patient died from cerebral symptoms, having previously had subcutaneous hemorrhages.

So far as I know, no examination of the blood in the latter months of pregnancy had been made; and I have no grounds for absolutely denying that leucæmia existed in the latter stage of the disease. But if it be supposed that the lymphatic glands and the spleen had acquired, after my examination, a very much greater development than that usually observed in cases of leucæmia, and that as yet leucocytes had not been in excess—if, again, we compare the case of this private patient with the cases we have now been studying, in which examination by the microscope never enabled us to detect excess of leucocytes—we are justified in supposing that the progress of the disease was that usually observed, and that, in all probability, the patient never had been leucocythæmic.

Notwithstanding this imperfection in the history, the case teaches the important lesson that a person apparently in perfect health, and whose blood seems in a normal condition, may be the subject of hypertrophy of all the lymphatic glands and of the spleen, and may sink very rapidly under the influence of glandular cachexia. If the patient be not leucocythæmic, are we to conclude that the blood has retained its normal composition? Certainly not: for in all the cachexiæ, the constitution of the blood is essentially modified: but the point I wish to establish is, that for at least the first ten months of her affection, the patient presented no symptoms of hæmatic lesion, nor of splenic or lymphatic leucæmia.

Once when commenting upon this case, I remarked to you, that should the patient not succumb from the cachexia, she might die asphyxiated from air not reaching the lungs. An example of this termination of general adenia I recollect occurring in the case of a young man whom I saw with my lamented colleague, the late M. Amussat.

More than twenty years ago, that distinguished surgeon summoned me to Néothermes to see one of his patients, a young man whose usual residence was at Poitou. For nearly a year, he had been the subject of general glandular engorgement. The lesion, however, existed more particularly in the neck, thence extending behind the clavicle and sternum. This condition produced so much difficulty in breathing that death seemed imminent. M. Amussat wished me to perform tracheotomy; but I said to him, that, although I was accustomed to perform tracheotomy in children having croup, and in adults having chronic diseases of the larynx, I felt that I ought in the case under discussion to leave to men of more authority, and to hands more practiced, an operation beset with peculiar difficulties. I perceived that both the trachea and the bloodvessels were displaced, lost, as it were, amid numerous tumors; and that I could not introduce the bistoury with safety into regions where I had no guide in the anatomical relations of the parts.

M. Amussat, however, refused to perform the operation, fearing that affection for the patient, who was his intimate personal friend, might make his hand unsteady. Under these circumstances, it became incumbent on me to undertake the perilous duty, relying on the assistance of my friend, who did not wish to undertake the principal part in the operation.

As soon as I had divided the superficial cervical aponeurosis, the tumors, till then imprisoned, spurted out, as it were, liberated by my incision, respiration, at the same time, becoming a little less difficult. It was then necessary for me to turn out with blunt hooks seven or eight tumors placed in front and at the sides of the trachea, which I exposed with extreme difficulty, having to avoid with the greatest care the numerous veins which presented themselves. Having opened the trachea, I introduced into it a large canula; but to my great regret, this hardly produced any alleviation of the dyspnœa: it was evident, that the passage, beyond the internal extremity of my canula, was compressed by other tumors. Having, however, dreaded this occurrence, I had provided myself with a canula wider than I generally employ in adults, and the length of which exceeded ten centimetres. This I substituted for the canula which I first employed. When I had introduced it nearly six centimetres, I came upon an obstacle which with difficulty, and by using an alternation of pushing and withdrawing movements, I was able to surmount. When once this obstacle was overcome, respiration became easy; but this did not prevent the patient dying on the following day.

For two reasons I have been anxious to lay this case before you: first, to show you the manner of death when the bronchial glands and the glands in the course of the trachea are involved in the adenia; and second, to point out how little hope of success tracheotomy offers in these cases. In a patient from Stockholm, who came to consult me at another period, attacks of asphyxia sufficiently informed me that the trachea and bifurcation of the bronchi were constricted by glandular tumors.

This is the manner in which the fatal issue is hastened in patients affected with adenia. We see a particular patient die eight or ten months after the beginning of the disease, who might have lived several years longer, like the patient of M. Bonfils, and like several other patients whose history I have related, had the disease more specially attacked parts in which the consequent disorders could not become immediately dangerous.

I have just given you proof of the necessity of performing tracheotomy in certain cases of adenia. You have seen that opening the trachea only prolonged life some hours: as, however, our object was to avert death, I should not hesitate again to recommend the operation, even though I were convinced that it was an almost desperate resource.

Let us now study the progress of the dyspnœa when the tracheal and bronchial glands are invaded. In the first stage of the disease, that is to say, before there is any cachexia, and when the hypertrophy of the glands has not reached this last limit, the patients often complain of oppression, of inability to walk quickly, and of not being able to go up a stair without being blown. Though this dyspnœa is organic, it sometimes shows itself with the characteristics of nervous dyspnœa. Generally, the patients are seized with dyspnœa during the night; they are unable to sleep in bed, and consequently pass their nights in an arm-chair. My Stockholm patient wrote to tell me that he felt a tightness in the respiratory tubes [*un resserrement dans les tuyaux respiratoires*]. One of the patients (an account of whose case has been communicated to me by Dr. Leudet) had had for some time shortness of breath and fits of dyspnœa. In both these cases, I was enabled by attentive observation to ascertain that there was no lesion of lungs or heart. The lungs may become accustomed to the pressure, and so the fits of dyspnœa may become less frequent; but in this case, either from rapid increase in the volume of the tumors or from the enfeebled patient requiring at times to inspire a greater quantity of air, it was observed that in the latter days of his life, the suffocative attacks were more formidable, being accompanied by purple lips, haggard eyes, great anxiety, and cold extremities. The pulse became thready and the inspiration hissing.

The embarrassed respiration may occasion passive hyperæmia of the lungs, causing mucous râles. Dr. Leudet has sometimes met with pleuritic effusion on one or both sides independent of any appreciable inflammatory action. Without seeking to explain this effusion into the pleural cavity, I ask: Is this effusion, which does not originate in inflammation, analogous to œdema of the extremities induced by impediment to the circulation when the glands in the course of the vertebral column are themselves hypertrophied, as was found at the autopsy of one of the cases communicated to me by Dr. Leudet? When I come to speak of the pathological anatomy of the disease, I will relate to you this case of the Professor of the Medical School of Rouen. At present, as my more immediate object is to establish the symptomatology of this strange disease, I wish to describe two cases which occurred under my own observation, which show the frequency of dyspnœa in patients affected with adenia. No doubt can exist as to the mechanical cause of the dyspnœa in M. Amussat's case—as will be still better seen when we discuss the pathological anatomy of the disease.

On the 5th April, 1863, I sent back to Bonnevau a patient who consulted me for the first time in February of that year. He resided in the vicinity of Fontainebleau; and his ordinary medical attendants were Drs. Escalonne and Leblanc.

N., by occupation a wheelwright, was 68 years of age. He had always enjoyed excellent health. In October, 1862, he perceived a small, perfectly indolent tumor in the neck, to which he attached no importance. A month later he had as it were an explosion of tumors in the neck, axillæ, and groins. When I saw him in February, 1863, three months and a half after the first appearance of the tumors, the neck was deformed by a multiplicity of round elastic tumors, which were blended with one another or soldered together. The tumors in the axilla were specially remarkable for their size; those in the groins were not so large. I could not feel any in the abdomen. There was no hypertrophy of the spleen. The complexion was good; and the general health was excellent. I prescribed baths medicated with the bichloride of mercury, and the internal use of the tincture of iodine.

When I next saw the patient, I was struck with the increased size of the tumors. The neck was monstrous, being in circumference as large as the head, and having a frightfully lumpy appearance. In front of each ear there was an enormous, vertically situated, long-shaped tumor, which was joined below to a circular band of other tumors occupying the submaxillary and lateral regions.

The face, fresher and more florid than is usual in an old man of sixty-eight, was as it were set in a frame formed by a mass of tumors occupying the neck anteriorly and laterally, and situated in front of the ears. At the base of the neck and behind the clavicles were numerous tumors imbedded in the muscles and extending into the chest. There were likewise engorged glands at the back of the neck, reaching to its base.

Under the axillæ the swelling had quite a monstrous appearance. In each armpit there was a tumor as large as the head of a full-term fœtus, looking like a large mamma, multilobular at its base, and resting on a mass of engorged glands.

In the groins the tumors were smaller, though of considerable size. On palpation of the abdomen numerous tumors were felt, particularly in the left side. The liver was slightly hypertrophied. The spleen was healthy.

The complexion was excellent, and the health was good; but respiration was difficult and whistling, and mucous râles were heard in the chest. The voice retained the normal tone and character, showing that the larynx had not undergone any morbid alteration. Compression of the trachea and bronchial tubes was probably the cause of the whistling respiration.

During the following month I saw the patient for the third time. The glandular tumors had acquired a still more monstrous size. Although those in the anterior part of the axilla had acquired a size and aspect giving them the appearance of mammæ covered by a network of veins, I did not discover any œdema of the hands. There did not seem to be any loss of health, and the appetite had improved: nevertheless, it was easy to make the gums bleed. Neither the spleen nor liver were hypertrophied. The abdominal glands were more numerous and larger than in the previous month.

I recommended the patient to continue the internal use of tincture of iodine, and to take thrice a week sublimate baths. The pain and slight redness which I had observed in one of the tumors of the right axilla led me to hope that these tumors might lose their hardness and be absorbed. I knew, however, that adenia never terminates in acute inflammation and suppuration of the glands. At first, the patient found himself the better of the treatment which I had recommended; but I afterwards learned from Dr. Escalonne that the cervical and axillary tumors had acquired an extraordinary size, and that compression had rendered the trachea impervious to air. The patient died in a state of asphyxia, no other morbid condition having apparently been induced. The tumors did not suppurate.

The specially remarkable feature in this case is the sudden appearance of a multiplicity of tumors a month after a small indolent swelling had shown itself in the neck. Within three months the submaxillary and cervical tumors had acquired so great a magnitude that the base of the face, continuous with the neck down to the clavicles, was larger than the rest of the head. It was apparent, moreover, as is shown by my description, that these enormous tumors were continuous behind the clavicles and sternum: they accounted for the patient's dyspnœa and whistling respiration. In this particular case the abdominal glands were numerous and of very large size; the liver and spleen were normal. Notwithstanding this general and rapidly developed hypertrophy, the general health of the patient did not at first appear to be affected: it was not till the close of the fourth month that there was bleeding from the gums. I regret that no microscopic examination of the blood was made at this period.

Some months later I received, in my consulting-room, a gentleman from Stockholm, aged about thirty, who came for my opinion regarding tumors in various parts of his body. For a long time he had had a running from the left ear: then, after having suffered during the whole of the year 1861 from a feeling of discomfort which he could not account for, he remarked, in June, 1862, that a small tumor had appeared below the discharging ear. Three weeks later several similar tumors became developed on the same side of the neck, and were not long in acquiring a great size. The right side of the neck, the groins, and the axillæ soon became the seats of tumors. Professor Malmsten (of Stockholm) recommended the baths of Kreuznach, which accordingly were taken in September, 1862, After the course of baths, the debility became more marked, the sweatings were profuse, and the appetite nearly lost. The tumors, however, did not seem to increase in volume, and from about the end of December of that year to the 20th January, 1863, they notably decreased in size; there was a perceptible return of the appetite, and, subsequently, of the strength. But after the end of January the patient's appetite diminished, he became very impressionable, and his glands reassumed the size which they had lost. Every evening there was a slight paroxysm of fever. Such was the condition of the patient when he came to consult me. The cervical and axillary glands were very large. I did not detect hypertrophy of the liver or spleen. I recommended sublimate baths and a tonic regimen. In the first instance this treatment did not seem beneficial; for, six weeks later, the patient wrote to me to say that he continued in a state of great debility; that, being unable to lie on his bed, he was obliged to pass his nights in an armchair; and that, as soon as he attempted to stretch out his legs, he was seized with oppression at the chest, and felt a sensation of constriction in the respiratory passages. In June of the same year, I learned that my patient had consulted Virchow. The learned professor declared the malady to be curable; and thought the adenia had originated in the previous discharge from the left ear. On this occasion, it was ascertained that there was no organic lesion of the lungs, heart, liver, or spleen. Virchow, moreover, affirmed that there was no leucocythæmia. With a view to soften the tumors, and facilitate their absorption, he recommended that they should be treated by local cold baths.

Here, gentlemen, I must remark that, in this particular case, adenia was confined to the upper part of the body, there having been no hypertrophy of the glands in the inguinal and abdominal regions, nor of the liver or spleen. Virchow himself had affirmed that there was no leucocythæmia. The affection was, therefore, special in its character; and, although the learned professor of Berlin did not anticipate a fatal issue, he agreed with me in considering the glandular affection to be of a special nature.

In giving you the history of this case, I mentioned that the chronic discharge from the left ear was the starting-point of the adenia. I will, by and by, make use of this etiological statement, connecting with it analogous cases in which acute or chronic local irritation—irritation of the nasal or ocular mucous membrane—seem to have played an important part.

Perhaps, there is reason to suspect that in these cases there had existed some forgotten irritation of the skin or mucous membrane to account for the original adenopathy, the precursor of the outbreak of general adenia.

I have told you that the patients may sink from the severity of the suffocative paroxysms: I have endeavored to point out to you the part which hypertrophy of the tracheal and bronchial glands seems to have in the production of those phenomena, admitting at the same time, that the nervous condition of the patient has some share in their production. Derangement of the functions of digestion, innervation, and nutrition, important in itself, though only of secondary importance in relation to the progress of the adenia, is sooner or later observed. In the second stage of the affection, there is loss of appetite: digestion is slow and painful: nevertheless, diarrhœa seldom occurs. Ere long, wasting supervenes, accompanied by great œdema of the extremities, and extreme debility. The hands are occasionally the seat of erythematous eruptions, and sometimes the legs are covered with ecchymotic spots, or the cutaneous affection may be cachectic pemphigus, as in the case of Dr. Leudet's patient. Some are exhausted by profuse sweating and hectic fever, as occurred in the cases of Dr. Perrin's patient,* and in my Stockholm patient.

It appears then, that adenia terminates by occasioning great disturbance of the principal functions: hæmatosis is soon interfered with. Adenia lowers the temperature of the body, or, to speak more correctly, it renders the patients more sensitive to cold. It disorders digestion. It induces cold sweats and hectic fever. It is, consequently, an affection of very great gravity, and one in which the physician has hitherto found himself pretty nearly powerless. The usual duration of the disease is from eighteen months to two years.

I am now going to read to you the report of a case observed by Professor Leudet, in his wards of the Hôtel-Dieu of Rouen: it is very complete, and will enable you to study the progress of the disease and its termination in cachexia.

"Victor R., a tall man, aged fifty-seven, was admitted to the Hôtel-Dieu on the 6th December, 1862. In former years, he had been exceptionally plump. When about eighteen years of age, he had begun to take alcoholic drinks freely: he was drunk at least once a week, and on the day following the debauch, could only eat food of high relish or dressed with vinegar. In his youth, he had never had engorgement of the glands, nor otorrhœa, nor ophthalmia. He had passed the whole of his military service in a regiment of cuirassiers: during that period, he contracted chancres followed by a suppurating inguinal bubo. No ulterior symptoms of constitutional syphilis were manifested by the skin, mucous membranes, or bones. After his discharge from military service, he took alcoholic drinks to still greater excess than formerly. The only morbid effects of these excesses were pains in the stomach, there being absence of vomiting, diarrhœa, jaundice, or derangement of the functions of the nervous system. The patient had had no serious illness prior to the adenia.

"Four years ago, tumors appeared in rapid succession on the anterior and external aspect of both legs, and about the middle of the posterior surface of the right forearm: they were hard, did not suppurate, and were the seat of pain equally by day and by night. These tumors, of which the patient gave a very incomplete description, must have disappeared after two or three weeks of treatment, during which recourse was had to the iodide of potassium. He continued very weak for nearly six weeks after this illness, nevertheless, not experiencing any notable pain in the joints or limbs. This weakness entirely left him, and he never again experienced anything resembling it.

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* Perrin: Bulletins de la Société Anatomique for 1861, p. 247.

"Fifteen months ago, chronic coryza appeared, with loss of the sense of smell, but unaccompanied by any feeling of general discomfort: some months after the beginning of the coryza (which was purulent and somewhat sanguinolent), an affection was observed at the inner angle of the left eye, in the situation of the lachrymal sac. This affection, which terminated in suppuration and cicatrization, must have occurred after the illness had lasted three months. It was during the last stage of the inflammation of the lachrymal sac that R. observed, for the first time, that the lymphatic glands of the neck were swollen. His strength became at the same time greatly enfeebled, without there being any paralysis.

"Ten months ago was the date of his first admission into the Hôtel-Dieu. He was received at that date into the surgical department, for conspicuous swelling of the lymphatic glands of the cervical, axillary, and inguinal regions. R. remained five months and a half in the surgical wards. During that period, the glands decreased in size, were devoid of pain, and presented the same characters as at my first examination. During the same period, he experienced no disturbance of the digestive organs, nor had diarrhœa, epistaxis, or any kind of hemorrhage.

"On leaving the surgical wards of the Hôtel-Dieu, on the 25th October, 1862, he was employed in the charity workshops, living on seventy-five centimes a day. During that period, he experienced great loss of strength. About the middle of November, 1862, he became affected with cough, accompanied by pain in the right side of the chest.

"When admitted into my wards, I found R. in a state which I shall now describe to you: The face was pale: there was still a certain plumpness of appearance, though according to the patient's statement, that had much diminished. There was a marked depression of vital power, particularly since the cough had set in. There was anorexia of recent date. There was no diarrhœa. The face was swollen, particularly in the submaxillary and subhyoid regions, in which, by palpation, could be recognized twenty round, movable tumors, which were not painful even when pressed. The most bulky of these tumors were as large as pigeons' eggs, and the smallest were the size of filbert-nuts. The hypertrophied glands gave the appearance of a double chin, with lateral enlargement of the face. There were tumors of similar character in the subclavicular regions, but none in the back of the neck. The largest tumors were in the axillæ, particularly in the right axilla, where the aggregate bulk exceeded that of an adult's fist: it was made up by other smaller tumors which were not fused together. There was a similar but not so great an enlargement of the glands in both inguinal regions, and in the inguino-femoral triangle. Several small enlarged glands existed above the condyles on both sides. The enlarged glands were nowhere adherent to the skin, which presented neither change of color nor vascular dilatation.

"The coryza continued, retaining its purulent and sanguinolent character. There was no deformity of the bones of the nose. The skin was adherent, in the situation of the left lachrymal sac, which adhesion at the inner angle caused depression—to the extent of one half—of the left upper eyelid. There was no redness of the eye; and vision was unaffected.

"For three weeks, the cough had been very severe; the expectoration was slightly muco-purulent, devoid of brown color, and yielding a fetid smell like that of sphacelus. By auscultation, numerous subcrepitant and even some sibilant râles were heard on both sides of the chest: they were more numerous on the right than on the left side. There was slight tension of the abdomen, but no ascites. In consequence of the abdominal walls being capable of only slight depression, the detection of enlarged glands within the abdomen was impossible. There was no morbid increase of spleen or liver. The urine did not deposit a sediment: on examination by heat and nitric acid, it gave no indication of albumen; nor did it yield any trace of glucose on being tested by potassa and Barreswil's solution. The blood, which was examined several days after the patient's admission to hospital presented nothing abnormal—there was no numerical augmentation of the white globules or globulines, and the red globules were normal. That examination, repeated several times between that time and the present date (7th April, 1863), has always yielded similar results. I prescribed Barèges water, twelve drops of essence of turpentine in a julep, weak wine and water as a drink, and ordinary diet.

"During December, 1862, R.'s condition ameliorated, the fits of coughing became less intense, and never occasioned real orthopnœa: the fetid odor of the sputa diminished, and finally disappeared about the end of that month. The strength was always below the natural standard. There was no diminution in the size of the glands. About the end of December, slight œdema supervened in the lower extremities. The urine continued to be non-albuminous. I ordered decoction of cinchona, pills of the iodide of iron, wine and water as a drink, and a double allowance of aliment and of wine.

"During January, 1863, there was a slight increase in the size of the glands, particularly in those of the neck and axillæ: there was some enlargement of the abdomen, and the inferior margin of the liver began to be appreciable below the false ribs: there was an extension of the splenic dulness, but the inferior margin of the spleen could not be felt below the left false ribs: there was a little increase in the œdema of the lower limbs. The bronchitis was nearly at an end: some sibilant and subcrepitant râles were heard at the base of both lungs. The purulent discharge from the nose continued. There was no longer any fetid expectoration. The patient improved somewhat in strength: he was able to be up during the greater part of the day: and his only complaint was of the inconvenience caused by the œdema of the legs and the abdominal swelling.

"From the middle of February, 1863, to the beginning of April, the lymphatic glands in the submaxillary, subclavicular, axillary, subepitrochlean, and inguinal regions, in succession, increased in size, but preserved the same characteristics which they presented when the patient was admitted to the hospital. Day by day, there was a marked increase in the volume of the spleen, which was observed to have descended to the anterior superior spine of the ilium, its sharp edge being apparently directed forwards. Ascites existed to a slight extent. There was never any symptom of cardiac disease. The liver extended, at the least, two finger breadths beyond the margin of the right false ribs. The patient's strength was good; and he occupied a part of each day in walking in the court. He continued under careful observation in my wards.

"From June, 1863, R.'s state became more serious; and there was a return of the diarrhœa. At the beginning of that month, the lower extremities became the seat of slight œdema, and some bullæ of cachectic pemphigus appeared on the dorsal aspect of the carpus of one hand. The ozæna continued. From about the middle of June, there was a continuance of slight dyspnœa, increasing when he moved, but without any roaring [*cornage*].

"At the beginning of July, the debility had become more marked. At this date, there was no albumen in the urine, *nor any leucocythæmic altera-*

tion of the blood. On the 15th July, I detected pleuritic effusion occupying the lower half of the right pleura. Death occurred on 19th July, without there having been any dyspnœal paroxysm or alteration of voice.

"An *autopsy* was made on the 21st July, thirty-seven hours after death.

"The cranium and brain presented no morbid appearances. The nasal fossæ (examined behind only, from the body being claimed) presented marked thickening with a grayish tint and softening, without ulceration of the membrane. The bones, examined by the cranium, seemed to be in a perfectly healthy state.

"There was no morbid change in the larynx, trachea, nor in either bronchus: there was neither flattening of their parietes, nor change in their calibre.

"In the lower half of the right pleural sac, there was about half a litre of effusion composed of a gelatinous serosity tinged with blood. A layer of soft citrine-colored serosity covered the rest of the lung. This lung was rather more resistant than a normal lung—somewhat hard as in non-granular pneumonia. There was no deposit of morbid tissue. The bronchial tubes were not dilated. There was hardly any engorgement of the left lung. The heart and pericardium were healthy. The pulmonary artery was free from embolism.

"There was a little effusion into the peritoneal cavity—about half a litre of citrine-colored serosity free from false membranes. The liver was normal in size, yellowish in color, without manifesting this hue externally: it was in a state of fatty or amylaceous degeneration. The spleen was much enlarged. Its length was $0^{m}{\cdot}26$, and its breadth $0^{m}{\cdot}17$. There was no thickening of the fibrous capsule. The parenchyma had a color like that of the lees of white wine, was not soft in consistence, nor had it any thickening of the stroma. The kidneys were of the usual size, somewhat pale, and healthy in structure.

"There was great enlargement of the lymphatic glands, particularly in the neck, submaxillary, and subclavicular regions, in both axillæ, in the abdominal cavity in front of the vertebral column, and in both inguinal regions. Most of these enlarged glands were collected and weighed together, when their weight was found to be a little under four kilogrammes. They varied in size: the largest were those of the abdomen, whether taken singly or collectively. In that region, they formed a mass nearly as large as the head of an adult. In the axillæ, the glandular masses were the size of a young person's fist. The largest of the enlarged glands—those of the abdomen—were the size of a turkey's egg. The cervical, axillary, and inguinal glands were redder than those of the abdomen, which had a whitish hue. The capsule was not thickened; and the contained gland was throughout rather soft, diffluent, and without thickening of the stroma.

"There were also some hypertrophied glands in the fold of both arms. A slightly flattened substance, formed by rather voluminous reddish glands, existed in both vertebral sulci, below the parietal pleura.

"The bronchial glands were relatively much less developed than those of the neck, and particularly than those of the axillæ: two of them, however, were as large as filbert-nuts, but did not compress the vessels or air-tubes.

"The thoracic cavity was healthy.

"The microscopic examination of some of the glandular tumors showed me that their parenchyma contained nothing cancerous, nothing fibro-plastic, and nothing of the nature of amylaceous degeneration, but only minute nuclei, less than the elementary cells of lymph, and somewhat resembling epithelial cells."

To sum up the case: A man, of fifty-seven years, a man who had always enjoyed good health, notwithstanding the frequency of his alcoholic excesses, and who in childhood had given no indication of scrofula, presented four years ago, on the legs and right forearm, tumors which terminated in suppuration, and might be regarded as syphilitic gummi. When this patient was admitted to Professor Leudet's wards, he was suffering from chronic coryza, and bore the cicatrix of a lachrymal tumor. It was during the suppuration of this lachrymal tumor that the first cervical tumors appeared; and it was not till a later period that similar swellings became developed in the subclavicular, subaxillary, and inguinal regions. These tumors were large and indolent. Afterwards, the adenia progressing, and there being no doubt as to its generalization, the increased size of the liver and spleen was detected. On several occasions, the blood was examined without the discovery of an excess of leucocytes or globulines; and there was an entire absence of the principal symptoms of splenic or lymphatic leucæmia. Four months before death, the glandular and splenic hypertrophy made rapid progress: the patient became more and more feeble, the œdema of his lower extremities more marked, and his ascites more considerable. At a later period, bullæ of cachectic pemphigus appeared on one of the feet: cough set in; and there was pleural effusion occupying the inferior third of the right side of the chest.

The autopsy showed that the serious lesions were confined to the spleen and lymphatic glands. It was also seen that these lesions depended on simple hypertrophy of the organs, microscopic examination disclosing nothing more than an increase of normal elements.

However, although to ordinary methods of investigation, the blood presented no appreciable alteration, it was impossible to get rid of the belief that there existed in the case I have now detailed, and in other similar cases, a special dyscrasia of the blood; for the patients sunk into a state of cachexia, when not carried off by a suffocative paroxysm. This dyscrasia, with the nature of the essence of which we are not acquainted, is not a matter of doubt with me; and although general adenia produces no excess of leucocytes or globulines, nor diminution of the normal number of blood-globules, it probably modifies the lymph elements in such a manner that at the end of a period of indefinite duration, anæmia sets in, which is followed by cachexia. English physicians, more particularly Dr. Pavy and Dr. Wilks, have described a variety of anæmia to which they gave the name of *lymphatic anæmia*, meaning to express by that term the lesions of glands which a special kind of anæmia may induce. I confess, gentlemen, that I am disposed to accept this view.

In the present day, every one admits that the formation and maintenance of the blood are functions performed by the co-operation of certain organs: pathology, better even than physiology, proves the hæmatopoietic agency of these organs. We know, for example, that chronic affections of the liver and spleen produce serious consequences in respect of the composition of the blood; for whatever be the nature of these affections, they are followed by anæmia with modification of the number, consistence, form, color, and chemical composition of the blood-globules. Tuberculosis of the mesentery leads to the same result: so does scrofula, the different manifestations of which have their principal seat in the lymphatic system.

Need I remind you of the influence of organic lesions of the lungs upon the composition of the blood? The necessary consequence of all these lesions is to interfere with the exchange of gas, the very essence of pulmonary hæmatosis, and so lead to anæmia. There is, then, a kind of anæmia which is of pulmonary origin, just as in all probability, there exists in the fœtus an anæmia of placental origin, when the placenta has undergone a partial fatty and fibrous degeneration. If then, we grant that there is a pulmonary anæmia, a splenic anæmia, and a hepatic anæmia, why should not we also accept a *lymphatic anæmia?* To be satisfied that it exists, we only require to bear in mind the function of the lymphatic system. The ramifications of this system, in the interior of organs and in the intestinal mucous membrane, derive the elements required for the maintenance of the blood. The lymph and the chyle undergo a modification in the parenchyma of the glands, and then all the lymphatic fluid, whatever may have been its origin, is poured into the venous system, and like the subhepatic, splenic, and intestinal blood, it passes on to the lungs, there to undergo such modifications as are necessary to convert it into blood suitable for the nutrition of all the organs. Gentlemen, I cannot tell you what special action on the composition of the blood is caused by general hypertrophy of the glands: I do not know whether it notably diminishes the leucocytes, but I am certain that it does not increase them. Microscopic investigation and chemical analysis have not informed us how the blood is modified in adenia, but the modification itself is demonstrated by the symptoms; and it cannot be otherwise when all the glands in the body have become greatly hypertrophied. Again, the modification of the blood is proved by the anæmia and cachexia under which the patients succumb when they do not die from asphyxia caused by compression of the trachea or bronchi. I hold then with Drs. Pavy and Wilks, that there is lymphatic anæmia, just as there is hepatic anæmia and splenic anæmia. Possibly, leucocythæmia, on which I have already lectured to you, is only a variety of anæmia characterized by an excess in the proportion of leucocytes or globulines. We know, in fact, that the presence of leucocytes is compatible, in a certain measure, with health, and is met with in a great number of morbid states, without appearing to aggravate them: on the other hand, we know, that those organic lesions which are regarded by Virchow and Bennett as the causes of leucocythæmia may be wanting; and, finally, we know that hypertrophy of the lymphatic glands and hypertrophy of the spleen—the condition of organs peculiarly characteristic of the lesion—may exist, and often do exist in cases in which there is no leucocythæmia. It appears, therefore, that the only symptoms which declare the gravity of the malady are anæmia and cachexia, the latter being always the ultimate consequence of deep and long-continued anæmia.

Clinical observation has told us that the malady is characterized by an augmentation in the size of the glands: necroscopic examination has absolutely proved that the tumors are in the lymphatic glands, and that the surrounding tissues do not present the least trace of inflammatory action. By careful dissection, we can isolate each of the individual glands which in the aggregate constitute the glandular masses. These tumors—mammary in form and elastic in consistence—have sometimes attained an enormous weight. Dr. Bonfils met with an inguinal tumor of this kind which weighed 2250 grammes, the axillary tumors weighing 1000 and 500 grammes. The superficial and deep cervical glands, the occipital glands and the whole circum-maxillary chain of hypertrophied glands gives the face a peculiar appearance: they may vary in size between a pigeon's egg and a hen's egg. The deepseated glands—that is to say, those in the thoracic and abdominal cavities—may become greatly hypertrophied. In the chest, the tumors are seldom larger than a pigeon's egg; but from their disposition around the trachea and bronchi, they may compress these organs. At no autopsy has it ever been found, that the glandular masses compressed and deformed the large vessels near the heart. The intra-abdominal glandular masses are found to be constituted by the pelvic, lumbar, and aortic glands; and by the mesenteric, mesocolic, gastro-hepatic, gastro-splenic, gastric, and pancreatic glands. In the case of Dr. Bonfils, the former (which belong to the general lymphatic system) weighed 3620 grammes; and in Dr. Leudet's case, some abdominal glands were as large as turkeys' eggs. The glands of the chyliferous system usually constitute less voluminous tumors: Dr. Bonfils, however, has observed them sometimes as large as pigeons' eggs. Let me add, that the glandular masses in the axillary and submaxillary regions are sometimes continuous with the intra-thoracic masses by means of bands of hypertrophied glands. A similar remark is applicable to the pelvic and inguinal glands. The chyliferous system is usually less affected, as I have already said, than the general lymphatic system: the glandular hypertrophy may also, however, have its seat in the glands of Peyer, as was found in Dr. Potain's case.* "From about the middle of the small intestine to the ileo-cæcal valve, a great number of the solitary glands were seen to be white and prominent: Peyer's patches to the number of twenty were very apparent, slightly prominent, a little granular on their surface, elastic, not notably hard, of a dull white color." It must also be stated that in this case some of the mesenteric glands were as large as hens' eggs.

My object in entering into these anatomical details is to show you, that no gland escapes, and that in all of them the hypertrophy may be great.

All observers concur in stating that the envelope and stroma of the gland do not undergo any change. Section of the parenchyma presents a grayish appearance in the smaller glands, a yellowish-gray in those of medium size, and a yellowish color in those of larger volume. The latter alone present ecchymotic spots in their interior; and their section, when scraped, yields a turbid whitish fluid which mixes with water: one might suppose that they contained a fluid analogous to that of cancer, containing large cellules, or nucleoli; but such is not the case: MM. Charles Robin, Leudet, and Potain have all stated, that the glands do not contain any of the elements called cancerous, but only nuclei and lymphatic cells heaped up one on another—that is to say, that in adenia the glands increase in size, but the connective tissue of the gland retains its natural disposition: there is only a hypergenesis of cells and lymph nuclei, which cells and nuclei, nevertheless, are perfectly normal. Such was the conviction of Drs. Laboulbène and Titon when they studied the structure of the glands of a patient, who, in 1852, was admitted into the Hôpital Sainte-Marguerite, under the care of Dr. Marrotte, to be treated for adenia, an affection, which, at that period, Dr. Laboulbène designated in his notes as "general adenitis" [*adénite généralisée*].

Up to that date, no one had injected the afferent and efferent lymphatic vessels: injection would probably have shown some interesting peculiarity in the rich parenchymatous network, and in its permeability.

Hypergenesis of the cellular elements of the gland is then, it appears, the principal anatomical fact in adenia: it is the starting fact, the origin, so to speak, of all the secondary lesions. These secondary lesions, however, ought to be more particularly studied in the spleen and liver. Hypertrophy of these organs is hardly observed till the second stage of the disease. The spleen, especially, may acquire a great size: as you have seen, that organ may occuppy the whole of the left side of the abdominal cavity, and descend to the pubes. In twelve cases of adenia which I analyzed, in four only was the spleen increased in size. In the case of Dr. Bonfils, it weighed one kilogramme, and measured 24 centimetres in length by 15 centimetres in breadth. In the case of Dr. Leudet, it measured 26 centimetres in length by 17 centimetres in breadth. In the examination made by Drs. Bonfils and Leudet, there was no observable structural modification; but in Dr. Potain's case, although the spleen scarcely exceeded the normal volume, its section presented a uniform bright red color exhibiting the cut surfaces of very many *white* points of the size of hemp-seed, and very apparent whitish trabeculæ. The white points presented granules of a slightly irregular rounded shape, of feeble consistence; and which were constituted by nuclei in all respects similar to the nuclei found in the lymphatic glands.

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* Potain: Bulletin de la Société Anatomique, 1861, p. 220.

In no case did the liver present any remarkable alteration. Generally, nothing more was discovered than slight hyperæmia of the organ, without any alteration of capsule, stroma, or hepatic cells.

No microscopic or chemical analysis was made of the contents of the receptaculum chyli, nor thoracic duct, the great lymphatic trunk of the right side. It would have been important to have discovered whether, in these different localities, there was any diminution or augmentation of the lymph-globules, or any alteration in their form and color. Be that as it may, observers have not noted, post mortem, any modification in the blood found in the heart and great vessels. Microscopic examination instituted at the beginning of the last stage of the disease, has established the non-existence of leucocythæmia. Hence, it is exceedingly probable, that there is no increase in the lymph-globules passing into the superior vena cava; and consequently, that the lymphatic ducts leading to that vein do not contain these globules in excess: it is even possible, that they are much less numerous than in the normal state.

The conclusion from all these facts is that, although the affection has its seat almost exclusively in the lymphatic glands, it is only in some exceptional cases that the intestinal glands share in the cellular hypergenesis constantly met with in the lymphatic glands. I ought, however, to mention that, in a case of adenia, reported by Dr. Hallé, which occurred in the hospital practice of M. Nélaton, the liver was studded by innumerable small white bodies cancerous in their general appearance, and varying in size between a lentil and a hazel-nut. The spleen, which was enormously enlarged, presented, we are told, a great number of soft cancerous masses, of the size of a walnut, the white color of which contrasted with the red of the tissue of the spleen. It is not stated that the white masses found in the liver and spleen were demonstrated to be cancerous on microscopic examination, so that we are entitled to doubt that they were of that character. This doubt is the more allowable, as similar masses found in the spleen of Dr. Potain's patient presented, under microscopic examination, a structure identical with that of the lymphatic glands of the same patient. I am inclined to believe, that a similar statement is applicable to the so-called cancerous masses met with in Dr. Hallé's case.

Gentlemen, we have now reached the most interesting part of the subject of adenia—its *etiology*. Struck by the general affection of all the lymphatic glands, the first hypothesis naturally presented to the mind is the existence of a diathesis. Can we associate the lymphatic affection with any known diathesis? Can we cite in explanation of the general hypertrophy of the glands, the constitutional diseases termed scrofulous, tuberculous, cancerous, and syphilitic? Certainly not; for in infancy, the patients presented no manifestation of the scrofulous diathesis, and the glandular affection was developed at an age when scrofula has lost all its power. Moreover, to prove that neither the scrofulous, cancerous, nor syphilitic diathesis has anything to do with causing the disease which I call "adenia," it will be sufficient to study the glandular affection as a local affection.

Syphilis itself does not explain in a more satisfactory manner the cellular hypergenesis of the lymphatic glands; for if it be true that in secondary pox, the glands may be the seat of chronic inflammation, the inflammation never extends to more than a number of glands; and further, only one patient—Dr. Leudet's patient—had any syphilitic antecedents, and none of the other patients, including the young woman whose case I have reported to you, ever showed any syphilitic manifestation. Let me add, that treatment by mercurial preparations or iodide of potassium, though it may cure the syphilitic gummata as in Dr. Leudet's case, exerted no action on that man's glandular system; nor on that of any of the other patients, who from deficiency of exact information, and in default of knowledge of the cause of the disease, were subjected to that medication.

We are thus constrained to conclude, that there is a new *special* diathesis, the essential nature of which is unknown, which we call the *lymphatic diathesis*. This diathesis may be described as a tendency in certain persons to present, under the influence of a determining cause, glandular engorgements, at first local, and becoming general in from eighteen months to two years. This glandular engorgement, as I have seen, may consist in a hypergenesis of the normal cellular elements of the lymphatic glands, a hypergenesis which in some cases may invade the glandular corpuscles of the spleen and intestine. The patient, consequently, has anæmia and cachexia, unaccompanied by leucocytosis.

Adenia, I have said, is a diathesis which has a determining cause. What is this cause, and what is its most common seat? When we attentively peruse the reports of cases of adenia, whether described by others or observed by ourselves, we are struck by the fact that, in the first instance, only one or two glands have been enlarged: some weeks, or, it may be, two or three months after the appearance of these swellings a veritable explosion of glandular tumors occurs in different parts of the body, while, at the same time, the original tumors rapidly increase in size. In the majority of cases, the submaxillary are the glands which first become affected: sometimes, however, the first seat of the affection is in the axillary or inguinal glands.

Whenever there is an acute or chronic engorgement of glands, we must search in the regions which they depurate for some organic lesion to explain the glandular irritation. This rule, which is absolute, will be found to lead to many important results. It is natural, therefore, in a case of general adenia, to inquire, what local lesion has occasioned the original engorgement. There are many cases, however, in which no light is thrown upon this question: we must be satisfied to note that the engorgement commenced in the axillary, inguinal, or maxillary glands—which is provoking. Viewed along with these incomplete cases, there are others—the cases of Leudet, Potain, and Perrin, and the case of my Stockholm patient—which possess great interest in relation to this question. I have thrice observed that there existed acute or chronic irritation at the great angle of the eye or in the external auditory passage: and observe, gentlemen, that the glands first attacked were situated on the same side as the ocular, nasal, or aural lesion, and that the submaxillary and cervical glands of the opposite side, as well as the other glands of the body, were only secondarily attacked. It is, therefore, well worthy of remark that, in the five cases to which I have referred, there were four with inflammatory lachrymal tumor, chronic coryza, and otorrhœa. One cannot help being struck with this alteration of the skin and mucous membranes, and with the primary glandular alteration. I ought, however, to remind you that, in one of Leudet's cases, and also in the case for which we are indebted to Perrin, the patients stated that the glandular engorgement began in the axillary region. Subsequently, however, MM. Leudet and Perrin discovered submaxillary engorgement, so that we may suppose the possibility of that engorgement having existed at the commencement of the adenia, but to so small an extent as not to attract the notice of the patients.

Be that as it may, it is a fact that, in twelve cases of adenia, there were four in which there existed lachrymal tumors, a chronic coryza, and an otorrhœa.

It is not a matter involved in the least doubt—it is a positive fact—that there is a relation between the primary adenopathia and the superficial lesions of skin and mucous membrane.

As to general consecutive adenia, I cannot understand, admitting certain persons to have a predisposition of such a special nature, that one or two lymphatic glands being engorged for a certain short period, in general of variable duration, but nearly always of recent date, should be the starting-point of the generalization of the malady to the other glands.

I have already told you the effect which adenia produces upon the composition of the blood. The microscope has shown that both before and after death, it contains no excess of white globules or globulines. And although there is no proof that the *quality* of the globules has undergone any modification, the characters of the adenia, at least in the second period of the disease, do not leave the smallest room for doubt as to the *quantity* of the red globules. It is probable, therefore, that in adenia, there is a diminution of the components of the red globules; and that this diminution is very probably the result of general enlargement of the glands.

It has also been observed by Wunderlich that, in a certain number of cases, the adenopathia does not become general till after it has been some time exclusively localized in a few glands. At other times, the affection begins with a general outbreak. Virchow is not in the slightest degree perplexed as to the secondary nature of the generalization of the malady: by his favorite embolism, he explains the metastasis. The explanation of primary generalization is not quite so simple. Moreover, Virchow's metastatic embolism explains nothing in respect of the special affection, because all those—and they are fortunately many—who have enlarged glands have not general adenopathia as a sequel. If the generalization be the exception, it is because it is dependent on exceptional causes. For want of a better name, I call these exceptional causes *specific* causes.

Wunderlich (much more the clinical physician than Virchow) is of opinion that adenopathia, whether it become general on the first manifestation of the disease, or consecutively, is one and the same thing; and that it is a constitutional affection, just as cancer, whether it be general at its first outbreak, or do not become so till a subsequent period, is in both cases a constitutional affection. This statement does not imply that Wunderlich and I are acquainted with the essential nature of adenia. Is there anything of which any one knows the *nature?* It is only *effects* that are known.

But Wunderlich goes further: considering that there is no difference, except in the state of the blood, between leucocythæmia and adenia (which he calls pseudo-leucocythæmia) he asks—Whether adenia be not an early stage of leucocythæmia; and whether the difference between the two affections does not consist in a diversity of accessory causes, by which there is produced a great quantity of white globules in one case, and no white globules at all in the other case? Wunderlich explains the nature of this accessory cause: in leucocythæmia, he says, that there is hyperplasia of the interstitial connective tissue in the stroma of different organs, such as the liver, spleen, kidneys, and intestines; and as the lymphatic vessels originate in the plasmatic network of the connective tissue, the cellules, as they multiply, may enter the lymphatic vessels, and so get into the blood. This would explain the profusion of white globules in leucocythæmia, in which we find these conditions.*

I confess that till more ample information be obtained I shall continue to look on leucocythæmia and adenia as two distinct affections, although very nearly approaching one another in respect of their lesions. To my mind, the fact of the progress of the disease being very different in the two cases is decisive of the question.

Does the study of adenia in which we have now been engaged furnish practicable therapeutic indications? Hitherto, no system of treatment which has been employed seems to have yielded a satisfactory result: it is well to state, however, that in three cases the waters of Kreuznach and Lavey and sublimate baths appeared, at least temporarily, to retard the progress of the disease. In the first case, the German waters reduced the volume of the engorged glands. A patient for whom I prescribed sublimate baths did not, for some months, appear to suffer in his general health from the morbid alteration of the glands. M. Cossy reports the case of a patient, aged 53, affected with general hypertrophy of the glands, without leucocythæmia, whose condition was remarkably ameliorated by a two months' course of the waters of Lavey. In this particular case, however, the treatment was complex, for the waters were given internally to an extent sufficient to produce slight purging; hot and cold douches were played upon the glandular tumors as well as on the body generally; the tumors were also regularly subjected to shampooing; and the patient took daily three of Blanchard's iodide of iron pills.

This treatment—on several occasions temporarily diminished in activity—led to amelioration, which began to show itself after the thirtieth day. The treatment was continued for two months; and when the patient left Lavey there was diminution, to the extent of one half, in the volume of the engorged glands. In this particular case resolution was more attributable to the physical process of applying the mineral waters than to the iodides and chlorides which they contain in small quantity, and which they impart to the mother water of the saline waters of Bex, with which they are generally mingled.

If in the case now related the amelioration ought in part to be attributed to the action of the iodides and chlorides, it would perhaps be advisable to prefer to the waters of Kreuznach and Lavey those of Saxony, which (without the addition of the mother water) contain, according to Dr. Aviolat, the enormous quantity of thirty-three grammes of iodides and ten grammes of bromides in a bath of three hundred litres.

The general indications of treatment are furnished by the state of the glands, and the anæmia. Whenever we find primary engorgement, and recognize its cause, we ought to use every means at our disposal to remove the primary irritation; but when there are already numerous glands manifestly engorged, we must not hesitate to endeavor to modify the general morbid state of the patient, either by giving repeated saline purgatives, or mineral purgative waters, such as those of Kreuznach, Lavey, and Saxony, or other waters of similar character. It is necessary at the same time to employ local treatment, by douches and shampooing, to induce absorption of the superficial tumors. Finally, preparations of iodine, iron, and cinchona, will have the double advantage of modifying the general state of the system and combating the anæmia, which is apt to make rapid progress.

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* **Wunderlich**: Pseudoleukæmia, Hodgkin's Krankheit, oder Multiple Lymphadenome ohne Leukæmie. [*Archiv der Heilkünde*, 1866, p. 581.] See also: **Paul Spillmann**, in the Archives de Médecine for August, 1867.

I have, I confess, gentlemen, only a moderate amount of confidence in this complex treatment of a disease which seems to me to have a specific character; but it is our duty, when we are without a specific remedy for a specific disease, to use every effort to counteract the disease by other medicines, even when it must be admitted that they only influence symptoms.

Gentlemen, I must not omit saying a few words regarding a glandular affection which I have very often observed in young Creoles, and particularly in Creoles from the Mauritius and the Island of Reunion. I cannot, however, be sure that it is not due to mere chance that I have seen this particular form of adenia in young persons born in the two colonies I have named, and never once in persons born in the French or English West Indies.

In adolescence, and more frequently in boys than in girls, we see the superficial and deep inguinal glands become swollen, sometimes only on one side, and at other times simultaneously on both sides. The disease makes its appearance in paroxysms, lasting for one, two, or three months, separated from one another by intervals, it may be of several months. An attack of greater violence than its predecessors then sets in, and some of the glands suppurate. In certain cases the suppuration extends to several glands, and in succession to the entire mass of glands. The patients are consequently condemned to a long confinement to bed or bedroom. Sometimes the suppuration goes on for a year. It sometimes happens, very seldom, fortunately, that perinephritic abscesses form. This occurred in a case of which I shall have to speak when I come to treat of that subject. There may be a band of glands suppurating from the groin up to the kidney, with large abscesses forming round that organ. You can appreciate the danger involved in such an occurrence.

In the majority of cases which have come under my observation, the disease has ceased at the age of manhood, without my being able to attribute much benefit to the medical treatment employed.

LECTURE XCII.

AMENORRHŒA AND MENORRHAGIC FEVER.

Menorrhagic Fever.—Amenorrhœa from Change of Residence does not call for any Treatment; or at least there are no Special Indications of Treatment.—Menstruation consists of Two Parts, viz.: Ovulation, and Hemorrhagic Flux from the Mucous Membrane of the Fallopian Tubes and Uterus.—Amenorrhœa from Chlorosis and from Anæmia.—Amenorrhœa consequent upon Disease, Acute or Chronic.—Therapeutic Indications derived from the State of the General Health.—Therapeutic Opportunity.—General and Local Bloodletting: Hot Baths: Iodine: Emmenagogues.

GENTLEMEN: An error in diagnosis, which you saw me commit the other day, affords me an opportunity of entering into some details regarding an affection to which I give the name of menorrhagic fever [*fièvre ménorrhagique*].

A young woman of 17, who had been ill for six days, was admitted as a patient to St. Bernard Ward. She was born in the country, and had only resided four months in Paris. Like most new-comers, she did not menstruate. From the commencement of her indisposition she complained of headache, giddiness, and insomnia. She had epistaxis, loss of appetite, foul tongue, and diarrhœa. The pulse was febrile, and there had been no cessation of fever. I concluded that the case was one of dothinenteria. On the following day the catamenia appeared, and the fever abated. The menstruation was normal; and two days after the appearance of the sanguineous discharge, health was completely re-established. Gentlemen, this is not the first case of the kind which has presented itself in our wards: not a year passes without my calling your attention to such cases, of which sometimes I am led to form an erroneous diagnosis.

When the menstrual function takes place every month in a regular manner, it is generally attended by discomfort of unimportant character; there are, nevertheless, headache, and a modification of the different functions which remind us of the febrile disturbance caused by slight indispositions: this arises from monthly ovulation being pathological to a certain extent, the turgescence of the ovaries and uterus, and rupture of the Graafian vesicle, constituting a sort of morbid process affecting some individuals more than others.

In many women, as you know, there supervenes not only the discomfort to which I have referred, but likewise symptoms of real fever: this is not to be wondered at when we reflect upon the individual peculiarities so frequently met with in practice.

Some persons, with a slight sore throat, a boil, or an inflamed superficial gland, have violent fever, even sometimes accompanied by delirium. It is, therefore, not in the least surprising that, in exceptional circumstances, the process of ovulation should be accompanied by pretty severe febrile paroxysms.

We know, likewise, that the economy, be it ever so excitable, becomes quite habituated to morbid impressions often repeated in a similar manner. If, however, the same morbid impression recur after a very long interval, it is much more severely felt: on the other hand, in respect of menstruation, the phenomena attendant upon congestion and hemorrhage are in general aggravated in proportion to the length of time during which the function has been suspended. There exists then a twofold reason for the menorrhagic fever being more violent.

Having been led to discuss this subject, I must not neglect the opportunity of speaking to you of amenorrhœa and its treatment. You have seen me so often employ very different forms of treatment, that you must naturally be disposed to ask me to explain my apparent versatility, and to declare the therapeutic principles which guide me.

I stated at the beginning of this lecture, that it is a frequent occurrence for young girls on arriving in Paris to have suppression of the catamenia. Change of residence is enough in itself to produce this result, without there being any change in the manner of living. Young girls who have lived a number of years in a provincial boarding-school, on moving to a similar institution in Paris, where the *régime* is evidently the same, often experience an interruption of several months in their courses: and young girls removing from Paris to the country similarly suffer. We constantly have in our wards servant-girls from the country, who, for some months after their arrival in Paris, have ceased to menstruate. Here, then, is amenorrhœa from a cause for the removal of which we are not called upon to do anything, unless unfavorable symptoms arise.

Some girls begin all at once to menstruate regularly: others, and they are probably the majority, menstruate very irregularly for two or even three years, without seeming to have the slightest derangement of health. This state is often a cause of anxiety to mothers: but the most prudent course to follow under such circumstances is to abstain from all interference, at least until some disturbance in the health render it necessary to institute decided treatment.

I need not tell you that, in acute diseases, menstruation is only slightly modified (as my colleague M. Hérard has shown), but that, on the contrary, in chronic maladies, it first becomes irregular and then, in the majority of cases, ceases altogether.* We can understand why a very strong morbid antagonism should powerfully modify ovulation, a function only occasionally in exercise, when we see that it disturbs those functions which are more immediately necessary for the maintenance of life.

Suffering women too frequently attribute aggravations of their maladies to suppression of the menses. Though, sometimes, acerbations arise under the influence of an abortive menstrual congestion, yet the disturbance thereby induced is generally transient and unimportant. There are cases, however, in which menstrual congestion may be regarded as the cause of severe symptoms in young girls of hæmoptysic tendency. Whilst the economy is preparing for the great work of ovulation, which constitutes so important a part in a woman's life—as important in her as the work of reproduction is in every animal and vegetable organized species—it produces an excitation which pervades the whole system, and gives rise to fluxions and hemorrhages, the latter being particularly apt to occur in woman. Women, likewise, often have headache, swelling of the breasts, and hæmorrhoidal congestions: epistaxis is also usual, and hæmoptysis, in those of tubercular predisposition. There is greater reason to fear accidental, when there is a cessation of the normal hemorrhage.

Although physiologists allege menstruation to be always connected with an act of generation—abortive or non-abortive—in this sense that it is always preceded by the development and rupture of an ovule—clinical observers must protest against so absolute a statement. Under the influence of some mental emotion, or some morbific cause, we often see the catamenia reappear some days after they had ceased; and in such cases, it is difficult to believe in the existence of a preparatory ovarian work. Sometimes, the blood appears a few minutes after the mental cause by which the woman has been affected, just as occurs in epistaxis. Although I do not dispute that there is a relationship between ovulation and hemorrhage from the uterus, I am disposed to consider the latter as a simple coincidence, determined especially by modifications of the uterine nervous system, similar to those produced by many other causes.

The study of the phenomena which precede each menstrual period has an important bearing on the treatment of amenorrhœa, for a reason on which I will afterwards enlarge, and which for the present I will merely mention. The reason is this: once the catamenia have been suppressed, it becomes impossible to calculate their return from their usual and normal epochs. I now recur to the phenomena of which I was speaking.

Pretty generally, menstruation is preceded by a certain change of disposition, not always very appreciable by the physician, but recognized by those in habits of intimacy with the woman. Frequently, also, there is a feeling of general discomfort and loss of appetite. In a very considerable number of cases, one or several small pustules of acne appear on the lips and chin. These are the primary signs of a menstrual period, which are apart from the organs of generation. The other more important precursory signs specially belong to the reproductive organs: I refer to turgidity of the mammæ, which become painful, and the lobules of which are more distinctly felt than at other times: also, to heat in the sacral region, sensation of weight in the loins, slight leucorrhœa, frequent desire to make water, and (in many who are generally constipated) to a tendency to diarrhœa. I need not add, gentlemen, that in most chronic diseases, there is a slight exacerbation of the symptoms, which, in many cases, has a positive significance.

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* HÉRARD: Mémoire sur l'Influence des Maladies Aigues sur les Régles. [*Actes de la Société Médicale des Hôpitaux de Paris, fascic. 2me*, 1852.]

You will at once perceive, gentlemen, the importance of questioning, with very great minuteness, women suffering from amenorrhœa, so as to elicit from them the presence or absence of the precursory signs which I have now succinctly noticed. It is necessary clearly to bear in mind that it is exceptional for hemorrhage from the uterus to occur at other times than the menstrual periods. It is vain to attempt to cause a return of the menses after they have ceased eight days—for such a purpose it would be useless to employ any imaginable diversity of measures; on the other hand, you would find it a matter of extreme difficulty to adopt any medication sufficiently bad to prevent their reappearance at nature's appointed term. If it be so difficult to induce the sanguineous discharge in absence of the normal preparative changes, what success can we hope for from giving any so called emmenagogue to a woman suffering from amenorrhœa?

Opportunity is as important a consideration in pathogeny as in therapeutics. A man has just had an attack of gout—not his first attack: consequently, he is a gouty subject. If he have got quite over the attack, he may commit excesses at table or with women, or keep late hours, without immediately inducing a new paroxysm of gout. But when the gouty principle has remained pent up for a long time, when it is ready prepared accumulated within the system, the slightest exciting cause will be sufficient to produce a violent attack. I may say the same in respect of herpes, megrim, asthma, and other diathesic diseases. A person when sweating profusely may get drenched with icy rain, or he may ten times rest in a marsh exposed to cold and damp, without suffering on return home the least pain, or even without a cold in the head. Eight days later, however, a half-open window may give him inflammation of the lungs or acute rheumatism. This is explained by there being no predisposition in the first, and in predisposition existing in full plenitude in the second state. In the same manner, you can understand that the general preparation in the economy which terminates in menstruation increases, and accumulates from the cessation of one catamenial flow up to the commencement of another; and also, that the influence of medical treatment will be quite different according as the measures are employed to-day or three weeks hence.

Emmenagogue treatment, gentlemen, is a very complicated affair; and it may be said, that if there be a method of treatment deserving of the epithet *emmenagogue*, there are very few medicines entitled to that appellation.

Do you suppose that you treat amenorrhœa in the same way in a strong plethoric woman, as in a subject of extreme anæmia? Do you suppose that if the morbid antagonism of inflammation of a lung or of any other organ has caused suppression of the menstrual flux, you ought to proceed as you would in the case of a young woman whose courses have been abruptly stopped by putting the feet into cold water? Gentlemen, the mention of these well-known matters is sufficient to point out to you the difficulty of the treatment.

In women and in men, there are necessary functions in constant operation: as the cutaneous, renal, and hepatic secretions go in uninterruptedly, you can understand that sometimes the physician requires to do very little to excite these different secretions, the system being always in a state of readiness to perform them. There exists a continuous functional aptitude, which can be augmented by means very slightly active.

But menstruation is an intermittent function—transient, and, in a sense, accidental. So numerous are the circumstances which disturb it, that it is easy to see that therapeutic difficulties have to be encountered when it is wished to augment or regulate it. This function is not so essential to the adult woman that it must be performed: it is also easily disturbed when the general functional harmony is seriously deranged. When this general derangement exists, it is useless to attempt to restore the menstrual function by emmenagogues. The primary indication is to restore the equilibrium; and then the special excitant of the uterus becomes an important weight in the balance. The menstrual function will be tardy in re-establishing itself, not only so long as an antagonistic flux continues, as is the case in certain acute and chronic phlegmasiæ, but also in the diathesic diseases (such as chlorosis, albuminuria, and diabetes), which exceedingly alter the constitution of the blood and disturb the harmony of the nervous system.

If in chlorosis, for example, the permanent and necessary functions, such as calorification, innervation, diuresis, and diaphoresis, are so strangely and inveterately perverted, what must it be in respect of an accidental function such as menstruation?

It is, therefore, gentlemen, too evident to require further remark, that if the menstrual flux be prevented by the antagonism of a fever or a phlegmasia, the sole object of the physician must be to combat fever or phlegmasia. Should plethora be the cause of the evil, it is by diminishing the mass of blood, and reducing its plasticity, that the uterine flux will be facilitated; whereas, if the functional derangement be attributable to chlorosis, the proper treatment will consist in administering ferruginous preparations and tonics. These different means, so opposite in their nature to one another, are not emmenagogues; and yet they are the most powerful agents in emmenagogue treatment. As this statement may seem a little obscure and odd to some of you, let me explain it, so that you may quite understand my meaning.

The better the health, the greater the regularity with which all the functions are performed; and if the advent of disease accidentally excite serious disturbance of the functions, their restoration to a normal state will be brought about simply by restoring the general health. In fact, cessation of the causes of disturbance is sufficient to enable the system to return to its right working order. The treatment which effects the cure is not an excitant of the disturbed function: it simply restores order, and so enables the laws which preside over the economy to resume their empire unhindered by any obstacles. Thus it is that tartar emetic, digitalis, quinine, iron, bloodletting, and many other remedies apparently so opposite in their properties, prove themselves to be emmenagogues, in the same way that they act as excitants of the secretion from the lungs, kindeys, and liver, in virtue of their power to restore the general health. They have no special properties. But, gentlemen, the health being re-established, there may still remain some functional incertitude, some hesitation in the functions to resume their usual course; and it is in such circumstances that it is useful to employ special excitants such as I am now going to consider.

There is no excitant emmenagogue with which I am acquainted so efficacious as the general warm bath: to impress this fact on your minds, it is sufficient to remind you, that after a somewhat protracted hot bath, nearly every woman experiences uterine congestion, manifested by lumbar pains, a feeling of weight at the lower part of the abdomen, leucorrhœa, and an increase in the occasional flux, which often appears before the normal epoch. To obtain, however, the desired result, the bath must be given, at least, thrice a week, and repeated daily when the menstrual period is imminent. By and by, I will recapitulate the signs which I have already mentioned; and on which I cannot too much insist.

Bleeding from the arm, when the precursory menstrual signs exist, is a measure of immense potency; and it is not unusual for the uterine flux to appear an hour after the bloodletting. I need not tell you, gentlemen, that this heroic treatment would scarcely be opportune in chlorotic women, or in those who have long been sufferers from chlorosis.

The application of leeches ranks next after bleeding from the arm; and as it causes less alarm to patients and their relations, it is a measure which, in general, is much more easily accepted. This, however, is a matter regarding which some explanations are required. It is very essential to know the number of leeches which ought to be employed, and the place to which they ought to be applied. First, I will speak of the place of application.

Some physicians think that the leeches ought to be applied to the labia majora, basing their opinion on I know not what theory, and also, perhaps, on the practical fact, that the application of a small number of leeches to any particular place, determines a great congestion to it. The proceeding is apt to occasion great inconveniences: it very often leads to local engorgements, furuncles, and boils. It has also, when the bites are healing, the serious drawback of exciting intense itching, which may sometimes occasion troublesome sensations in girls, leading them to contract bad habits. I now always apply the leeches to the inside of the knees; and I have never seen this practice prove less efficacious than the other. It possesses, moreover, the advantage of enabling the physician to arrest the bleeding, with the greatest ease, by making pressure on the condyles of the femur or the head of the tibia: and this is the more necessary that the application of leeches acts much less by subtracting blood, than by causing congestion. So convinced am I of this, that I invariably advise the bleeding to be stopped by agaric, as soon as the leech falls off; and I obtain equally good emmenagogic results, the patient having only lost a few grammes of blood, a consideration of great importance. This practice is repeated on three successive days. Should the first application cause the menstrual flux to appear, I do not prescribe a second: should the flux stop, I order another application of leeches to be made.

It is quite true that the subtraction of even this small quantity of blood enfeebles chlorotic subjects; but it is a remarkable fact, that the reappearance of the flux in a natural manner, and with increased abundance, is often a precursor of a restoration to health. Notwithstanding the enfeebling which this treatment causes, I do not the less recommend it, because it really renders very great service.

When the stomach will bear it, the tincture of iodine ought to be administered three times a day, from five to fifteen drops being given each time in a weak infusion of saffron: this is a powerful emmenagogue. It ought to be taken continuously for several weeks.

You have so often heard of iron as an emmenagogue, that it is incumbent on me to make some remarks to you on that subject.

On chlorotic subjects who have amenorrhœa, iron seems to act as an emmenagogue. On the other hand, when they have menorrhagia, iron acts as a hæmostatic, which shows that this therapeutic agent is neither an absolute hæmostatic, nor emmenagogue, but a medicine which seems either to suppress or cause menstruation by re-establishing the health under normal conditions—conditions under which menstruation ought to take place.

By this very summary enumeration of the chief measures which ought to be adopted to restore menstruation, you may have observed that iron, the last which I have mentioned, is not absolutely an emmenagogue; so little has it the properties of an emmenagogue, that in a healthy woman, it rather diminishes than augments the monthly flux. It ought, consequently, to be considered as a *relative* emmenagogue: the others are *absolute* emmenagogues, as they will, in certain states of a woman's health, augment or cause the menstrual flux, provided there be not a too frequent repetition of the bloodletting of which I have spoken.

You recollect the remarks I made a little while ago on the necessity of considering *opportunity* in employing this or that remedy: never is this more absolutely necessary than in emmenagogue treatment. Under ordinary circumstances, neither iodine, bloodletting, nor baths, will cause a return of the menstrual flux when it has ceased; but if the preparatory work has begun in the system, these measures will possess great emmenagogue power. In amenorrhœa, from menstruation not having occurred a long time, there is no way of recognizing the signs of the precursory work going on, and no way, consequently, of recognizing the *opportunity* for employing emmenagogues.

However, gentlemen, by exercising vigilant attention, the physician may find out the exact time for prescribing emmenagogues.

Before speaking of the signs by which to recognize the opportune time for action, let me remove a prejudice, which I regret often to see possessing a strong influence on the minds of physicians. I do not suppose that there is a single medical practitioner who believes in lunar influence, and is not perfectly aware that out of one hundred women, there is not one perhaps in whom the menstrual flux corresponds with the same phase of the moon. Many women maintain that they have their courses on particular days of the month, for many months in succession. However, on coming to exact count and reckoning with them, when they have been obliged to note the exact dates of a series of epochs, it is easy to satisfy oneself, and not difficult to convince the women themselves, that there is very little foundation for their assertion. This, gentlemen, is a matter of some importance: in place of reckoning the epochs by the revolution of months, it is necessary, in each case, to compute the return of a period, by the time which, in the particular individual, has been ascertained to separate one period from another. This is an important matter for observation, when it is proposed to administer emmenagogue remedies at the epochs when the catamenia are presumed to be due. But we must clearly bear in mind, that computation is impossible when there exists amenorrhœa of some months' duration. The suppression of the menses which occurs during pregnancy is purely physiological: it has, moreover, this curious feature, that during the first three or four months most women have very distinct menstrual signs; and experienced accoucheurs are well aware that abortion generally takes place at a menstrual period; accoucheurs, also, from a dread of the fluxionary phenomena which thus appear, enjoin absolute rest at these periods upon women liable to miscarry. But when the amenorrhœa is not caused by pregnancy, the fluxion to the organs of generation at once loses its usual regularity, and no longer furnishes data whereby to calculate the probable return of the courses; and experience teaches us that the sanguineous flux may reappear at very irregular, and, indeed, at quite indeterminate intervals.

I required, gentlemen, to enter into these details, to enable you to understand the utility—I would even say the necessity—of instituting treatment at one time, and avoiding to institute it at another. I have already told you, the approach of menstruation is announced to women by a certain amount of discomfort, by altered disposition, by swelling of the mammæ, by leucorrhœa, and by a frequent desire to pass urine. Both physician and patient ought to have their attention constantly directed to these phenomena, because it is at the time of their appearence, and only then, that the more direct excitants of the catamenia find their opportunity. Then it is, that the application of a few leeches, or such a bleeding from the arm as I have spoken of, brings on the menses. Then it is, that the tincture of iodine, saffron, and ammonia, produce effects which are decidedly emmenagogue. Then, also, it is that protracted baths will prove most efficacious.

When the signs of fluxion have passed, we must not continue the use of these means; we must wait for a new indication to employ them, and take special care to make use of them during the increase, and not during the decline of the fluxionary condition. When there is nothing in a woman's state to indicate congestion of the ovaries or uterus, the time is not so opportune for the physician's intervention. Then it is necessary, without any reference to the periods, as they are not recognizable, to proceed with continuous medication, employing such remedies as iodine and iron, taking care, of course, that there exist no contraindications to their being used.

Some physicians have recommended as a more direct measure for causing menstruation the establishing each month an artificial menstruation, a proceeding which has its advantages. In pursuing this plan, we must bear in mind the normal interval which, in each individual we have to treat, separates one period from another. For four or five successive days a very prolonged hot bath ought to be given in the morning; and there ought to be introduced into the rectum, in the evening, a suppository containing five centigrammes of tartar emetic, or twenty-five centigrammes of rue or sabine. When a fluxionary movement has been excited by these measures, a leech ought to be placed to each knee for three days in succession, care being taken to stop the bleeding as soon as the leech falls off. On the following month this treatment is repeated; but it is very necessary to bear in mind that nature does not easily respond to our injunctions, and that, frequently, the return of the fluxion which indicates coming menstruation will occur during the interval between the epochs which we have determined to be the menstrual epochs. When this occurs, we must follow the plan which I have just been pointing out.

LECTURE XCIII.

PELVIC HÆMATOCELE.

Physiological and Pathological Anatomy of Pelvic Hæmatocele.—Catamenial Hæmatocele: from Hemorrhage into the Fallopian Tube; Excess of Fluxion, or Deviation in the flow of the Sanguineous Discharge is frequent, slight, and often recurs.—Accidental Hæmatocele from Ovarian Hemorrhage, Alteration of the Parenchyma, or Varix of the Organ, is a rare and almost always a mortal malady.—Hæmatocele from Blood Ascending from the Uterus by the Fallopian Tube, and being Effused into the Peritoneum.—Cachectic Hæmatocele.—Hæmatocele caused by Alteration of Blood.—Tubal Hæmatocele.—Diagnosis: Tumor behind or around the Uterus.—Intra-peritoneal, Catamenial Hæmatocele.—Extreme Pallor.—Slightness of Peritoneal Pain.—Intra-peritoneal, Accidental, or Ovarian Hæmatocele: Slight Hemorrhage from Rupture of the Hæmatic Pouch: Acute Peritoneal Pain.—Extra-peritoneal Hæmatocele: Slight Pain and slight Hemorrhage.—Differential Diagnosis: Phlegmon and Abscess of the Lateral Ligaments, Extra-uterine Pregnancy, Hydatid Cysts of the True Pelvis.—Treatment: Surgical Intervention to be avoided.

Gentlemen: The case to which I wish to call your attention is that of a girl of sixteen, who has been admitted to St. Bernard Ward. She has only menstruated once, and that was two months and a half ago when resident in the country. She came to Paris, two months ago, in the capacity of domestic servant: she was then jaded, and had suffered from mental distress. On November 7th, the day of her admission to the hospital, she had the symptoms of continued fever. Two days later, there was no longer any doubt as to the nature of the febrile malady: the tongue was dry, the teeth were covered with sordes, and there was diarrhœa: the pink-colored lenticular spots were seen: there was pain in the abdomen, particularly in the right iliac fossa. The other symptoms were—a rapid pulse, drowsiness, low delirium, and mucous râles in the chest.

Towards the end of the second week she had a better appearance, but it did not last long: she soon fell into a state of deep adynamia; and on Thursday, 21st November, that is to say, about, probably, the eighteenth or nineteenth day of the disease, she died.

At the autopsy, we found an effusion of reddish serosity, free from clots, in the pelvis. In the midst of this effusion was a tumor as large as a hen's egg. It belonged to the right ovary, and did not adhere to the neighboring parts: there was no trace of peritonitis. The effused serosity weighed about from one hundred and fifty to two hundred grammes.

The right Fallopian tube had no adhesions: its fimbriated extremity was much injected: in the cavity of the tube, about its external third, there was a small quantity of sanious, gray-colored, muco-purulent fluid. There was a tumor on the ovary resembling an egg in size and form: it was brown, smooth on the surface, and without false membranes. On its descending part, there was a projecting clot the size of a pea, which was hard, to a great extent fibrous, and was strangled by an ulceration across which it was continuous with another clot situated in the centre of the tumor. On making an opening in the posterior part of the tumor, it was ascertained that we had to do with a hæmatic cyst, which had contained a very large clot, which had partly escaped by the ulceration. Before opening the cyst, it was remarked, that the red serosity exuded drop by drop from the ulceration, which was imperfectly closed by the clot. The hæmatic cyst, therefore, was the source of the intra-peritoneal effusion, which most probably was effused during the last hours of life, as there was no trace of peritonitis.

The walls of the cyst were formed by the serous membrane of the ovary, lined internally by several very thin layers of fibrin, yellow, and easily detached. The cyst rested on the ovary, and was in direct communication with the cavity of a Graafian vesicle, the walls of which were velvety, and the seat of the original apoplexy. In the same ovary, other Graafian vesicles presented small apoplexies. It appears, then, that we surprised a retro-uterine hæmatocele in progress of formation, consequent upon rupture of a hæmatic cyst of the ovary.

Let me add, that there was no trace of ovarian pregnancy: I discovered no vestige of a fecundated ovum in the cyst, which only contained serosity, clots of blood, and fibrinous deposits. The uterus was perfectly healthy, and its neck was untorn. The hymen was intact.

I believe that the ulceration of the walls of the hæmatic cyst was dependent upon the state of the patient's general health.

The glandular patches of Peyer presented large, deep ulcerations, in some of which the reparative process was in progress.

In listening to the account of this autopsy, you must have been struck with the special anatomical lesion—the ovarian hæmatic cyst, with its ulcerated wall. I remarked that a sanguineous intra-peritoneal effusion was found; and this must have led you to form a correct view of those peculiar effusions, to which much attention has been directed during the last ten years, and to which has been given the name of *retro-uterine hæmatocele.*

Bear in mind this fact: but beware of supposing that it is the rule, and that every hæmatocele has its origin in a hæmatic cyst of the ovary.

I consider that there are two principal kinds of hæmatocele. One of them has its origin in the ovary, which I, therefore, call *ovarian* hæmatocele: this I look on as the least usual form, although it affords the most frequent opportunities of studying the pathological anatomy of the affection. The other may be called *tubal*, or better still, *catamenial*, a name which recalls the fact that the hæmatocele originated in a hemorrhage, during the catamenial flux, into the mucous membrane of the tube or its pavilion. This is the most common form, and may be seen several times in the same patient. It is not so serious as ovarian hæmatocele; and as it seldom has a fatal termination, little is known regarding its pathological anatomy. This circumstance, likewise, explains why the existence of this kind of hæmatocele is less generally admitted.

I long believed that I was the first to describe catamenial hæmatocele: indeed I had been for a long time under the impression that it had never been mentioned by any one else when my lectures were published in the Gazette des Hôpitaux in 1858; but in 1860, a prior claim to the theory was made. I am far from complaining of having been preceded by a distinguished observer: the theory will not be looked on as less sound because I am not its sole defender: I regret, however, that the honorable clinician to whom I refer should have vindicated his title to priority with so little moderation. In any case, I am far from adopting an exclusive explanation of the manner in which the hemorrhage takes place in hæmatocele: this statement is sufficiently proved by my division of hæmatoceles into ovarian and catamenial.

The etiology of hæmatocele is dominated by a great function: that function is menstruation. In catamenial hæmatocele, the exaggeration or deviation of the function may be said to constitute the disease: the hæmatocele is only a symptom, and the hemorrhage is the necessary and immediate consequence of the modification of function. Increased determination of blood to the tube and its expanded extremity produces either sanguineous exudation or rupture of a vessel; and the catamenial blood is poured into the cavity of the peritoneum. The effusion is often considerable, and may give rise to the symptoms of internal hemorrhage. In ovarian hæmatocele, the ovary is diseased in the first instance, and rupture of the softened organ, or of a hæmatic cyst, may be only an accident which will be often found to have menstruation as its determining cause; and at other times external violence, such as a strain, a fall, or the jolting of a carriage. These causes may likewise lead to the rupture of the utero-ovarian veins, if they be varicose.

Another kind of hæmatocele may result from reflux of the catamenial blood from the uterus towards the tube, and its subsequently passing into the peritoneal cavity. This will be observed in cases of excessive menorrhagia, or when there exists an accidental or congenital obstacle to the natural exit of the blood from the uterus into the vagina. Dr. Bernutz has ably supported this theory of hæmatocele, admitting, however, at the same time, that there are other forms of the affection.*

There is yet another form of hæmatocele which merits special mention, although in the cases upon which its description is founded, the hemorrhage has often been incomplete, remaining in the tubes without passing into the peritoneum. The term *cachectic* is applicable to this form, because it is met with in cases of purpura, malignant jaundice, scarlatina, measles, and small-pox; that is to say, in circumstances in which the blood is modified in its constitution in such a way as to have a great tendency to exude by the mucous membranes. Intra-tubal sanguineous tumors, unconnected with previous fecundation, occurring during menstruation, or at a longer or shorter period after the menstrual epoch, testify to the facility with which the mucous membrane of the tubes may become the seat of hemorrhage. In the cases of Barlow, Simpson,† Hélie (of Nantes), and Laboulbène, there was cachectic tubal hemorrhage which might have passed into the cavity of the peritoneum, as was observed to have taken place in the cases reported by Scanzoni‡ and Barlow.

The etiological fact to which I wish specially to direct your attention is the determination of blood to the genital organs at the time of menstruation. This determination which presides over the reproductive function is not met with exclusively in the higher orders of organic life: it is met with in the lower classes of animals and in plants. The generative act is always accompanied by a very remarkable fluxion. In spring, the young shoots of plants are the seat of a special congestion. This afflux of sap at a determinate time has no other end than the development of the shoot destined to bear the flower. We also see an afflux of the juices towards the delicate organs of reproduction till the time has come for the pistil to receive the pollen secreted by the stamina. The flower is then full-blown: all its parts are rigid; but immediately after the great act of reproduction has been accomplished, as soon as the perpetuation of the species has been assured, the congestive afflux of sap disappears, and all the organs soon become withered.

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* Bernutz: Clinique des Maladies des Femmes.

† Simpson (James Y.): On Vesico-uterine, Vesico-intestinal, and Utero-intestinal Fistulæ, as results of Pelvic Abscess. [*Edinburgh Monthly Journal*, for October, 1852.]

‡ Scanzoni: Traité Pratique des Maladies des Organes Sexuels de la Femme. No. 304. Paris, 1858.

In the lower animals, we also see congestion presiding at the fissiparous and gemmiparous processes.

In the human species, the principal functions cannot be performed without a considerable afflux of blood to the organs. Thus, during long-continued intellectual occupation, there is a flow of blood to the head: during mastication and stomachal digestion, there occurs a profuse secretion of saliva and gastric juice, which can only be explained by congestive fluxion of the salivary glands and glands of the stomach.

During ovulation, this afflux is still more remarkable. In that act, congestion proceeds to hemorrhage: at each spontaneous ovulation, there occurs rupture of a Graafian vesicle, and each rupture is accompanied by a slight hemorrhage which becomes the origin of an ovarian hæmatic cyst, of the nature of those which have been so well studied by Charles Robin.* Generally, this hemorrhage is slight, the process of enucleation of the ovule is accomplished in a few days, and the ovuliferous vesicle is untorn except at one point, which limits the source of the hemorrhage. We shall afterwards see, that if the reflux of blood to the part, or the softening of the ovary, occasion profuse hemorrhage, a hæmatocele will be formed. There is a sanguineous afflux towards the external organs of generation, as well as towards the ovary at the time of ovulation. The vagina and labia majora then acquire a temporary increase of vascularity and temperature. During the time of rut, the cow has a glairy, sanguineous discharge from the vulva: the mammæ become swollen, hard, and full of colostrom. All these phenomena result from the erethismal congestion of the organs of generation.

Similar changes take place in a woman. Each menstrual period is accompanied by a remarkable turgescence of the mucous membrane of the vagina and vulva. There are also pains in the breasts, and in the regions of the kidneys and ovaries; these pains being indicative of local congestion.

These statements rest on experiments and clinical observations; and are confirmed by pathological anatomy when death occurs at a menstrual period. Normal anatomy, if properly studied, will show that considerable congestion must take place during the exercise of the ovarian and uterine functions.

Professor Rouget's beautiful experiments have explained the way in which erethismal congestion takes place in the genital organs of women. This ingenious anatomist, after immersing the dead body in a warm bath, injected the vessels, and thus demonstrated the erectile property of the plexuses which envelop the ovary and form its vascular bed. During the artificial erethism caused by injection of the vessels, every part of the generative organs become turgid. The ovary, the lateral movements of which are limited by its ligament and mesentery, was seen to bear itself upwards and towards the highly injected fimbriated extremity of the Fallopian tube, by which it was grasped and capped.† There was thus presented to the eye, a picture of the physiological occurrences which take place at a precise moment; and when we see a vascularity so intense, and so liable to very active congestion, we readily understand how the mucous membrane of the tube, which (according to M. Béraud) contains vascular rings, may become the seat of hemorrhage at the menstrual period. We can also understand, that under the same circumstances, the ovary may become softened, apoplectic, varicose, originating, possibly, a catamenial hæmatocele. But in the event of the latter supposition being realized, there must previously exist a structural change of the ovary; for a healthy ovary covered with its peritoneal coat could not, from the mere menstrual effort, become the source of any notable hemorrhage; otherwise, hæmatoceles would be infinitely more common than they are.

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* Robin (Charles): Note sur les Hómorrhagies des Vésicules Ovariennes. [*Mémoires de la Société de Biologie:* 2me série, t. iii, p. 139 for 1856. Paris, 1857.]

† Rouget: Recherches sur les Organes Erectiles de la Femme; sur Ovulation et la Menstruation. [*Journal de la Physiologie de l'homme et des animaux*, 1858.]

Hemorrhage from the mucous membrane, on the other hand, may take place without these membranes having undergone any previous change: for its production, it is only necessary that there exist previously intense congestion, or an alteration in the constitution of the blood. Thus, normal physiological ovarian hemorrhage, not being sufficient in quantity to produce a hæmatocele, and abnormal ovarian hemorrhage requiring for its occurrence a previous alteration of the parenchyma of the ovary, we have a right to suppose that ovarian hæmatocele is very rare. On the other hand, in the tubal mucous membrane, from its special structure, from the hemorrhagic tendency which it possesses in common with all mucous membranes, from the determination of blood which takes place as an accompaniment to menstruation, there arises a condition of all others the most favorable to hemorrhage, and so, likewise, to the formation of catamenial hæmatocele.

I am, I confess, not much disposed to believe that the peritoneum can be the source of a hemorrhage: sanguineous effusion seldom takes place from serous membranes which have not been previously the seat of inflammation, and then the inflammation is often of a special nature, as in cancerous and tuberculous peritonitis. The cases reported by Dr. Tardieu* seem to me to point to catamenial hæmatocele, that is to say, to tubal hemorrhage, particularly when it is remembered that the ovaries were carefully examined, and found to present no alteration of structure.

We have just seen that there are two principal causes which give rise to hæmatocele—organic lesion of the ovary, and functional lesion of the Fallopian tube; on this subject, scientific opinion is divided. Some authors attribute hæmatocele exclusively to one or other of these causes; and others are eclectic, accepting both theories, and believing that under determinate circumstances ovary or tube may be the starting-point of the bleeding.

Among the exclusives may be ranged Nélaton, Denonvilliers, Huguier, Lenoir, and Laugier, who believe that the seat of the hemorrhage is always in the healthy ovary, when it occurs at the time of ovulation; and in the diseased ovary, when it occurs at other times. Formerly, I used to maintain, that the tube always was the seat of the hemorrhage. New cases have modified my opinion, and I am now eclectic in my views; that is to say, I hold with Drs. Puech, A. Voisin, Bernutz, and Gallard, that there are various causes of hæmatocele. I consider that there are two principal causes—one ovarian and accidental with organic alteration of the ovary, and the other tubal and catamenial without organic alteration of the tube.

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* TARDIEU: Observations Pratiques de Médecine Légale sur les cas de Mort Naturelle et de Maladies Spontanées qui peuvent être Attribuées à un Empoisonnement. [*Annales d'Hygiène Publique et de Médecine Légale*, t. ii, p. 157. Paris, 1854.

The existing dissidence of opinion is explained by the rarity with which hæmatocele occurs, and the still greater rarity of autopsies in such cases. Then again, the autopsies, being frequently made long after the commencement of the hemorrhages, are far from advantageous for the elucidation of the pathogenetic problem. The ovary, tube, and peritoneal cysts may produce such structural changes as sometimes to render it impossible to determine whether the hæmatocele is within or without the peritoneum: and *à fortiori*, it is often impossible to make out whether the organic lesions of the ovary are primary or secondary. In saying that catamenial hæmatocele does not cause death unless the amount of hemorrhage be excessive, which is very unusual, and that ovarian hæmatocele proves fatal by inducing peritonitis, which is more common, I mean, that autopsies having been chiefly performed under the latter conditions, many observers have been led to believe only in hæmatocele of ovarian origin, produced either by an ovarian cyst, an apoplexy of the ovary, or a partial suppuration of the ovary.

In catamenial hæmatocele, on the contrary, death occurs only in exceptional cases; and consequently the autopsies which would demonstrate the hemorrhage to have originated in the tube itself, or in its expanded extremity, must necessarily be also exceptional. However, the archives of science contain a certain number of necroscopic observations possessing a twofold interest, from their establishing, *first*, that there had been tubal hemorrhage which had passed into the peritoneal cavity, and *second*, that the patients had not sunk in consequence of a moderate hemorrhage into the peritoneum, but rather from a state of general disease, as in the cases related by Barlow, Simpson, Laboulbène, Proust, and Hélie. In these cases, there was no inflammation of the peritoneum, and the death of the patients was caused by purpura, small-pox, measles, and scarlatina.

In these cases, there is no ground for supposing, with Bernutz, that the hemorrhage had taken place by a reflux from the uterus through the tube into the peritoneum, seeing that both the superior and inferior orifices of the cervix were free, as was proved in Scanzoni's case by the catamenia being abundant, and in Laboulbène's case by the occurrence of an extensive flow of blood from the uterus at the commencement of the symptoms. It is therefore more reasonable to suppose that the mucous membrane of the tube may, like that of the uterus, be the seat of hemorrhage, and that that hemorrhage from the tube may be poured into the peritoneum.

It is the more rational to believe in tubal hemorrhage, as it has been shown by the anatomical researches of MM. Rouget and Béraud, and by the admirable inquiries into the physiology of menstruation of Lee, Raciborski,* and Pouchet,† that there is a catamenial transudation from the Fallopian tubes.

These statements do not rest on mere theories. Follin and Oulmont have reported two cases in which they found, post-mortem, that the intra-peritoneal hemorrhage had proceeded from the mucous membrane of the oviduct. Three cases of Dr. Tardieu, reported by Dr. Auguste Voisin,‡ support, in my opinion, the doctrine of tubal hæmatocele. The cases are of so much importance that I will quote the passage in which they are described. It is to the following effect:

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* RACIBORSKI: De la Puberté et de l'Age Critique chez la Femme au point de vue physiologique, hygiénique, et médical, et de la Ponte Périodique chez la femme et chez les mammifères: Paris, 1843. Du Rôle de la Menstruation dans la Pathologie et la Thérapeutique: Paris, 1856. Traité de la Menstruation: Paris, 1868.

† POUCHET: Traité Positive de l'Ovulation Spontanée et de la Fécondation dans l'Espèce Humaine et les Mammifères. Paris, 1847.

‡ VOISIN (Auguste): De la Hæmatocèle Rétro-utérine, et des Epanchements Sanguins non enkystées de la cavité peritonéale du petit bassin. Paris, 1860.

"We have met with examples of this remarkable kind of effusion into the true pelvis, behind the uterus, occurring in two young women without any relation to conception or attempted abortion; death took place in both so rapidly that suspicions of poisoning arose, and led to judicial inquiries, in which there was not elicited any other cause of death than that which I have stated. One of these women had been married three weeks; and the catastrophe, according to the avowal of the husband, might be attributable to excessive coitus. The other was a young Jewess whose malady began after sexual excesses with some students."

In addition to these particulars, which Dr. Tardieu communicated to Dr. A. Voisin, I must add that in the case of a third woman (in which there was an autopsy) death followed a kick which the woman received on the left haunch from her husband.

Dr. Tardieu ascertained by examination that there was intra-peritoneal hemorrhage; and he thought that it was the result of a sanguineous exudation from the peritoneum. It was thoroughly established by Dr. Tardieu, that there was no lesion of the ovaries, Fallopian tubes, or pelvic bloodvessels. There was no apoplexy of the ovary, nor was there any rupture or ulceration of a hæmatic cyst. As it was not reasonable to suppose that there could be an extensive hemorrhage from the peritoneum, unless it had previously been the seat of inflammation, I was obliged to regard the sanguineous discharge as coming from the tubes. Now Dr. Tardieu does not say that peritonitis had existed. On the other hand, it was proved that the first two patients were suffering from excessive coitus, in consequence of which the utero-ovarian bloodvessels were exceedingly congested. In the third case, the patient was subjected to external violence during menstruation; the intra-peritoneal sanguineous effusion was very extensive.

Thus it seems that during or after excessive sexual indulgence or after injuries received during menstruation, extensive intra-peritoneal hemorrhages may occur, without its being possible to trace them to any other source than the mucous membrane of the tubes; because there being no lesion of the ovaries or utero-ovarian vessels, they are only to be explained by the structure of the mucous membrane of the tubes.

In making this statement, I am not denying that hæmatocele is produced by reflux of blood from the uterus into the peritoneum, when there is an obstacle to the menstrual flow by the natural passages, such cases having been observed by Ruysch, De Haen, Delpech, and Bernutz.

There is reason to believe, however, that in women in whom there is absence or extreme narrowness of the vagina, the deepseated sexual organs are slightly developed; were menstruation as abundant in them as in other women, the bad consequences of retention of the menstrual flux would be very common in the former; but this is not the case. Pretty frequently, we meet with cases of atresia of the vagina in women between twenty and twenty-five years of age. Several years ago, I attended, along with Professor Velpeau, a young lady twenty-seven years of age, who, for seven years, had had monthly attacks of dysmenorrhœa. On examination, we found that there was complete absence of the vagina; but by introducing the finger into the rectum, we discovered a tumor which, from its situation and form, was evidently the uterus. Every month, this individual experienced pains in the hypogastrium and loins: the breasts became hard: and at the period of spontaneous ovulation, she had severe abdominal pains, which were, I believe, dependent on the fall of an ovule, accompanied by some blood, into the cavity of the peritoneum. This young lady menstruated internally. A febrile condition continued for some days, the pains then diminished, and ceased in eight or ten days. Similar symptoms recurred every month; and at each of these recurrences, there was a beginning of catamenial hæmatocele.

I concur in the opinion of Drs. Richet and Devalz,* that in a varicose state of the broad ligament, rupture of vessels may occur, and become the source of a hæmatocele which will generally be extra-peritoneal, because the varicose veins are situated between the two folds of peritoneum covering that ligament. I also concur with Dr. Devalz in believing that the varicose condition of the large veins, by extending to the veins of the parenchyma of the ovary, may cause œdema, partial softening, and apoplexy of the ovary. There will take place something similar to that which we see in varicose ulcers of the lower extremities: hemorrhage (extra or intra-peritoneal) may be the consequence of the ovarian softening or ulceration. It is evident that hæmatocele originating in this way must be of rare occurrence.

The special varicose condition of the veins may predispose to apoplexy of the Graafian vesicles, and become the beginning of hæmatic cysts, which themselves constitute one of the immediate or accidental causes of hæmatocele.

I have just been mentioning the part which rupture of varicose veins may have in the formation of hæmatocele: Dr. Devalz relates the cases of Drs. Richet, Depaul, and Gueneau de Mussy, which show that hæmatoceles from rupture of the utero-ovarian veins have their seat in the peritoneal cavity, or in the subperitoneal cellular tissue.

Bear in mind, that whenever you wish to demonstrate venous hemorrhage on the dead body, you must, as Dr. Devalz recommends, previously inject the utero-ovarian veins. I regret that in a very remarkable case of hæmatocele, which occurred in the wards of Dr. Alfonse Becquerel, reported by Dr. Benjamin Ball, no such injection was made, for had it been made, the source of the hemorrhage might have been discovered. This inquiry possessed a twofold interest: the effusion was subperitoneal, and yet the right ovary was so changed, that it was nearly impossible not to consider it as the origin of the hemorrhage. Keeping in mind these two cases, it is impossible not to admit that rupture of the ovary in the ovarian mesentery may occur. However, in the history of the case, it is not stated that rupture of the ovary was ascertained to have taken place: perhaps, from some abnormal opening in one of the engorged tubes, the catamenial blood escaped into the substance of the broad ligament.

I cannot do better than read to you the details of this case, reported very carefully by Dr. Ball, who, when he made the necropsy was thoroughly well informed upon the subject now before us.

"An unmarried woman was admitted to Dr. Becquerel's wards on 18th January, 1858. She was carried on a stretcher to the hospital when in a state of profound coma.

"Two days previously, it was stated, this woman had been obliged to take to her bed in consequence of a chill during menstruation. The symptoms having rapidly become aggravated, it was considered necessary to have her taken to the hospital on the third day of her illness.

"At that time, her condition presented all the symptoms of the utmost gravity. The face was pale, and the features contracted: the forehead perspired profusely: the lips were white, the extremities cold, the pupils dilated: respiration was stertorous, and there was a little sanguinolent foam coming from the mouth: the skin was nearly insensible. The pulse, 140 in the minute, was thready, irregular, and very rapid. Respiration was so noisy that auscultation of the heart was impossible. Mayor's hammer was applied without obtaining any result. An hour later, the patient died.

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* DEVALZ: Thèse. Paris, 1858.

"*Autopsy.*—On opening the abdomen, no liquid flowed out, nor was any sanguineous effusion found in the peritoneum: there were neither false membranes, nor adhesions of the intestinal convolutions.

"After having raised up and detached the mass of small intestine, there was discovered a large sanguineous accumulation prominent on the sides of the uterus: that organ, pressed towards the pubes, had left its print upon the coagulated mass. When this mass was raised up, it was found that the effusion had taken place into the peri-uterine cellular tissue, between the uterus and rectum; the coagulum extended down to the vicinity of the anus, pushing forwards and upwards the posterior wall of the vagina: the tumor nearly filled the true pelvis, the cellular tissue of which was destroyed: the broad ligaments also contained coagulated blood. The peritoneal covering of the posterior surface of the uterus was partially detached by infiltration, which extended up to the middle of the body of the uterus, passed above the mass of coagulum, and was continuous with the peritoneal coat of the anterior surface of the rectum.

"The Fallopian tubes were shrouded in the tumor, and *contained soft, red clots.* Upon washing out the tubes, it was found that their mucous membrane was red, swollen, and vascular.

"The left ovary, completely infiltrated with blood, was coverted into a blackish pulp, in which no remains of the structure of the organ could be detected. The right ovary presented no appreciable alteration of tissue.

"The effused mass had the consistence of currant-jelly, and a blackish-red color. No membranous envelope encysted it, and it contained no fluid.

"The uterus may be described as of ordinary volume, although its exact dimensions were not measured. Its cavity contained no clots, but *its mucous lining presented a vascular arborescence.* The os tincæ was thickened: its lips were split in different directions: *the uterine orifice was half open.*

"The state of the other abdominal viscera was normal."

In 1855, when M. Nélaton's theory was generally accepted, M. Laugier, perceiving that it did not explain the majority of the cases of hæmatocele, set himself to demonstrate that, unless the ovary had previously undergone structural change, it could not originate an intra-peritoneal hæmatocele. He arrived at the following conclusions:

"1. Spontaneous ovulation, as had been previously stated, is the immediate cause of retro-uterine hæmatocele.

"2. Physiological congestion of the ovary during spontaneous ovulation, with persistent aperture of the Graafian vesicle, does not cause hæmatocele.

"3. To produce hæmatocele, there must be a state of excessive congestion: such a state is sometimes induced by accidental causes during, or a few days after, menstruation. Abortion is not (as had been erroneously supposed) an immediate cause of hæmatocele.

"4. The chief immediate causes of hæmatocele are the returns of spontaneous ovulation, which gradually augment the volume of the hæmatocele.

"5. The successive ovarian vesicles open into the hæmatic cyst, and there remain; so that the ovary is destroyed by a small number of spontaneous ovulations, occurring under the conditions presented by the organ when the formation of a hæmatocele has commenced.

"6. As rupture of a Graafian vesicle affords passage for the blood from the ovary, the cyst of the hæmatocele is generally intra-peritoneal.

"7. A character which belongs in common to spontaneous ovulation and hæmatocele is unilateral abdominal pain, the seat of which is the ovary during vesicular evolution.

"8. Rut in animals may cause ovarian congestion followed by rupture of the ovary, that is to say, by pathological phenomena similar to those belonging to retro-uterine hæmatocele."*

M. Laugier directed his attention only to ovarian hæmatocele; and was ready to admit other etiological explanations if sufficiently demonstrated to him.

There now remains only one variety of hæmatocele for me to speak of—a variety which I believe to be exceedingly rare. Though I have not found in the cases related by Dr. Tardieu sufficient proof of tubal hæmatocele, I cannot absolutely deny that under certain special conditions, which cannot be very definitely described, exudation of blood into the peritoneal cavity may take place irrespective of any cancerous or tuberculous cachexia. Though I have seen no such case, and though I do not know of any, yet, reasoning from analogy, I am constrained to admit with Dr. Bernutz that these effusions may take place into the peritoneum, just as they take place into the cavities of the pleura, pericardium, and arachnoid; while, I repeat, however, that I believe such hemorrhages to be very rare, unless the serous membranes, or their contained organs, are the seat of cancerous or tuberculous deposits.

I have directed your attention to the varied etiology of retro-uterine hæmatocele; but I still require briefly to indicate the points in relation to the proximate causes of sanguineous effusion which are indicated in the remarks I have now made.

In cases of catamenial hæmatocele, the menstrual epoch is the exciting cause: possibly, sexual intercourse during menstruation may be a determining cause. In animals, however, coitus takes place only during the period of rut, the rut being a state similar to menstruation, and it is very unusual to meet with hæmatocele in female animals, who have not had connection with the male, yet you will find in Dr. Laugier's memoir a very curious case of sanguineous effusion into the peritoneum of a cow, which occurred during rut, though it is not stated that the animal was fecundated. The hemorrhage came from a hæmatic cyst of the ovary, from which there flowed several litres of blood. After menstruation and ovulation, external violence, jolting in a carriage, a fall, or a strain, may cause rupture of an ovarian cyst, of a tubal cyst, or of a varicose vein; and when we reflect upon the great development and turgescence of the ovarian vascular plexuses during pregnancy, the rarity of hæmatocele from rupture of the ovarian veins seems remarkable, till we remember the fact, noticed by Devalz, that, under the circumstances, the walls of the veins acquire great thickness.

Hæmatocele can be detected by digital examination *per vaginam*, and by palpation. It is ascertained by digital examination, that the cervix uteri is situated immediately behind the pubes, that the vagina is shortened, that, behind the cervix there is a hard tumor, which has often been mistaken for the body of the uterus in a state of retroflexion, but which is recognized as being independent of the uterus from having neither the form nor consistence of that organ. This tumor is a hæmatocele.

By abdominal palpation, the bladder being empty, a hard round tumor is detected behind the pubes, which tumor is not the uterus, as can be proved by practicing simultaneously abdominal palpation and digital examination of the uterus. It is then ascertained that the uterus is inclosed by, and rests upon, a more or less resistant and, sometimes depressible tumor, which occupies the whole of the lower and posterior part of the true pelvis. By introducing the index finger into the rectum, we can circumscribe the sanguineous effusion, so as to determine its lateral boundaries; whilst by abdominal palpation, we can ascertain its superior boundaries, so very extensive sometimes as to reach up even to the umbilicus.

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* LAUGIER: Mémoire sur l'Origine et l'Accroissement de l'Hématocèle Rétro-utérine. [Presented to the Institute, 26th February, 1855.]

It is unusual, however, for the tumor to be so large. It frequently extends more on one side than the other of the uterus, this inequality in development not always bearing any relation to the situation of the origin of the hemorrhage. Sometimes adhesions, resulting from previous inflammation of the uterus and its appendages, may explain the inclosure of the sanguineous effusion, and its greater bulb on one side than on the other.

The following are the symptoms of hæmatocele: During, or irrespective of, menstruation, a woman is all at once seized with acute pain in the abdomen, soon becomes pale, the skin in all parts of the body loses its color, and the slightest movement is unendurable: already there is reason to suspect that the case is one of intra-peritoneal hemorrhage: the pain and pallor lead you to this conclusion, the correctness of which will soon be confirmed by physical examination. The degrees of intensity of these two symptoms —the pain and the pallor—or the predominance of one of them, may give you a clue to the origin of the hæmatocele.

Should the pain be very acute, and the pallor not very marked, while the pulse is very low, thready, and compressible, while vomiting and diarrhœa exist, you may conclude that the amount of hemorrhage is small, that its seat is the peritoneal cavity, and that very probable its source is the ovary. The amount of hemorrhage is small, because it is furnished by the rupture of an ovarian cyst or disorganized ovary: in the latter case, the effused blood may be changed, and of so irritant a character as to produce peritonitis.

On the other hand, the hemorrhage is catamenial and profuse, when the pallor is extreme and the abdominal pain slight: the source of the hemorrhage is then the tube and its expanded extremity. The pallor is extreme, because the mucous membrane of the tube has rapidly yielded a profuse hemorrhage: and the pain is not great, because the effused blood, not being altered, has produced very little irritation of the peritoneum. Experiments which I formerly made with Dr. Leblanc proved, that normal blood does not irritate serous membranes; but that blood which has become modified by being external to the vessels exceedingly irritates such membranes.

Let me add, that these two symptoms in conjunction—pallor and pain—are particularly observed in cases in which the cavity of the peritoneum is the seat of the hæmatocle; so that, when they are less marked, the inference is that the hæmatocele is extra-peritoneal. To make a complete diagnosis, it is necessary to make a digital examination by the rectum and vagina, and to employ abdominal palpation.

The two affections with which retro-uterine and peri-uterine hæmatocele have been confounded are pelvic peritonitis and peri-uterine cellulitis. You know that after delivery, abortion, or cauterization of the cervix uteri, it is not unusual to have pelvic cellulitis and pelvic peritonitis. Besides mentioning that both these inflammations are almost exclusively observed under these circumstances, I ought to state: 1st, that in pelvic peritonitis, the sero-purulent effusion, which is its consequence, never gives rise to a *solid retro-uterine* tumor of as resistant a character as in hæmatocele: 2d, that, on the contrary, in peri-uterine cellulitis, there is formed, in the first instance, a resistant tumefaction of the cellular tissue of the true pelvis and broad ligaments. The swelling is always greater on one side than on the other: the uterus generally inclines to the right or the left, and very seldom forwards: ultimately, after a variable period, fluctuation becomes evident, and the inflammation shows a great tendency to invade the iliac fossæ. Peri-uterine abscesses frequently open into the rectum, bladder, or vagina: and some of them point towards the inguinal region or crural canal: again, the inflammation may gain the anterior abdominal wall, and then the pus, detaching the peritoneum, makes an exit for itself above the groin.

When the hæmatocele is intra-peritoneal, it soon becomes encysted, and absorption of the serous fluid rapidly takes place, particularly when the hæmatocele is catamenial; and it is only in exceptional cases that the tumor evacuates itself into one of the natural reservoirs. It happens otherwise when the hæmatocele is of ovarian origin; the acute peritoneal irritation may then extend to the cellular tissue, and as in extra-peritoneal hæmatocele) the tumor may open into the vagina, rectum, or bladder. When hæmatocele is extra-peritoneal, that is to say when it is situated in the cellular tissue of the true pelvis, the diagnosis may be made at the commencement of the hemorrhage; but at a later period, if there be inflammation of the pelvic cellular tissue, it is only the suddenness of the primary accidents which can give us a clue to the diagnosis.

I will not enlarge upon the diagnosis between hæmatocele and extra-uterine pregnancy: let it suffice to remind you, that Dr. Gallard, in his first memoir, was disposed to regard all retro-uterine and peri-uterine hæmatoceles as cases of extra-uterine pregnancy; but, besides the fact, that hæmatocele is met with in virgins, it is important to remark that the commencement of hæmatocele is sudden, while the beginning of extra-uterine pregnancy is latent, its progress slow, and the hemorrhages to which it may give rise only observed at a period far removed from the period of impregnation.

I will not enlarge upon the differential signs of ovaritis and ovarian cyst. I would remark, however, that cysts of the true pelvis, situated in the subperitoneal cellular tissue, may, in women, be mistaken for hæmatocele. I refer to hydatid cysts which have become the seat of inflammation. The following case will show you the possibility of making an erroneous diagnosis:

A young woman of nineteen, irregular in her menstruation, had experienced rather violent abdominal pains at the time of an imperfect menstrual flux. Some months later, she was suddenly seized with pain in the true pelvis, pain which particularly extended to the right side of the abdomen. Digital examination, *per vaginam*, enabled me to ascertain that the entire uterus was pushed towards the symphysis of the pelvis: there was a tumor as large as the fist situated laterally behind the uterus, and occupying the right side of the true pelvis: the rectum and tumor seemed to constitute one mass: digital examination by the rectum occasioned great pain. Pain interfered with defecation: the desire to make water had become very frequent. Absolute rest, low diet, and expectant medication were enjoined. Ten days after the commencement of the symptoms, there occurred anal tenesmus, and sanious discharge from the rectum, attended by comparative relief, and by collapse of the abdominal tumor. The stools, which had a dysenteric appearance, were examined daily with care: it was found that they contained no clots of blood; but, during three days, fragments of soft cystic false membrane. In a few days, the patient's health was completely re-established. I regret that the membranes thrown off were not examined by the microscope: my impression was, that they came from suppurating hydatid cysts of the true pelvis.

I at first thought that this young woman was the subject of hæmatocele, an opinion supported by the sudden commencement of the abdominal pain. It might have been supposed that there had been an extra-uterine pregnancy, the cyst of which had become the seat of inflammation. For months, this young woman had suffered from disturbance of the catamenial function. The expulsion of a false membrane, smooth and soft, resembling the envelope of a hydatid, and the simultaneous sudden disappearance of the tumor, led me to abandon the opinion, that there was either a sanguineous tumor or a fœtal cyst, and by exclusion, to conclude that the case was one of an inflamed hydatid cyst eliminated by ulceration through the rectum. Cysts of this kind are met with comparatively seldom in the true pelvis, particularly during the first half of life, as appears from the researches of Charcot and Leudet. They do, however, sometimes occur; and when they become inflamed, they originate a group of symptoms which are of such a character as to demand notice in discussing the differential diagnosis of retro-uterine hæmatocele.

The details into which I have entered in reference to the formation of hæmatocele, and in reference to the seat and extent of the hemorrhage, point out that the prognosis cannot be the same in the different kinds of hæmatocele. Catamenial hæmatocele is not, in general, a serious affair: if there have not been much hemorrhage, the volume of the tumor soon diminishes, the pain disappears, and the uterus regains its normal position. If, on the contrary, the hemorrhage be profuse, the tumor has not only a much greater size, and involves a larger portion of the peritoneum, but induces an extreme degree of anæmia predisposing to new tubal hemorrhages, which, by continuing for some months, may augment the tumor, and prevent its absorption.

Last year, you may have seen in bed 8, St. Bernard Ward, a young woman with the symptoms of peri-uterine cellulitis: the tumor increased in size, became fluctuating, and yet showed no tendency to open spontaneously into any of the natural cavities. I resolved to make a puncture at its most salient point, that is to say, in the lower part of the abdomen, four or five centimetres from the linea alba. The progress of the phlegmonous abscess led me to believe, that adhesions had formed between the cyst and the walls of the abdomen; but I was greatly surprised to see from two hundred and fifty to three hundred grammes of sanguinolent serosity issue from the canula: the remainder of the tumor was composed of fibrinous clots. Immediately after the puncture, great relief was experienced by the patient, the fever ceased, and sleep returned. By slow degrees, the tumor was absorbed; and the patient left the hospital cured, after having exhibited, when under our observation, during more than a month, signs which led me to form an erroneous diagnosis. The principal causes of my mistake were the persistence of inflammatory symptoms, and the progressive increase of a fluctuating tumor. It must be noted that, for the first sixteen days, this woman had had bloody discharge from the vagina; and probably in this particular case, the tube had at the same time, poured into the peritoneum a constantly increasing quantity of blood: this patient had a devious, intra-peritoneal menstruation. It is well to remember that these hæmatoceles, which may be called (so to speak) continuous or intermittent, are often associated with a special condition of the blood predisposing to hemorrhages.

The remarks which I have made on the progress of extra-peritoneal hæmatoceles obviates the necessity of my discussing at any length the gravity of their prognosis. A considerable number of published cases sufficiently establish the frequency of inflammation of the subperitoneal cellular tissue.

These hæmatoceles have a tendency to open into the rectum or vagina; and as, even after these spontaneous openings, general poisoning has been observed, we ought to avoid opening them with a knife, unless there are special and urgent indications for the adoption of that proceeding.

There are, however, at the commencement of every hæmatocele, two indications demanding the physician's intervention, viz., hemorrhage and peritonitis. Should the hemorrhage threaten to continue, we must resort to local and general hæmostatics: the application of ice to the abdomen, the administration of rhatany, sulphuric acid, and ergot of rye may be prescribed with advantage. If, on the other hand, the peritoneal pain be the predominating symptom, it must, in the first place, be calmed by narcotic, obtunding drugs: opium or belladonna must be administered internally, and applied externally by frictions to the abdomen.

When the hæmatocele is catamenial, that is to say, when it is most probably tubal, the principal object to be kept in view is to restrain the hemorrhage and prevent its recurrence.

It is often necessary to seek for the cause of the hemorrhage in the general state of the patient. If she have chlorosis, or anæmia, employ invigorating treatment, selecting in each case the particular medicine best suited to the state of the individual. Some patients, when placed under favorable hygienical conditions will lose all symptoms of chlorosis and anæmia: the open air, exercise, good food, and mental satisfaction will cure them. Others will require ferruginous preparations and bitters. Bretonneau recommended the preparations of cinchona in hemorrhages, and administered them successfully in the epistaxis which is so common in young people. I believe that, to prevent the renewal of hemorrhagic attacks seemingly associated with chlorosis or anæmia, we may, in cases of hæmatocele, advantageously employ the preparations of iron and cinchona.

Is it necessary to discuss at length the value of puncture in the treatment of hæmatocele? The surgeons who recommend the tumor to be punctured by the rectum or vagina were the first to point out the necessity of abstaining from all surgical intervention, so long as there was no threatening of the cyst bursting into the peritoneum. Their caution is worthy of imitation; and indeed, I cannot too strongly advise you to follow their example. From numerous cases, it has been learned, that there is danger in establishing a communication between the interior of the cyst and the external air, particularly when the opening is made through the walls of a natural passage, the rectum or vagina, in which fluids undergo very rapid alteration. When there is an unquestionable indication of the necessity of surgical intervention, a puncture ought to be made through the anterior abdominal wall, provided it be united to the hæmatic tumor by adhesions. Proceeding in this manner, we avoid one cause of general poisoning, by avoiding doing anything to permit the contents of the rectum or vagina to enter the interior of the cyst.

LECTURE XCIV.

PUERPERAL PURULENT INFECTION.

Puerperal Fever is not a simple morbid state.—The Physiological State called "Puerperal."—It predisposes Lying-in Women and New-born Infants to a variety of affections, such as Peritonitis, Phlebitis, and Lymphangitis.—In these Puerperal Affections, there is a great Tendency to Suppuration.—A Primary Purulent Diathesis exists in Puerperal Women.—A Secondary Purulent Diathesis may exist, the consequence of Phlebitis, Inflammation of the Lymphatics, or the direct Absorption of Pus from the Placental Wound.—Secondary Purulent Infection of Lying-in Women and of New-born Infants is identical with the Purulent Infection consequent upon Amputations.

GENTLEMEN: United under the generic denomination of *puerperal fever*, there are a great many morbid conditions which all in common possess pyogenesis as a characteristic. Is there such a thing, correctly speaking, as a puerperal fever? The several great efforts which have been made to describe this morbid entity have only served to show the great extent and variety of the pathology of the recently delivered woman.

In an academic discussion, which is still of recent date, the most competent observers expressed entirely different opinions as to the malady called puerperal fever; which shows that it is not *one* like such fevers as measles, scarlatina, and small-pox.*

Some regard puerperal fever as a local disease—a peritonitis or a phlebitis. Others look upon it as a general disease, like typhus, in which the lesions are only secondary, and are infinitely various. There are others again, who, while they admit the affection to be general, consider it to be the result of a purulent or putrid infection of local origin, such as phlebitis, or gangrene of the uterus. Then there is another class—perhaps the most sagacious—who attribute the diversity of opinion among observers to the diversity in the disease they describe.

My own opinion is that in a matter of this kind, we must be eclectic. I believe that puerperal fever, like typhus fever, is happily a rare disease; and I also believe, that the different puerperal symptoms described as peritonitis and phlebitis are very common. Before describing the different forms which are assumed by puerperal symptoms, let us recall the physiological conditions of the pregnant and recently delivered woman. They constitute a special morbid predisposition—*a real diathesis*.

From the very moment when conception takes place, great changes occur throughout the woman's whole organism. The countenance acquires a peculiar aspect: generally, the features are drawn, the eyes are surrounded by a dark areola, the nose is pinched, and sometimes the face is covered with ephelides. The nipple, the linea alba, and the mucous membrane of the genital organs acquire a brown color: the follicles of the vagina, the labia majora, and mammary areola become increased in size. Menstruation is stopped: the breasts soon become swollen, and prepare themselves for the secretion of milk. There is sometimes increased salivary secretion, capricious appetite, vomiting, diarrhœa or constipation. The liver enlarges, and its acini become loaded with fat: the thyroid gland may show increased development, and even the heart may undergo well-marked hypertrophy. The blood is modified in respect of the proportion of its component parts: there is an increase in the fibrin, and a diminution in the red globules: a soft blowing sound is heard in the vessels of the neck and at the base of the heart. There then exists a variety of chlorosis which was first described—and very well described—by Cazeaux.* Albuminuria is sometimes met with.

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* DE LA FIEVRE PUERPÉRALE, DE SA NATURE, ET DE SON TRAITEMENT: Communications à l'Académie Impériale de Médecine, par MM. Guérard, Depaul, Beau, Hervez de Chégoin, P. Dubois, Trousseau, Bouillaud, Cruveilhier, Piorry, Cazeaux, Danyau, Velpeau, Jules Guérin, &c., précédés de l'indication bibliographique des principaux écrits publiés sur la fièvre puerpérale. 8vo., pp. 464. Paris, 1858.

Such are the principal organic and functional modifications of pregnancy. They already constitute the puerperal state—a physiological state which may lead to disease, and is often manifested by a special—a *purulent*—pathological condition.

Lorain† and Tarnier‡ have lately laid much stress on the fellowship of the morbid state of the mother and her new-born infant, and still more of the mother and fœtus. The infant, whether an appendage of the placenta or the breast, lives by its mother's blood. It must, therefore, share the conditions predisposing to disease which act on the mother, and must similarly receive at a given moment—at the time of an epidemic, for example—the same morbid influence. As the infant grows older, it proportionately loses this morbid solidarity. In the womb, the fœtus lives solely by its mother: but when surrounded by the atmosphere, on the contrary, it may, *proprio motu*, contend against its inherited maternal morbid conditions. It will, therefore, be less and less under puerperal conditions, the longer the interval of time since its birth.

We see then, that the fœtus shares, and the infant retains, for a period of varying duration, the maternal puerperal state. Both derive from the mother a puerperal state, and all its consequences, the chief of which is a predisposition to purulence.

Some have wished so to extend the puerperal condition in women as to include the condition at each menstrual period; and certainly, each menstrual flux is accompanied by an exfoliation from the internal surface of the uterus. In certain cases, the uterine mucous membrane exfoliates in shreds or in one entire piece; and when this sort of delivery is painful and difficult, morbid phenomena are observed similar to the ordinary phenomena of parturition: there occur renal pains, uterine colics, hemorrhage during and after the detachment of the mucous membrane, followed by a sanio-purulent discharge, and consecutive separation: this incomplete delivery, which accompanies the menstrual ovulation, lasts for some days, after which nature resumes its natural order, till next menstruation. Whilst these changes are proceeding, a modification shows itself in the performance of the functions, and in the temper of the individual. There is an afflux of blood to the ovaries and tubes, and almost always a sympathetic turgescence of the mammæ. All these phenomena, which are principally localized in the uterus and its appendages, are analogous to those which appear at the end of pregnancy and after delivery. I must remark, however, that the general state of the menstruating woman is different from the general state of the recently delivered woman: though in the former, the blood, the humors, and the organs have undergone modifications, they are not of so profound a nature as in the latter. The general state is different; and that is sufficient to account for the morbid predisposition to puerperal symptoms being less. There is, however, in the analogy of the states, matter of high clinical importance; and the cases of peritonitis and purulent infection observed among menstruating midwives during puerperal epidemics, strongly support the views of MM. Lorain and Tarnier.

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* CAZEAUX: De la Chlorose des Femmes Enceintes. [*Bulletins de l'Académie de Médecine.* Paris, 19 Fév., 1850: t. xv, p 448.]

† LORAIN: De la Fièvre Puerpérale chez la Femme, le Fœtus, et le Nouveau-né. Paris, 1855

‡ TARNIER: De la Fièvre Puerpérale observée à l'Hospice de la Maternité. Paris, 1858.

The new-born infant, as I have said, has received, during intra-uterine life, a special influx from the mother, which imparts to it a predisposition to her morbid affections. The umbilical wound, which corresponds with the placental wound of the uterus, may occasion symptoms analogous to those observed in suppurative uterine phlebitis, so that we may have identically the same purulent infection in mother and infant. The general conditions, therefore, of the new-born infant are nearly the same as those of its mother. There exists in both a physiological solidarity, showing itself in an excess of formative power [*puissance organisatrice*]: there is also in both a pathological solidarity, showing itself in a tendency to purulence, and in a remarkable similarity in the local affections [*localisations morbides*].

If it be correct to say that the puerperal state begins with menstruation, or, according to some pathologists, begins with fertilized menstruation [*menstruation fécondée*], and ends with lactation, it is unquestionable, that the puerperal symptoms most frequently occur, and are most severe, at the epoch when the puerperal state is most marked, that is to say, during the three or four weeks immediately following delivery, when the uterine wound exists. This puerperal state is of shorter duration in the infant, and generally terminates with cicatrization of the umbilicus.

Both in the recently delivered woman, and in the new-born infant, there exists a general state of the system which creates a *morbid predisposition*, and also a local condition which may give rise to different morbid manifestations, all showing in common one characteristic—the tendency to purulence: peritonitis, cellulitis, and metritis are affections of this kind. Should the purulent tendency invade the venous system of the placenta or umbilicus, the local condition may become the starting-point of a general purulent infection of the system, analogous to purulent infection in one who has undergone an amputation.

Phlebitis and purulent infection are not, nevertheless, in my opinion, all that is necessary to constitute puerperal fever, any more than puerperal fever is peritonitis, as Gasc, Pinel, and Beau suppose, or angioleucitis as is believed by Cruveilhier, Nonat, and Botrel. There does exist an epidemic puerperal typhus; but in every case it must have a wound as its determining cause.

It is necessary, therefore, to establish clinical distinctions. To maintain that all puerperal symptoms result from a fever, or are produced by an inflammation having various localizations, would be to contradict observation.

I hold, consequently, that there are puerperal symptoms, and that there is a puerperal fever properly so called. To-day, I do not intend to speak to you about puerperal fever, as there is no case of it in St. Bernard Ward;

I wish to direct your attention to the purulent infection of recently delivered women, an infection of frequent occurrence; and with several cases of which I meet every year. The young woman whose case I am now going to recapitulate died of that malady.

She was delivered at the Maternity on the 29th November. The labor was natural. On the third day after delivery, she had slight milk fever; but there was no loss of appetite. On the tenth day after delivery, she left the hospital, walking home. Five days after returning to her lodgings, this young woman was seized with shivering, which recurred on the following days; on the 15th December, she was admitted to the Hôtel-Dieu. Immediately after her admission to the hospital, she had a return of the rigors. She nevertheless said that she was not ill, and had no pain in any part of her body. Her pulse was rapid, small, and compressible. She asked for food; and had neither vomiting nor purging. Still, from several returns of the rigors taking place, I suspected purulent infection.

No pain was excited by palpation of the hypogastric region, or by digital examination per vaginam. This absence of pain was abnormal in a woman only fifteen days after delivery. The cervix uteri was soft; the mouth was open, and from it there issued a small quantity of fetid sanious discharge. The uterus was movable. When the patient lay on her right side, it was impossible to detect by digital examination any tumefaction of the broad ligament of that side; as the introduction of the finger occasioned no pain, I neglected to explore carefully the left broad ligament. The prognosis in respect of the fever was very unfavorable from the frequent recurrence of the shivering, and from the patient having no feeling of being ill.

During the night between the 16th and 17th December, she again had shivering. At the morning visit on the 17th, there was a change of expression and pain in the right shoulder; as the arm could be moved about, I concluded that there was no affection of the shoulder-joint, and that the pain was probably dependent upon the presence of a purulent deposit round the articulation. Next day, the 18th, there was a renewal of the shivering; pains were felt in the left shoulder, but in no other part of the body. There existed rapidity of pulse, profuse sweating, and redness over the cheek bones. The mental faculties remained unimpaired; there was neither squinting nor deafness. Respiration was anxious; râles were heard in both lungs, particularly in the lower part of the right lung; there was neither bellows-sound nor ægophony. Vomiting, diarrhœa, and abdominal stains were all absent. Towards evening, respiration became embarrassed, the râles became coarser and more general, the power to expectorate ceased; and death occurred during the night.

At the autopsy, it was found that the uterus was larger and more flabby than it was fifteen or twenty days after delivery; there was, however, nothing abnormal in its parietes. Its internal surface was smooth, and except in the situation where the placenta had been inserted, there was an absence of rugosity; at that point, however, there was no suppurating wound, the corresponding uterine sinuses were contracted on themselves, obliterated by small fibrinous clots; there were no traces in their interior of inflammation or suppuration. The neck of the uterus was bluish and soft, and its lips were jagged; on making a section of the cervix, no inflammation of its circular vein was disclosed. On making a double longitudinal incision through the walls of the uterus where the sinuses and utero-ovarian veins meet, no purulent collection was met with.

On the left wall of the vagina, in the part nearest to the cervix uteri, there were ten pustules, as large as the full-sized pustules of small-pox; a creamy pus flowed from them when incised. In the cellular tissue lining the vagina, in the part corresponding to the pustules, numerous small abscesses were observed, the presence of which gave the tissue the appearance of a sort of purulent sponge. The left hypogastric vein was carefully dissected; creamy pus, free, unmixed with blood, was found in its interior; there was no obliterating clot and no phlebitis, although from the point at which the intra-venous pus was met with, the dissection was carried up as high as the common iliac vein. The pus, which could only come from the parts adjoining the vagina, or from the vagina itself, had been carried into the hypogastric vein, and subsequently drawn into the circulation by the other affluents of the same vein. This case seems to me to be one of the best for the demonstration of the direct passage of pus into the general circulation. This was the visible source of the purulent infection, of which the lungs, liver, spleen, and joints, furnished the irrefragable proofs.

The lung presented numerous ecchymotic spots which gave great value to the existence of small abscesses surrounded by ecchymoses and minute apoplexies. At the base of the lung, at its edges, there were abscesses the size of a haricot, of which some were and others were not fluctuating. The liver and spleen only contained superficial ecchymoses and yellow spots.

Within the articular cavities of both shoulder-joints, there was a large quantity of pus. No appearances of purulent peritonitis or purulent pleurisy were visible.

Most assuredly, gentlemen, you have been struck with the resemblance —the identity, I may say—between the case now described and cases which come under your notice in the surgical wards. Have you not recognized in that case the same purulent infection seen in persons who have undergone amputation? Observation during life and the autopsy, must, I think, have quite satisfied your minds on this point. The symptoms of purulent infection in a delivered woman, as in a person who has had a limb amputated, are frequent rigors, recurring several times a day, pains in different parts of the body, cerebral disturbance, a general state testifying the presence of a general disease, recognizable superficial abscesses, in the joints or subcutaneous cellular tissue, extreme prostration, and speedy death. Metastatic abscesses are found disseminated in the principal viscera, and purulent effusion in several of the synovial cavities. Finally, to complete the description of the resemblance between recently delivered and recently amputated patients, let me state, that in both, there is an opening for the entrance of the poison by the venous system.

In a surgical wound, a placental wound, a tear of the cervix uteri, a contusion of the vagina, a phlegmon, a phlebitis, we have the source of the general infection of the system—the origin of the pus, which, carried into the circulatory torrent, acts either as a foreign body or as a ferment, in such a way as to determine a morbid state of the organism, which almost always terminates in death.

Purulent infection is the same affection in the recently delivered and recently amputated patient. Why, then, does not the placental wound and the surgical wound always give rise to purulent infection?

For a long time, gentlemen, it seemed as if the general affection known under the names of "purulent infection," "purulent absorption," "pyæmia," &c., belonged exclusively to the domain of surgery, because physicians had fewer opportunities of observing it, and because we are indebted to surgeons for the majority of works on phlebitis.

At a date not yet very distant, M. Tessier maintained, with great ability, the theory of puerperal fever in opposition to that of phlebitis.

At a later period, experiments on animals were made by MM. Darcet (de Castelnau) and Ducrest, which were afterwards repeated by Sedillot.

The questions of purulent absorption, phlebitis, and lymphangitis were first supported and then assailed: those who will take the trouble to read the principal works on this subject will find that, looking at Tessier's doctrine from a clinical point of view, it is at least a close approximation to the truth.

This is a question in general pathology which I propose to discuss with you. It is one which every physician ought to study thoroughly; and I hope that our attentive examination of the predisposing and determining causes of purulent infection, will lead us to understand its progress and etiology, and enable us also to seize the indications of a preventive treatment.

Every disease, at the first, has two elements: there is the cause, properly so called; and the state of the economy recipient of the morbid impression. These two elements are always present—the first to follow out its results; and the second, to react upon, to combat the cause. The action of the cause will be principally either in respect of its quantity or in respect of its quality. The reaction of the economy will differ in each individual according to his varying aptitude for resistance.

Even specific causes sometimes encounter refractory economies, which rebel against their morbific powers. This refractory aptitude may be either natural or acquired. It is natural, when we see a number of individuals remain exempt during an epidemic: it is acquired, when, in virtue of a previous attack of the same disease, or in virtue of an antidote, the morbific cause is obliged to remain inoperative. A person who has once had small-pox will rarely have it a second time; and a person who has once been vaccinated is always protected for a longer or shorter period from a second attack.

In some persons, habitual contact with a disease confers relative or absolute immunity from it. Physicians, nursing-sisters, and hospital servants seldom take the eruptive fevers, to the contagion of which they are constantly expôsed.

If a man, day by day, for a certain period, take a particular poison, he will at last be able to take large quantities of it without experiencing the slightest discomfort. In Hungary, as you know, arsenic is very largely used as an invigorating medicine; and, as I have often told you, certain wretched victims of epileptiform neuralgia of the face have acquired so great a tolerance of the preparations of opium that one has been able to take a litre of laudanum in a day, and another from thirty to forty centigrammes of the extract of opium in the twenty-four hours.

Opium and arsenic though toxic agents possessing a determinate, almost specific action, require sometimes to be administered in augmented doses to obtain the desired and usual effect. So it is with specific morbid causes. Though, as a general rule, a specific element acts with certainty and produces anticipated results, yet sometimes specificity claims its quota. Thus, for example, a particular individual who resists contagion when the malady is endemic is unable to do so when a great epidemic is prevailing, because, under the latter condition, the specific cause is more powerful or is more prolonged in its action. This remark applies to epidemics of cholera and small-pox.

The economy will resist an ordinary or specific morbid cause with a power varying according to the individual and the cause: the resistance will vary infinitely according to the varying receptivity of individuals and the varying receptivity of the same person at different times.

These, gentlemen, are principles in general pathology, without which you will be stopped at every step in practical medicine. They enable you to understand how the effects of morbid causes vary according to the idiosyncrasies of individuals, the nature of the prevailing epidemic, or the medical constitution of the season.

The principles which I have now recalled to your recollection admit of being applied to purulent infection in amputated and recently delivered patients? Without these principles, how can we understand the extreme frequency of purulent infection at a determinate epoch? How can we without them, understand its gravity being relative to the nature of surrounding circumstances? Or without them, how can we explain its mildness under certain conditions which do not admit of doubt as to its being actually present, such as articular and numerous subcutaneous abscesses? Are you not thus led at once to perceive the importance of this relative mildness and gravity? Every surgeon and physician knows that there are cases of purulent infection which terminate in return to health. Yet, how often has it been written that purulent infection is inevitably mortal? We shall see by and by whether the etiology, the progress of the infection, and the morbid epiphenomena supply us with indications whereupon to deduce rules of prognosis and treatment.

There is undoubtedly a purulent diathesis, that is to say, a predisposition in virtue of which certain individuals form pus with very great facility, In some subjects, every wound undergoes protracted suppuration: there are others, again, in whom every solution of continuity cicatrizes rapidly. In the former, there is a persistent predisposition to form pus; and in the latter, a natural tendency to secrete that plastic lymph by which the lips of wounds become agglutinated, and their surfaces covered with a cicatricial coat. In the two classes of cases, therefore, there manifestly exists a diversity of predisposition, the particular predisposition being usually allied to a particular state of the general system which it is impossible to define, as those under its influence may present all the conditions of apparent health.

At other times, this general state which constitutes the purulent predisposition, is consequent upon external violence: and at other times, again, it follows an eruptive continued fever from which there has been imperfect convalescence. So great is the alteration in the crasis of the blood after small-pox, that we may see the body covered with a numerous succession of subcutaneous and submucous abscesses. In these cases, the purulent diathesis seems to confine its manifestations to the subdermic cellular tissue; sometimes, however, in cases consequent on typhoid fever, we find, at the autopsy, numerous parenchymatous abscesses. You recollect the case of the young man under the care of Dr. Horteloup, who succumbed to advancing general purulent infection, which probably originated in ulcerated Peyerian glands, and the existence of which infection was proved by numerous pulmonary and intra-muscular abscesses. A prick of the finger when dissecting, sometimes also, though not often, gives rise to purulent infection; more frequently, however, as you know, it gives rise to a phlegmonous or special general putrid infection, which may almost always be recognized by the stools, the odor of which recalls the primary source of the infection.

The disposition to form pus which follows continued fever is not generally a serious element in the prognosis, except, of course, in those exceptional cases in which numerous abscesses form in the muscles and parenchyma of viscera. Perhaps, the cause of the exceptional gravity of purulent infection occurring as a sequel of fevers, consists in some special modification of the wounds which serve accidentally as the origin of the infection, whether it be in a suppurative mesenteric phlebitis, or in an ulcerative phlebitis permitting the direct passage of pus into the circulatory torrent, while the benignity of this affection may be due in other cases to *the digestion of the pus.* How are we to understand why it is that in small-pox, when the dermis is in contact with so great a quantity of purulent matter, there should be no purulent infection, unless we admit that the purulent matter becomes modified in some peculiar manner by which its noxious properties are removed? There is good reason for supposing that, in each pustule, prior to desiccation, a modification takes place in the purulent fluid by which it is deprived of its infectious power.

Whatever there may be in these explanations, it must be admitted that there is a class of cases, in which, notwithstanding the profusion of pus which bathes the surface of the dermis or the deepseated tissues of organs, purulent infection is a very exceptional occurrence; while in another category of cases, the affection originates in a wound of small extent, or in an erysipelas. Let me remark that, in most of the cases which belong to this category, there has been observed a noxious epidemic influence, sometimes manifesting itself in unhealthy suppurations, erysipelas, phlebitis, causing great mortality among lying-in women and new-born infants.

While, however, we attribute its own share to the prevailing epidemic influence, we must admit the existence of individual tendencies, for many amputated patients and many recently delivered women placed under the same general influence escape: those who resist that influence are in the most favorable circumstances for organic resistance. We must, therefore, always take into account the general etiological influence, and the individual's power of resisting it. Though we cannot bring treatment to bear upon the epidemic cause itself, let us see whether it be not possible to place individuals in such conditions as will enable them to resist the epidemic influence.

Principal Theories of Purulent Infections.—1. Absorption of Unaltered Pus by the Absorbent Vessels.—The Pus-globule inadmissible: only the Serum of the Pus is admissible. The Vascular Oscula of Van Swieten and Transverse Sections of Veins becoming Absorbing Mouths.—2. Purulent Fever of Haen and Tessier.—Pyogenic Fever of Lying-in Women of Voillemier.—3. Suppurative Phlebitis causing Purulent Infection of Dance, Velpeau, Blandin, and Marechal.—Capillary Phlebitis of Ribes.—Pus in the Thoracic Duct.—4. Absorption of the Serum of the Pus.—Experiments of Darcet, of MM. Castelnau and Ducrest, and of Sedillot.

Before I state my views of purulent infection, it will not be uninteresting to recapitulate the principal theories which have been brought forward.

The theory of absorption consists in the belief that the pus of an abscess or of a superficial wound may, as pus, be absorbed by the absorbent vessels, and be carried by them into the torrent of the circulation: it is supposed that the pus which is thus transported by the blood to different parts of the body, originates metastatic abscesses. Boerhaave and Van Swieten are of opinion that this absorption takes place by the oscula of the vessels, or by the transverse sections of the veins. The pus mixes with the blood, vitiates it (*inquinat sanguinem*), and so becomes the source of cacoplastic deposits in the viscera, the functions of which become implicated to such an extent as to indicate the presence of the most formidable maladies. Boerhaave and his commentator believe that the pus is absorbed *en nature.* As you are aware, this theory has been most severely criticized. The researches of anatomists hardly allow us to believe that pus can pass directly through oscula, or absorbing mouths, which have no existence! But the entrance of pus in consequence of the lesion of vessels is not the less possible, as Boerhaave observes, *after amputations and operations for aneurisms*, or after extensive and deep wounds—*ingens vulnus factum*. Hunter, in his chapter on "ulcerative phlebitis," defends the opinion of Boerhaave: and every day, we see how easily pus enters the uterine sinuses without there having been obliterating phlebitis.

Van Swieten had the idea that the pus did not merely act as a foreign body in causing the numerous abscesses, but that its presence in the blood produced a *special fermentation* engendering pus in other parts of the body.*

Morgagni, when speaking of wounds of the head, and Quesnay,† declare in favor of the absorption of pus *en nature* by the veins; but they add, that the pus being carried to, and arrested in, the liver, lungs, and other organs, becomes the cause of a secondary suppuration—an opinion adopted by Professor Cruveilhier,‡ based on experiments tending to show that a globule of pus, like a globule of mercury, acts as an excitant of inflammation of the lungs and liver to which it has been transported.

De Haen, the pupil of Van Swieten, but not his follower in this matter, believed in the existence of a purulent fever, *analogous to the fever of small-pox*. He thought that, in these conditions, the blood, in virtue of a special predisposition, contains the principles of purulence, which show themselves everywhere throughout the organism, after the manner of the virus of small-pox. He adds, that the cause originating the buffy coat on the blood is sufficient likewise to produce pus. Around these two opposite theories are grouped all the other theories. One party believes that the organism is infected by pus absorbed at a certain point; and others teach that it is the organism itself which generates the poison, and spontaneously produces pus everywhere.

Tessier, who has defended the doctrine of purulent fever with remarkable ability,§ is satisfied to remark that, in amputated patients and recently delivered women, there is a great tendency to suppuration, a tendency which shows itself, he says, in three quite distinct forms, viz., in the purulent phlegmasiæ, the purulent state, and the purulent fever. He looks upon the purulent fever as a febrile state, under the influence of which active suppurations suddenly appear in various parts of the body.

The object of Tessier's work was to prove that there was no such thing as purulent absorption, and that phlebitis, lymphangitis, and every local phlegmasia, arose from the general purulent state. He considered, that it exists before every fluxion and every local phlegmasia—that the predisposition, the purulent diathesis, originates in the traumatic condition, the puerperal condition, or other serious morbid states of the economy; but that these conditions are only predisposing causes, physical pain and crowding being the determining, and perhaps not the least important, causes.

Gentlemen, I cannot adopt this too exclusive view of purulent fever maintained by M. Tessier, because I am now going to lay before you cases in which it is evident that the suppurative phlebitis, the lymphangitis, and the suppuration of the capillary vessels, were the source of the purulent infection; that is to say, of the infection of the blood. Still, I have pleasure in recognizing the very happy manner in which M. Tessier discusses the question of the purulent diathesis.

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* Van Swieten: Commentaries on the Aphorisms of Boerhaave.

† Quesnay: Traité de la Suppuration.

‡ Cruveilhier: Anatomie Pathologique, du Corps Humain: livraisons iv, vii, xi, xiii: folio; Paris, 1833. See also his article on Phlebitis in the Dictionnaire de Médecine et de Chirurgie Pratiques, t. xii. Paris, 1834.

§ Tessier: De la Diathèse Purulente. [*L'Expérience*, 1838.]

Side by side with Tessier's purulent fever, we must place Voillemier's pyogenic fever of lying-in women.*

The doctrine of phlebitis has more scientific precision than that of purulent absorption; but resembles it in this respect, that it admits the infection of the organism to be consequent upon the introduction of pus into the blood. Dance, in 1828, ably maintained this theory, which, notwithstanding the powerful opposition of Dupuytren, was generally accepted.

The starting-point, according to this theory, is suppurative inflammation of a vein: the infection of the blood is looked upon as secondary, and the metastatic abscesses, so to speak, as tertiary symptoms. This phlebitis, already described by Hunter, was anew investigated; and pathological anatomy soon showed that the bodies of patients dying with symptoms of purulent fever presented phlebitis of the stump, phlebitis of the capillaries, or phlebitis of the uterus. The cause of the phlebitis was discovered: it was discovered in the parenchyma of organs, in the osseous tissue, the diploë of the cranial bones, and in other situations.

But is not this phlebitis, the source of the infection, itself really a primary manifestation of a special predisposition, without which the vein could not become the seat of inflammation? Without this predisposition, suppurative inflammation would then be the necessary consequence of every lesion of a vein: but this is not the case—suppurative phlebitis is, relatively, a rare complication.

Whenever a vein is involved in an operation, one of two things must occur: either the secretion of plastic lymph, the wound healing by the first intention; or, cicatrization by the second intention, after a necessary preliminary stage of inflammation. In the first case, the lips of the vein in the wound are glued to one another; and this condition is maintained by fibrinous plastic lymph secreted by the internal surface of the vein: this is adhesive inflammation. In the second case, the vessel remains open: a cruoric clot, adherent to the internal surface, projects from the opening, and mingles with the secretions from the surface of the solution of continuity. This clot has a free external extremity, and an intra-vascular extremity adherent to the serous coat of the vessel. The length of the clot varies with its distance from the point at which the collateral venous circulation is established in the same vessel.

What occurs in the second class of wounds takes place in respect of the placental surface of the uterus, which may, at every point, be compared to a bleeding wound. In both, obliteration of veins is accomplished by coagulation of blood. In the uterine sinuses, the obliterating clots seldom measure more than from one to two centimetres, which is explained by the rich venous circulation of the uterus for some days after delivery. But should venous inflammation occur, and spread by degrees, it is usual to find clots prolonged into the utero-ovarian and hypogastric veins, as is sometimes observed in the brachial and femoral veins, after amputations of the arm and thigh.

The secondary clots undergo very remarkable transformations; but it is not known whether these clots have the power of inducing purulent infection. My opinion is that purulent infection generally originates in inflammation of the vein nearest to the solution of continuity, that is to say, in the wound itself. This, in fact, is the situation in which the existence of pus has been demonstrated; and if there be no fibrinous plug, there is a direct passage for the pus into the blood. This fact admits of easy demonstration in the uterine sinuses and hypogastric and ovarian veins of women who have died within three weeks after delivery. Again, in these circumstances, it is not unusual to see in the placental wound several large open sinuses with thickened walls, canals of communication by which is conveyed the pus of the placental surface, and the pus, free or mixed with blood, which is found in the hypogastric and ovarian veins. In these cases, there is observed in the veins nearest to the source of pus, a sanguineous fluid identical in color and consistence with that of leucocythæmia. These facts were observed by Hunter, Clarke, and Hodgson, and are mentioned in their respective works: they may be easily confirmed by any one who will carefully examine the lateral parietes of the uterus in a recently delivered woman, and the veins nearest to the surface of the wound in a case of amputation.

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* VOILLEMIER: Histoire de la Fièvre Puerpérale, Pyogénique, observée en 1838, à l'Hôpital des Cliniques. Reprinted in 1862 in the "Clinique Chirurgicale."

Can the elongated clots in the veins be the source of purulent infection? In favor of an affirmative answer is the fact, that these clots, in softening, often present a purulent appearance; but I am doubtful of this being the source of the infection, because we can only detect by the microscope, even in the midst of this purulent appearance, granules of fibrin, no pus-globules being visible.

Suppurative phlebitis properly so called, which is observed chiefly in the large veins near wounds, may also exist in the capillary veins, as has been demonstrated in cases of ostitis and caries. Ribes observed capillary phlebitis in cases of erysipelas and gangrene.

We have now seen that pus deposited on the surface of a wound, or contained within a vein, passes directly into the torrent of the circulation, and so originates purulent infection. Are there any other sources of this infection?

During the last few years, there has been a belief that there can be no purulent infection without suppurative phlebitis, the purulent inflammation of the vein being the necessary cause of the infection: without that inflammation, it has been held that there can be no passage of pus into the blood. After delivery, as I have already remarked, pus may be met with in the uterine veins, although these veins are not inflamed, not even at the point where pus is seen. It must, therefore, be admitted, that in these cases, the pus has been transported to the place where it is found. Again, I lately showed you a beautiful example of this transport of pus in the hypogastric vein, occurring in a case of purulent infection following delivery. It may thus happen, that one of the afferent branches of the hypogastric vein may be the seat of suppurative inflammation. I believe that it so happens in the majority of cases. For this reason, I wish to recall to your recollection two somewhat interesting cases, which prove that pus may exist in these veins, and may nevertheless escape the notice of conscientious attentive observers.

Lenoir, surgeon to the Hôpital Necker, had performed ablation of a sarcocele. Complete cicatrization seemed to have taken place, when the patient was seized with repeated attacks of shivering; and died, having metastatic abscesses, which were seen on dissection after death. Lenoir himself dissected the veins which he supposed to be in a state of inflammation; and, after two hours of fruitless search, was unable to recognize any signs of phlebitis. Dr. Gubler, at that time a pupil in the wards, continued the dissection, and discovered inflammation of the prostatic venous plexus. The pus contained in the plexus freely discharged itself into the hypogastric vein.

In Marjolin's service, at the Hôpital Beaujon, a man was bled from the arm: he soon had pain in the arm, and probable phlebitis. Symptoms of purulent infection set in; and the patient died. MM. Castelnau and Ducrest carefully dissected the veins, in the situations in which they expected to find phlebitis: for four hours their search was fruitless. M. Castelnau then anew examined the veins of the arm, and found pus in an inflamed vein opening directly into the brachial vein, in which there was an obliterating clot, and a free passage for the pus into the subclavian vein.

In both of these cases, the existence of phlebitis would have been denied, had not renewed search been made; and, certainly, no renewed search would have been instituted, had not a firm conviction existed in the minds of the observers, that pus would be discovered, to show the source of the general purulent infection. In similar circumstances, therefore, we cannot make our examinations with too much minuteness.

How many patients die after catheterism with symptoms of purulent infection! In the autopsy of such cases, never omit to examine the prostatic plexus, and the whole venous system of the pelvis, for there, the source of the infection will probably be found. You ought to know where to look. Recollect, that these are the chosen spots where pus is present in uterine phlebitis, as well as in phlebitis following operations on the testicle, urethra, bladder, and prostate.

It does not follow, that phlebitis is the sole source of purulent infection. In cases of purulent infection observed by Velpeau, Nélaton, and Denonvilliers, numerous metastatic abscesses were found on dissection after death, and yet, no pus could be found in the veins near the seat of the operation.

For example, amputation was performed in the case of a man admitted to the wards of Dr. Denonvilliers for comminuted fracture of the humerus. He died fifteen days after the operation, with all the symptoms of purulent infection. At the autopsy, enormous abscesses were found in the liver. All the superficial and deep veins of the upper extremity, from the surface of the stump to the right auricle of the heart, were very carefully dissected without the smallest trace of pus being discovered. The remaining portion of the humerus was sawn in several directions; but not the minutest drop of pus was found in the spongy tissue of the bone.

Though disposed to admit the great value of cases such as I have now described, I am not without doubts as to their negative importance. It is possible, that the primary source of the infection may have existed in some situation which was not thought of. The sinuses of the dura mater may be the seat of purulent deposits consequent upon a seemingly unimportant attack of otorrhœa: again, in the course of a vein, there may be suppuration of a gland, cellular tissue, or parenchyma; and, from ulceration taking place, pus may enter the vein, causing general infection, although the lesion of the vein may remain undiscovered.

At other times, the source of the infection has been in the thoracic duct or in the receptaculum chyli. How did the pus get there? That matters little: there it was; and thence it might easily pass into the subclavian vein, and so into the venous circulation. In these cases, there was purulent infection.

You see then, gentlemen, that suppurative phlebitis plays an important part in purulent infection. Pus may, however, exist in the veins without any trace of inflammation of the walls of the veins having been found: at other times, we neither find pus in the veins, nor phlebitis: but in such cases, the thoracic duct and lymphatic vessels have not always been examined; and as we have just seen, these affluents of the venous system may contain pus in cases of purulent infection. It consequently follows, that in the great majority of cases, purulent infection simultaneously exists along with pus in the venous system and its affluents. Adulteration of the venous blood by pus appears then to be the proximate cause of purulent infection: this theory has on its side an imposing majority of facts.

Nevertheless, the partisans of purulent fever see in them only coincidences; or rather, they regard metastatic abscesses and phlebitis as consequences of a general purulent state. From their point of view, there is no relation of cause and effect between the surgical or placental wound and the metastatic abscess, both being looked upon as results of the same cause, and as similar in their nature though different in their seat. The objection to this interpretation of the facts is, that purulent infection never exists without a wound, however small that wound may be. On the other hand, to this objection a counter-objection may be urged, viz., that there are many wounds without purulent infection.

Here, let me remind you, that certain conditions are necessary for the production of *suppurative phlebitis:* with most of these conditions we are unacquainted. But this we know—that every wound of a vein is a grave accident, liable to become the source of the greatest danger. Surgeons have observed, that the knife is more apt to produce suppurative phlebitis than the actual or potential cautery; and that almost never is there anything to fear from venous lesions produced in crushing or tearing operations. We also know, that intra-venous suppurations, which predispose to purulent infection, are induced by comminuted fractures and articular wounds, by any active interference with the uterus during labor or at the time of delivery, by partial retention of the placenta, or by extensive lacerations of the cervix uteri. I may add, that individual and hygienical conditions, crowding, and the pain of an operation, by their action on hæmatosis and the nervous system, create a predisposition to purulence. All this I accept within certain limits; but as you know, cases are recorded in which purulent infection declared itself almost suddenly in persons whose general state was satisfactory: some were suffering only from otorrhœa,* varix, corns, or chilblains. Spontaneously, or consequent upon intervention with the knife or ligature, a vein having been touched, suppurates, and the patient dies with symptoms of purulent infection. Persons in good health, with the exception of some insignificant malady, are operated on in the most favorable conditions—a vein becomes inflamed, and the patients die: metastatic abscesses form: pus is found in the petrous portion of the temporal bone, in a vein at the point where it was ligatured, or only above the ligature. Now I ask, are we, with all these facts before us, not to see the relation of cause and effect existing between purulent infection, between the general state, and the wound? That would be entirely to abandon logic.

Nevertheless, doubt was permissible: it was necessary to have recourse to experiments. At a period when attention was keenly directed to alteration of the fluids caused by the entrance of putrid matters into the circulation, Gaspard in 1823, and I, in conjunction with M. Dupuy of Alford, in 1826, came to the conclusion, that the injection of putrid matters into the veins of animals (dogs, horses, and sheep), produced symptoms similar to those caused by purulent infection, leading to death in a few days when the quantity injected was sufficient. On examination after death, the blood was found exceedingly altered, and numerous ecchymoses were observed in the parenchyma of the viscera and the subserous cellular tissue. After injecting from thirty to forty-five grammes of putrid pus into the veins of animals, we found tumors presenting a carbuncular appearance, and abscesses, which we called tuberculous abscesses [*abcès tuberculeux*]. I merely, at present, mention the results of the experiments performed by M. Dupuy and me conjointly, remarking, that when we made them, we had no intention of producing a state of poisoning similar to purulent infection.

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* An interesting case of this kind is described at p. 183 of volume first.

MM. Renault and Bouley, in 1840, injected two centilitres of pus into the jugular vein of an apparently healthy mare; and, after the lapse of a few days, were not a little surprised to see the symptoms of acute glanders: it is worthy of notice, that by simply inoculating the matter of the nasal ulcerations of that animal, acute glanders was produced in another animal. I do not enter into any discussion regarding this experiment: I am satisfied to state the fact.

M. Darcet in 1842, and MM. Castelnau and Ducrest in 1843, repeated these experiments upon dogs and rabbits. In 1844 and 1845, M. Sedillot made similar experiments with pus derived from different sources.

All these experiments show, that most animals into whose veins pus, varying in quantity and quality, has been injected, have presented, from the very first, prostration, a dejected appearance, and great restlessness: they have nearly all refused to eat, and have been exceedingly thirsty; some have had paralysis of the sphincter of the anus. Before the reaction which indicated a return to health, they have had profuse alvine discharges. When, by repeating the injections, the animals were subjected to successive and progressive poisoning, they succumbed in from five to ten days after the commencement of the experiment: on dissection, ecchymoses were found, particularly on the surface of the lungs, and numerous abscesses, of different dates, in the same organs.

It appears then, gentlemen, that whether we inject pus into the veins of an animal, or whether it enter a vein as the result of inflammation in the case of a recently amputated or recently delivered patient, the symptoms and the lesions are identically the same. Purulent infection is produced artificially in the first case; and is the result of phlebitis in the second case. What more is necessary to prove, that in both cases the presence of pus in a vein is the cause of the purulent infection? This double check, clinical and experimental, is almost as conclusive as the experiments made to elucidate vaccination, the inoculation of small-pox, of syphilis, and of glanders.*

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* For the history of this question, and for all the experimental and clinical researches, see DARCET: Thèse Inaugurale, Mai, 1842: CASTELNAU and DUCREST: Recherches sur les Abcès Multiples comparés sous leurs différents rapports; Paris, 1846. SEDILLOT: De l'Infection Purulente ou Pyohémie; Paris, 1849. This work, rich in experiments and clinical observations, is the most complete treatise on this subject. DUMONTPALLIER: Thèse Inaugurale; Paris, 1857. See also, the works of BOERHAAVE, VAN SWIETEN, DE HAEN, STOLL; and the more recent publications of BLANDIN, VELPEAU, CRUVEILHIER, and TESSIER.

Doctrinal Statement.—Parallel between Experimental Purulent Infection and Clinical Purulent Infection.—Similarity of Symptoms and Anatomical Lesions.—Similarity of the tendency to Critical Evacuations by the Skin and Intestines.—Possibility of Recovery from Purulent Infection: Complex Etiology of Purulent Infection from Inflammation of the Large and Capillary Veins: from Absorption of Pus itself: from Absorption of Purulent Serum, Assimilated, or Poisonous Serum.—Epidemic Purulent Fever.—Theory of Ferments applied to Purulent Infection: Experiments of Pasteur, Chalvet, and Reveil.—Treatment of Purulent Infection: to avoid the Causes of Phlebitis: there is no Specific: Endeavor to excite Crises, and to support the Strength.

Be on your guard, when successive attacks of rigors occur in a newly delivered woman, or in a patient who has a wound in any part of the body: for then, there is reason to fear that a certain quantity of pus has entered the blood, and that pyæmia is threatened. The shivering is soon succeeded by fever, sometimes a copious sweat covers the whole body, or a profuse diarrhœa sets in, after which all seems to return to its normal state, and the idea arises that an unnecessary amount of alarm has been created. Wait twenty-four or forty-eight hours! Rigors will then set in anew, sometimes accompanied by chattering of the teeth and horripilation; and these symptoms will perhaps recur several times within a few hours. The general discomfort increases, the patient has a presentiment of approaching death, reaction takes place slowly, and the pulse is rapid and weak. At other times, were it not for these strange shiverings, the patients would make no complaint; and yet, they suddenly experience pain in some particular part of the body, such as the calf of the leg, the knee, or a joint. The swelling and redness which accompany the pain disappear in a few hours; and then other parts of the body are invaded. Under such circumstances, you need not hesitate to affirm, that purulent infection exists, that is to say, that pus has entered the blood, and that there are abscesses in the parenchyma of the viscera, in addition to those visible on the limbs and trunk: nearly always, several metastatic abscesses are found in the lungs and liver.

Anatomical experience enables us to state that these abscesses exist, when by mere clinical investigation we could hardly even suspect their presence: it is not unusual for such abscesses to exist in organs although the functions of the organs do not seem to be disturbed. The liver or lungs may contain abscesses in different stages of evolution; and yet, pain in the hepatic region and chest, cough and expectoration, may all be absent.

Metastatic abscesses at the base of the brain may be sometimes diagnosed by the occurrence of strabismus, by an inequality in the dilatation of the two pupils, and by the patients complaining of diplopia and obscurity of vision. Delirium and coma do not show themselves till the last stage: at the commencement of the purulent infection, they afford no assistance to the diagnosis, but at that early period, important cerebral disturbance may be observed. There is a discord between the quiet state of the patient and the gravity of his condition: he cannot understand why he is so carefully and attentively interrogated: he will tell you that he is not ill. This quiet state is a prognostic of very great danger. You remember the young woman who occupied bed 20, St. Bernard Ward, who told us that she was not unwell: and in respect of whom, nevertheless, I had no hesitation in affirming, on learning that she had had repeated rigors, and ascertaining the date at which she had first been attacked by them, that the blood was poisoned by pus, and that the source of the infection was in the genital organs, although I had not been able to recognize decided pain in these parts.

It is generally after the fourth or fifth day from delivery, that the symptoms of purulent infection declare themselves, because three or four days are required for the pus to be secreted in sufficient quantity from the surface of a wound or within an inflamed vein. When the symptoms do not appear till the fifth day, the delay has probably been caused by the obliterating clots having in the first instance prevented the pus from entering the blood.

It is easy to understand, that when four days have elapsed after delivery, the date of commencement may vary very much; for once pus has been secreted at any point in the venous system, it may enter the torrent of the circulation by the obliterating clot being broken or displaced by any sudden movement or blow. Persistent inflammatory action may cause softening of the clots, without there being a sufficient contraction of the vessel upon itself: then the pus secreted on the other side of the broken or softened clot may be poured intermittingly or continuously into the circulation.

There is a remarkable similarity between the principal symptoms of purulent infection and the phenomena observed in the experiments of Darcet, Castelnau, Ducrest, and Sedillot.

Each rigor announces the entrance of pus into the blood: for a time, variable in duration, the patient, or the animal experimented upon, struggles to eliminate the morbid poison from the circulation: profuse sweating, diarrhœa, and vomiting supervene: but as a new supply of the infecting fluid is always pouring in from the source, the patient becomes exhausted in useless efforts, and the great functions of the economy become almost simultaneously compromised: the stomach rejects food and medicine: there is constant diarrhœa: the circulation and respiration become accelerated: the mental faculties gradually fail: the face does not bear the impress of suffering: by and by, the breathing becomes jerking and quicker: the pulsations of the heart are more rapid: the body is covered with a cold sweat: at last, the patient dies without a struggle. The calm which characterizes the last scene is perhaps explained by the blood being poisoned to such an extent as to carry death simultaneously to every organ, so rendering it impossible for any of them to maintain the struggle. Every part of the body, even the parts which seem to be perfectly healthy, are impregnated with the morbid principle, which seems, as Bérard remarks, to manifest itself by the repugnant smell which issues from every part of the body a few minutes after death.

Death is almost always the prognosis in purulent infection. It is obvious, that if a large quantity of pus be introduced into the economy, and carried to every part of the organism, it must speedily produce changes incompatible with life. On the other hand, it is easy to see that a small quantity of pus will not be sufficient to infect the whole of the blood: the infected portion may then be eliminated by the stools, by sweating, or by the renal secretion, in which case, the symptoms of infection will cease, provided there be no more of the infecting cause admitted.

This arrest may occur even when metastatic abscesses have formed: and after a time, these abscesses may themselves be absorbed without any new metastatic manifestation.

You recollect the young woman, aged twenty-eight, who after delivery at the Hospice des Cliniques, was admitted to our St. Bernard Ward, on the twelfth day after delivery, with all the symptoms of purulent infection, viz., recurrent shiverings, diarrhœa, vomiting, a subicteric tinge of the skin, and metastatic abscesses under the skin as well as in the sterno-clavicular and metacarpo-phalangeal articulations. All at once, the symptoms were arrested: the patient's general state improved: the fever, vomiting, and diarrhœa ceased: there was no recurrence of shivering: the appetite returned, the subicteric tint disappeared, and we saw that, little by little, *digestion* of the superficial, intra-articular, and subcutaneous metastatic abscesses was being accomplished. This *digestion* took place exceedingly slowly. Nothing interrupted the progressive return to health.

In this patient, in whom there was uterine phlebitis and entrance of pus into the blood, it is probable that the continuance of the passage of pus into the blood was prevented, either by a change occurring in the relation of the affected parts, or by obliterating phlebitis taking place beyond the seat of suppuration: as the infection was arrested, and its cause ceased, whilst the struggle was still possible, the patient triumphed over the first morbid effects. I was, therefore, justified, gentlemen, in reminding you, at the beginning of these lectures, that in the practice of medicine we must take into account the quantity as well as the quality of morbid causes, and likewise individual aptitudes for resistance. The case which I have now detailed strengthens this precept in general pathology.

Let the case of this young woman remain impressed on your memories, to make you less absolute in your prognosis, and to prevent you from losing all hope in these cases.

Recovery, then, is possible in a case of purulent infection. The experiments on animals led us to this belief: MM. Castelnau and Ducrest have shown, that animals into whose veins insufficient quantities of pus were injected, returned to health after suffering for two or three days from fever and diarrhœa, whilst those into whose veins a succession of injections were thrown died with the anatomical lesions of purulent infection.

With such facts before the mind, it is natural to believe that death is not the inevitable issue in every case of purulent infection. What are the conditions required to modify the prognosis of an inevitably fatal issue in purulent infection? Arrest in the action of the morbid cause, and sufficient capacity of resistance on the part of the patient.

From this statement arise two leading indications of treatment: *first*, to diminish, and if possible suppress the source of the infection; and, *second*, to supply the patients with necessaries for the struggle.

In discussing the theories of purulent infection, I have, as I proceeded, discussed all its conditions. There now, therefore, only remains for me to present a dogmatic summary of its etiology.

Gentlemen, you recollect that, at the commencement of these lectures, I laid before you my views regarding the disposition to make pus which is observed in some patients, particularly in recently delivered women. There is often nothing to indicate the existence of this special predisposition; and women, during the whole puerperal state, especially after delivery, have this purulent predisposition in a more marked degree than at any other time. In them, every inflammation is apt to pass into suppuration, as is seen by the course of their peritoneal, pleuritic, phlegmonous, and arthritic attacks. In such persons also, when inflammation attacks the venous system, it is not unusual to discover pus in the veins.

Before there can be purulent infection, there must be a wound, and it is also generally required that veins of a certain size should be involved in the wound; but then the primary essential state is phlebitis, with the possibility of pus passing into the blood. Pus is seldom absorbed from the surface of wounds by large veins; but it may sometimes take place in suppuration of the placental wound. In that particular case, there is a special organic structure which may keep the uterine sinuses open. Still, that mode of purulent absorption is necessarily exceptional; for if the sinuses were always open, there would be uterine hemorrhage. I am rather of opinion that in this class of cases there is inflammation of the sinuses, and that this inflammation may give rise to infecting phlebitis. During the first days after delivery the placental wound is rough with small projections constituted by the free extremities of the intravenous clots, the length of which seldom exceeds one centimetre: these clots show a remarkable tendency to have their placental extremity purulent, and their intravenous extremity cruoric; they are adherent to the internal surface of the sinuses, and are kept in place externally by their relation with the placental surface.

When the course of events is normal, the placental portion of the clots is eliminated, and the venous portion is absorbed; while at the same time the vessels diminish in calibre, under the influence of the progressive and continuous contraction of the womb. But if suppurative inflammation affect the walls of some of the sinuses, there will be reason to fear infecting uterine phlebitis.

I have already said that what takes place within the uterine sinuses, in the situation of the placental wound, also occurs on the surface of wounds uniting by the first or second intention, though in a manner less evident, in consequence of the veins being there less numerous. If the wound heal by the first intention, there will be adhesive phlebitis, or merely an effusion of plastic lymph sufficient to establish adhesion of the walls of the veins. Should the wound suppurate, phlebitis is then necessary, and should the phlebitis be suppurative beyond the wound, there will be reason to dread purulent infection.

Ribes, Neucourt, and Velpeau regard erysipelas as an inflammation of the radical veins of the skin. As you know, erysipelas of new-born infants is prevalent during epidemics of puerperal fever. Again, both traumatic and spontaneous erysipelas is specially prevalent when surgical wounds complicate purulent infection. If then, we observe not only the simultaneous occurrence of erysipelas and purulent infection, but likewise their fellowship at a determinate period, are we not somewhat warranted to inquire, whether there does not exist in the atmosphere a morbid germ, which being deposited on the umbilical, or placental, wound, or on any surgical wound, will produce in one case phlebitis, and in another erysipelas, with or without purulent infection?

According to M. Charles Robin,* the pus-globule is simply a leucocyte possessing no particular property; and according to him, it is the purulent serosity which gives infectious properties to pus. If it be admitted, that the serosity of normal pus modified by a germ, a morbific spore, floating in the atmosphere, is of itself sufficient to give rise to purulent infection, that serosity will then be held to have the power similar to those serosities which are charged with a special virus, such as variola, vaccinia, or glanders, and the minutest wound will suffice for the absorption of this new poison.

Absorption of the thus modified serosity will explain the cases in which purulent infection is produced with its usual characteristics without any trace of suppurative phlebitis being found; just as the hypothesis of a morbific ferment floating in the atmosphere explains the epidemics which simultaneously strike down amputated patients and lying-in wonen. The works of Pasteur,* Reveil,† Chalvet,‡ and d'Eisell, show the very probable correctness of this hypothesis.

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* Robin (Charles): In the Journal de la Physiologie, for 1859, p. 62.

M. Pasteur has shown, on the ohe hand, that the real ferments are organized bodies, the aliment of which is albuminoid matter. He has shown, on the other hand, that the dust floating in the atmosphere contains starch and vegetable and animal spores, capable of attaining life. The same philosopher has made it appear, that special spores are necessary for the production of special fermentations; and that these different ferments are recognizable by their form. The spore is an organized, living cellule vegetating upon the elements which surround it: but to enable this vegetation to take place, it is necessary that certain determinate conditions exist in the medium in which the germs are placed. When you have once determined these conditions, you may, by employing the spores of the alcoholic, acetous, and lacteous ferments, obtain respectively the alcoholic, acetous, or lacteous fermentation.

Here, then, is the great theory of ferments, by which fermentation is ascribed to an organic function. Every ferment is regarded as a germ, the life of which is manifested by a special secretion.

Perhaps, this is so likewise in respect of morbid viruses. Possibly, they are ferments deposited in the organism, which at a given moment, and under certain determinate circumstances, make themselves known by the multiplicity of their products. Thus, the variolous ferment will give rise to the variolous fermentation, in the midst of which a profusion of pustules will appear: so is it with the viruses of glanders and tag-sore.

Other viruses seem to have a local action; but in course of time they do not the less modify the entire organism: as examples, I will mention hospital gangrene, malignant pustule, and contagious erysipelas. Under these circumstances, may we not admit that the ferment, or organized matter of these viruses, is transported to one place by the lancet, and to another by the air or the dressings?

These statements are not mere hypotheses: chemical analysis and the microscope prove the existence of morbific dust in hospital wards. Thus, M. Chalvet, in his interesting researches into the causes of the insalubrity of hospitals, found that the air of the Hôpital Saint-Louis contained a large quantity of starch-corpuscles: besides, on the walls, window-frames, curtains, and beds, there was found, by the aid of the microscope, a great quantity of putrescible organic matter. It has been stated by the same observer—and I have lately had the opportunity of confirming his remark—that dressings returned from the lavatories have been found still soiled with organic detritus, with linseed, and with spots, recalling the uses to which they had been formerly applied. Might not these linens, stained with blood and altered pus, be vehicles of contagion? Was it not an old custom to preserve vaccine matter on linen and cotton threads? Imagine the consequence of using the imperfectly washed linen of small-pox patients!

M. Chalvet has also stated, "that the vapor of water condensed near a source of suppuration is, prior to the dissemination of miasmata, greatly charged with irregular corpuscles, in all respects resembling dried pus. It is not unusual to find there some fragments of the coloring matter of the blood."§

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* PASTEUR: Annales de Chimie et de Physique.

† REVEIL: Des Désinfectants, et de leurs Applications à la Thérapeutique; **Paris**, 1863.

‡ CHALVET: Des Désinfectants, et de leurs Applications à la Thérapeutique et à la Hygiène. [*Mémoires de l'Académie Impériale de Médecine*, 1863, t. xxvi.]

§ CHALVET: Op. cit.

Eiselt (of Prague) states that he saw small pus-cells in the air of a ward occupied by patients with epidemic purulent ophthalmia.

On this subject, M. Chalvet, in reporting to me the result of his experiments, thus expressed himself:

"The *nosocomial atmosphere* has now ceased to be a term without meaning: the air of an hospital differs so essentially from pure air, that attention to the fact can no longer be neglected. Since 1860, I have had an opportunity of witnessing the experiments of M. Reveil (quoted by M. Devergie); and have satisfied myself, in the most positive manner, that organic corpuscles were present on the platinum plates of the apparatus constructed by that chemical savant. I saw on them principally cells, débris of epithelial cells, corpuscles of different forms (which assumed a yellow color under the action of nitric acid), and shreds of charpie charged with the same organic corpuscles.

"On one particular occasion, I saw, along with M. Kallmann, in M. Reveil's laboratory, incrusted organic débris, and a granular substance which produced a reaction with copper. The dust examined had been collected in an ophthalmic ward where the sulphate of copper was extensively used as a caustic.

"The dust dusted off the walls [*par l'époussetage sur les murs*] of St. Augustin Ward, in M. Richet's service at Saint-Louis, yielded thirty-six per cent. of organic matter in my first analysis. Dust taken from the same walls upon another occasion, and analyzed in M. Reveil's laboratory, yielded forty-six per cent. of organic matter, consisting chiefly of epithelial cells exhaling the odor of burnt horn. On moistening the dust so collected, a strong putrefactive odor was exhaled. No doubt, the vast coat of mixed dust which clothes the rarely cleaned walls of the wards in the old hospitals, has the power of generating gases calculated to favor the aerial transport of bodies which perhaps play an important part in the constitution of the nosocomial atmosphere."

These facts are, in my opinion, very significant. There has always existed, from reasoning, a strong disposition to attribute much in the spread of endemics and epidemics, to the transport of morbific molecules; and now, the same conclusion has been arrived at from direct examination of the atmosphere. With some justice, M. Pasteur remarks: "It would be very interesting to compare the organized bodies disseminated in the atmosphere in the same place at different seasons, and in different places at the same period. It appears to me, that we would gain an increased knowledge of the phenomena of morbid contagion, particularly during the progress of epidemics, by researches pursued in that direction."

The germs which may exist in the atmosphere at a given time, under circumstances to be determined by future inquiry, will not become developed with equal facility in all patients, because persons differ exceedingly in respect of the condition of receptivity: certain organisms, like certain soils, will not accept certain germs. Throughout a country, the winds have diffused the same seeds; and yet the same seeds do not spring up everywhere. In one situation, the ground is too wet, and in another it is too dry: in some places other seeds having already grown up, the new seed is choked: or, perhaps, the seed springs up wherever it falls: in one place there is too much light and too much heat: in another place the temperature is too high, while in another it is too low, so that here the plants are puny, and there they are strong.

Before inquiring whether purulent infection can be foreseen or arrested, let me speak to you of the state of the blood.

The blood in purulent infection often presents a brown chocolate color.

In different parts of the venous system, especially in the right side of the heart, there are cruoric clots of the consistence of pitch, in the midst of which are seen small whitish masses, which might be mistaken for coagulated fibrin. Many observers have been nearly certain that these post-mortem modifications in the consistence and color of the blood were attributable to the presence of pus in the blood. Recently, M. Donné, and afterwards, M. Bouchut, published cases in which they made out, that in purulent infection, we can recognize the presence of pus in the sanguineous fluid: in fact, they described bodies identical in dimensions and in microscopic appearances with pus-globules properly so called. It appears then, that the presence of pus in the blood may sometimes be detected, but that the search for it is often fruitless.

Some micrographers, however, hold that the supposed pus-globules are only the white globules of the blood, which, under certain conditions, unite and become deposited after death, in small masses, within the sanguineous clots.

In cases of purulent infection following delivery, I have observed—as had already been noticed in cases of suppurative phlebitis—that the blood of the inflamed veins had a chocolate color, like the color of the blood of patients affected with leucocythæmia: this coloration of the blood was less and less marked, the greater its distance was from the seat of suppuration in its onward passage to the heart.

Several times, I have detected pus mixed with blood in the hypogastric and common iliac veins, up even to the vena cava inferior. Note well, that in these cases there was no obliterating clot.

At other times, this purulent mixture, verified by microscopic examination, was in the ovarian veins, and I have demonstrated it in the left ovarian vein, up to its junction with the emulgent vein. Thus, one could follow step by step, so to speak, the gradual decrease of this special coloration of the blood, up from the uterine veins filled with pus to the vena cava inferior or emulgent vein. Then, if we at the same time examine with the microscope the blood supposed to contain pus, and the purulent fluid of suppurative phlebitis observed in the same subject, we find in both absolutely identical pus-globules: these globules, however, are fewer in number, the more remote from the seat of suppuration is the place from which the blood is taken. The fact was observed by Hunter.*

The examination, then, of the blood after death leaves no doubt in my mind. I am convinced that it can be shown, after death, that the blood of patients who have died with purulent infection, is contaminated by pus in varying proportions.

But M. Charles Robin has come to the conclusion, that the white globules may be formed in every part of the organism, and that there is no difference between the leucocyte and the pus-globule. M. Robin thinks that leucocytes give pus its color, but not its nature, the latter being due to the fluid of which it is principally composed. He holds that pus derives its essential characters from the serum, and not from the leucocytes.

This assertion involves two allegations, viz., that the pus-globule and the leucocyte are identical, and that the special nature of pus is derived from its serosity.

In respect of the identity of the pus-globule and the leucocyte, I must say, that I have several times examined the blood of patients affected with diseases in which M. Robin has pointed out the frequency and comparative identity of leucocytes, and that I have sometimes met with very decided differences, a circumstance which I cannot omit mentioning—for example, in the puerperal state, in confluent and non-confluent small-pox, in paludal cachexia, and purulent infection.

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* HUNTER: Œuvres Complètes; traduites par G. Richelot. Paris, 1848.

In all the patients embraced in this comparative study, the blood was taken from a prick at the end of one of the fingers, and was received on glass plates. On every occasion, the microscopic examination of the blood was made quite close to the patient's bed. Here are the results of my observations:

In a little child, who presented clinically all the symptoms of hemorrhagic leucocythæmia, I found a very great number of large white globules, from twenty-seven to thirty white globules to each preparation. Each of these leucocytes, being fixed on the field of the microscope, was found to measure ten, twelve, or thirteen thousandths of a millimetre by Nachet's micrometer, using a magnifying power of five hundred and eighty diameters. The leucocytes were formed of a cell containing numerous nuclei. The red globules were of natural form, and of the normal dimensions, varying from the six to the seven thousandth part of a millimetre.

The blood of several small-pox patients was examined at the time the eruption was coming out, and also during the period of suppuration. The pus-globules were identical in dimensions and appearance with pus-globules taken from the pustules: these globules did not measure more than the six, seven, or eight thousandth part of a millimetre: there were from six to twelve of them in the field of the microscope, and it was with great difficulty that I detected on some of the preparations one or two white globules of from ten to twelve thousandths of a millimetre.

In a woman, who died after presenting the symptoms of purulent infection, examination of the blood before and after death, showed a great number of pus-globules, and very few large white globules, in the blood. It does not seem necessary to give you an abstract of my other similar, and similarly conducted, observations. I only wish to remark, that I consider that these inquiries seem to have established the existence, in certain cases, of pus-globules properly so called, and that the large white globules were few in number. I must add, however, that in other researches, I observed a greater number of white globules, both in small-pox patients and in lying-in women suffering from purulent infection.

Great consideration is due to the importance accorded by M. Robin to the serum of the pus. That importance has already been manifested in the case of the virulent pus of variola and glanders, the respective specific differences of which have been demonstrated by inoculation. These differences also certainly exist in respect of benignant and malignant pus; that is to say, between pus the serosity of which when absorbed, does or does not produce general infection. The considerations into which I entered, when speaking of wounds modified by the germs or specific spores, seem to prove that these differences exist.

In purulent infection, has the serosity of the pus the power of transforming blood into pus? I need not repeat the assertion of Hippocrates, Galen, Van Swieten, and De Haen, whose idea was that the pus, diffused throughout the organism, engenders pus at the expense of the humors. Looking to the great rapidity with which pus is diffused throughout the entire organism in cases in which there is no suppurative phlebitis, one is constrained, I think, to attribute a very large share to the absorption of the serosity when numerous metastatic abscesses are formed.

To sum up: I consider purulent infection to be the result of poisoning of the blood with pus.

Suppurative phlebitis generally furnishes the pus, which sometimes passes directly into the venous sinuses and plexuses, although they have not been the seat of any suppurative inflammation. Ulceration of the arterial walls, and particularly of the aorta or sigmoid valves, may be the source of purulent infection, as has been shown by Dr. Leudet of Rouen, in a memoir published in the Archives Générales de Médecine. But although purulent infection is most frequently the consequence of suppurative phlebitis, there are some epidemics in which we must, perhaps, seek for the cause, not in crowding, but in a special state of the atmosphere, in its containing, at a given time, altered pus-globules, which, being deposited on the wound, act in such a manner on the serosity of the pus of the wound as to modify it specifically, and give it the power to engender purulent infection of the system.

The serosity of the wound thus modified, may, in respect of its immediate and remote action, be compared to a virus.

My view is, that pus makes pus, and that putridity makes putridity, just as variola, syphilis, and glanders make variola, syphilis, and glanders.

But that pus should make pus, it is necessary that the pus be of a certain nature; and probably, it is in the serosity of the pus that we must search for the material specific differences; for according to M. Robin, it is in the serosity, and not in the globule of virulent pus, that the specific virus resides. From this point of view, all pus is looked upon as having a common element—the white globule; and also, a special element—the serosity. We are ignorant of the special principle of the serosity: what we fear is its absorption. Can we oppose a barrier, and dry up the source of infection?

I am now naturally led to speak of the *treatment* of purulent infection. But here, let me say, that my object in discussing so fully the doctrinal question of purulent infection in general has been for the purpose of throwing light upon *puerperal* purulent infection in particular. I leave you, therefore, to learn from systematic authors on external pathology the means to be used for the prevention of phlebitis, that too fertile source of purulent infection.

I believe that hospital hygienics embrace means by which the germs of infection may be neutralized, and its progress modified, should the germs become developed.

There is no specific cure for purulent infection. One ought not, however, to neglect the use of means, which, by being directed to the entire system, may determine salutary crises, and sustain the strength of the patient. Thus, sulphate of quinine may be successfully employed to check the periodic recurrence of the paroxysms of fever; but this medicine will not be any more useful than tincture of aconite in staying the progress of the infection, once pus has become mingled with the blood. The efforts of the physician ought to have another aim: his aim ought to be, to study the curative proceedings of nature in cases of spontaneous cure, endeavoring to imitate, or better still, to second her efforts to eliminate the morbific principle. In sketching the progress of purulent infection, I mentioned, that many patients had profuse viscid sweating and severe diarrhœa: I mentioned that the sweat and the diarrhœa present characters more easy to verify than to describe, but that their dominant characteristic is a special odor. These cutaneous and intestinal evacuations may be considered critical; and they ought to be promoted by sudorifics and purgatives, in the hope of obtaining results equally favorable with those obtained by Sanson and Vidal. It is useful to employ, at the same time, slightly stimulating stomachic beverages, and to place the patients under the best hygenical conditions.

To bring these remarks to a close, I shall now state my conclusions.

Purulent infection never occurs unless there be a wound. A wound is the essential obligatory condition. Every wound may have suppurative phlebitis as a sequel. Suppurative phlebitis pours pus *en nature* into the circulation. Perhaps this takes place continuously, although the intermissions in the rigors seem to indicate that the poisoning only takes place in an intermittent fashion.

Purulent infection may also originate in abscesses of the heart and aorta. This form of infection is rare.

Capillary phlebitis may induce infection by leading to the formation of pus: but in epidemics of purulent infection, the serosity of wounds, specially modified by atmospheric conditions, may be absorbed by the capillaries, without there being any oscula or erosions of the vessels; and infection is the consequence of this absorption. *The serum of the pus acts in a manner similar to the virulent inoculable serosities.*

Two principal indications ought to be fulfilled with a view to prevent or arrest infection. The first consists in so acting on the wounds as to prevent the occurrence of suppurative phlebitis, or interpose an obstacle to the absorption of infectant serosity. The second indication, based on the progress of the infection and the study of the crises, consists in the use of such means as promote and maintain the curative crises.

In conclusion, it is the duty of the physician to place his patients under the most favorable hygienical conditions which can be obtained, and thus enable them to struggle sufficiently long to finally triumph over the infection.

LECTURE XCV.

PHLEGMASIA ALBA DOLENS.

Phlegmasia in Recently Delivered Women.—Phlegmasia in Cachectic, Tuberculous, and Cancerous Subjects.—Semeiotic Value of Phlegmasia in Cachectic Diseases.—Phlegmasia in Chlorosis.—Phlegmasia in Recently Delivered Women: 1st, by Spontaneous Coagulation: 2d, Consecutive upon Uterine Phlebitis.—Symptoms of Phlegmasia: Pain, Œdema.—Venous Cords.—Collateral Circulation.—Temperature of the Affected Limbs.—Absence of Lymphangitis and Adenitis.

GENTLEMEN: Those of you who attend my clinical service must have remarked the frequency of the occurrence of phlegmasia alba dolens, an affection quite special, and well deserving attention from the numerous circumstances under which it is observed. You recollect that we have studied painful white œdema, not only in recently delivered women, but also, and more frequently, in persons of both sexes affected with pulmonary phthisis or internal cancer. To-day, I propose to speak to you of this affection which always has, as its primary cause, a special alteration of the blood, an alteration which exists in the puerperal state, and in many cachexiæ. I shall not attempt to establish on a statistical basis, the relative frequency of painful œdema in the cachexial and puerperal state; I merely wish to remark that this affection is frequently observed, and that, independent of the puerperal state, it may become a valuable element of diagnosis.

It is one of those diseases which demand minute study, because doubts still exist as to the nature of the affection, notwithstanding the frequent opportunities which occur of observing it. Painful œdema is a disease the nature and etiology of which have been differently explained. I must, consequently, state, in detail, the cases which will serve as the basis of the general descriptive sketch which I am about to place before you. I must also carefully recall to your recollection the anatomical details, because these details will have great value when we proceed to fix the precise seat of the affection. When the seat of the disease is well understood, you will be better able to understand, with the aid of anatomy, the symptomatology of the disease, and the complications, sometimes very serious, to which it gives rise.

A woman, 33 years of age, was admitted to St. Bernard Ward, presenting all the signs and symptoms of the third stage of pulmonary phthisis—amphoric blowing, gurgling, pectoriloquy, purulent expectoration, great emaciation, profuse sweating, Hippocratic fingers, hectic fever, functional derangement of the stomach and bowels, ulcerative laryngitis, and dysphagia. The patient had been six weeks in my wards when I ascertained she had white œdema of both superior extremities, involving the whole of the left arm, but limited in the right arm to the region of the elbow. On the first day, I searched, but in vain, for an obliteration of the superficial veins of the arm: I then thought that the deep veins were the seat of a spontaneously formed coagulum. The patient had not experienced any pain in the regions affected with œdema; and to cause suffering, it was necessary to compress the limb, either in its entire circumference, or over the course of the deep venous branches. Next day, the superficial veins were seen, blue and turgid, through the skin: they then seemed to be very probably the seat of a collateral circulation, rendering it still more probable that there was obliteration of the deep veins. During the first few days, I did not observe a fact which I afterwards noticed, and which must be added to the above description, viz., that on the inner surface of the arm, along each side of the humeral artery, a hard cord could be felt.

Soon afterwards, on each forearm, the superficial radial veins were felt to be obliterated, the skin was observed to be red in the course of the veins, and there was slight pain on pressure in that situation. The œdema of the forearm slowly decreased, till it quite disappeared, while, at the same time, the bulk of the superficial veins diminished, they recovered their elasticity, and again became permeable, as was clearly proved by the effects of compression made above and below the affected parts: when pressure was made near the elbow, the vein became dilated, and when made in the inferior third of the forearm, the vessel became quite empty. From the 31st January to the 14th February, 1862, I had the advantage of being a clinical witness of the formation and disappearance of the clots by which the superficial veins of the forearm were obliterated. Probably, a similar process took place in the deep veins of the same regions, in proportion to the progressive diminution of the œdema and pain.

From the 12th February, both legs became the seat of œdema, extending upwards from the feet, and soon invading the thighs. On the first day this œdema was especially marked in the left limb, where pressure with the finger left its print and caused pain. In the upper part of the leg, one could feel a hard superficial vein, which became lost in the popliteal cavity. The internal saphena vein was distended with fluid blood; and on exploring the femoral vein in its superior third, that vessel was found to be knotty, hard, and painful.

The right leg and thigh were œdematous, but in a less degree: the superficial circulation was manifested by the blue color of the veins, which seemed more than normally numerous: a hard painful cord, the obliterated femoral vein, was felt in the triangle of Scarpa.

On the following days, the œdema of both legs increased, but always continued greatest in the left. In that limb, where the œdema was greatest, there was soon perceived complete obliteration of the internal saphena vein up to its junction with the femoral vein, while only the lower third of the right saphena was hard and knotty. By compression at the lower third of the thigh, it could be positively ascertained, that the circulation was still possible in that situation. There likewise appeared some days later, in the legs, thighs, and round the knees, numerous venous capillaries, forming isolated groups, and apparently developed on the surface of the dermis. Some of these groups were red, others were white, and others again, were the seat of obliterated capillaries, quite appreciable by the finger: and when these vascular groups were subjected to pretty strong pressure, pain was occasioned.

Do you not observe in this last statement, distinct evidence of a well-marked tendency to the re-establishment of the circulation. The deep veins were seized in the first instance, the saphena veins supplied their place till they themselves became involved in the obliterative process: the capillary vessels then became very visible from congestion, and were invaded by obliterative clots, while the œdema went on constantly augmenting. The pain, limited in the first instance to the course of the principal vessels, soon became general, both on the surface and in the deeper parts: the skin in the situation of the capillary groups became very painful. In some places, where no trace of vessels could be discovered, light quick rubbing with the pulp of the finger occasioned pain.

Eleven days after the commencement of these symptoms, the œdema of the lower extremities had greatly increased: this was particularly the case with the left limb, where the saphena vein, and the posterior superficial vein of the leg, were decidedly hard: there was a little redness in the calf, where the acute character of the pain led one to suppose that there was inflammation of the vein in that situation. All the superficial veins of the right leg were greatly distended: on palpation no clots could be recognized: for several days the right limb remained in the same state, while in the left leg the œdema increased, and the superficial veins became more and more painful in the popliteal space and in the whole course of the internal saphena vein: the skin in these situations presented an erysipelatous redness.

On the eighteenth day, the left leg, and particularly the foot, had a notable lividity, where the slightest rubbing was exceedingly painful. The superficial abdominal veins were very much injected. There was no pain in the groin, nor in the left iliac fossa. On several places of the left leg and thigh, there was slight marbling of the skin and true ecchymosis; and in these situations, pressure caused pain. Similar phenomena were soon observed in the right limb; and the skin was figured with numerous capillary veins.

The œdema continued in the same degree. There was acute pain in the region of the liver. On the twenty-second day, the patient was seized with diarrhœa; and died without presenting any notable disturbance of the brain, lungs, or heart.

The autopsy was very interesting. It afforded opportunity of studying the obliterative clots in the situations where they still existed, although the œdema had continued till death; it likewise afforded an opportunity of studying the successive modifications which some of these clots had undergone; and also, the state of the parietes of the vessel where the circulation was re-established.

In the left limb, the veins of the calf, the femoral, the internal saphena, and the external iliac, were obliterated by fibrinous clots, the highest of which was situated at the opening of the hypogastric into the common iliac vein. There was found at that point a clot, rounded in form at its free surface, and bestriding, so to speak, a spur formed by the hypogastric and external iliac veins, with which its lower portions were continuous. The terminal clot was not adherent to the walls of the vessel: its form was cylindrical: its upper portion was rounded and devoid of any rent. It was composed of concentric layers, softened in the centre, so that portions were liable to give rise to pulmonary embolism by being drawn into the torrent of the circulation flowing from the hypogastric vein.

In the right limb, the clot, the ramifications of which occupied the deep femoral veins, the popliteal and tibial veins, stopped short where the internal saphena opens into the femoral: at that point, the clot was fibrous, similar in appearance, form, and structure, to that of the common iliac vein of the left limb. The clot contained in the saphena vein was cruoric, of recent origin, and non-adherent to the parietes of the lower veins.

This autopsy shows clearly that the continuance and extent of the œdema of the lower extremities was explained by the continuance, extent, and structure of the clots.

This is not all: the examination of the veins of the superior extremities ought, still farther, to demonstrate the correspondence of the anatomical appearances after death with the symptoms during life. During life, there was a partial phlegmasia of the superior extremities, the commencement, progress, and termination of which coincided with the appearance, duration, and disappearance of the coagula in the superficial veins. At the time of the appearance of the œdema, there was induration of the brachial vein in both arms, and of the superficial radial vein in both forearms: in the course of these vessels, a red line, pain, and swelling were observed; and under the finger, there rolled a cord, the limits of which could be easily determined: at the same time, there was partial œdema: little by little, the œdema disappeared, and proportionately to its disappearance the pain, swelling, and hardness of the vein went away. It was ascertained, by anatomical examination, that there was no trace of inflammation remaining in, or around, the veins: the veins contained no obliterative clots, and were free; their walls had regained their normal elasticity. It was only in the left brachial vein that there was a fusiform cruoric clot, fringed at both extremities, adherent only at some points of its surface, and sufficiently small to allow the circulation to go on round it. It is probable, then, that the clots by which the veins had been formerly obliterated, had been absorbed *in situ.*

The case is a rare example of general intra-venous coagulation in the four extremities. Under what conditions does the blood present this tendency to spontaneous coagulation? Gentlemen, you are aware, that in the cachexiæ generally, and in the tuberculous and cancerous cachexiæ particularly, the blood has undergone important changes. The beautiful works on hæmatology of MM. Andral and Gavarret* and MM. Becquerel and Rodier leave no room for doubt on that point: the modifications consist chiefly in a change of the proportions of the constituents of the blood: thus, in every cachectic affection, there is a diminution of the red globules, and an augmentation of the fibrin and serum. Now, the blood having a great tendency to spontaneous coagulation, we are entitled to ask, whether this coagulation be not due to an excess of fibrin, or to the fibrinogenous element?

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* Andral et Gavarret: Recherches sur les Modifications de Proportion de quelques principes du Sang dans les Maladies. Paris, 1842.

One word, gentlemen, on this element. You know that in serous effusions into the pleural cavities, there exist two isomeric substances, fibrin and albumen. When serous fluid which has been effused into the pleuræ is exposed to the air, coagulation of the albumen does not take place at a temperature under 70° or 75° Centigrade, while there is spontaneous coagulation of the fibrin some degrees below zero. Moreover, when we extract the spontaneously coagulable fibrin, either by heating or straining through a linen cloth, and leave the remaining serosity exposed to the air, we are surprised to see, that, some hours after the first straining, a new fibrinous coagulation has formed in the fluid. It is evident, therefore, either that all the fibrin has not been removed in the first experiment, or that the fluid contains a special substance which may at a given time present all the characters of fibrin. To a substance of that character, Virchow has given the name of "fibrinogenous substance," which might be more properly designated *inogène*, that is to say, a substance capable of giving birth to a new quantity of fibrin. Perhaps this substance exists in serum of the blood of cachectic persons; and if so, its coagulating properties explain the tendency to spontaneous coagulation in the blood of cachectic subjects.

The fact, long well known, that the blood of cachectic persons has a strong tendency to coagulate, explains the frequency of coagula in the vessels of tuberculous patients who have reached the cachectic stage of their disease. I wish also to recall to your recollection some of the facts which prove that the same spontaneous coagulation is common in cancerous patients, and gives rise to *phlegmasia alba dolens.* In St. Bernard Ward, you have had frequent opportunities of observing this phenomenon in women affected with cancer of the uterus. Patients of this description, in the last stage of their disease, present the symptoms of cachexia, and then, all at once, the inferior extremities become swollen, soon after which the saphena and crural veins can be felt to be hardened: in such cases, it is found, on examination after death, that their hard condition is owing to cruoric or intra-vascular fibrinous clots.

I have long been struck with the frequency with which cancerous patients are affected with painful œdema in the superior or inferior extremities, *whether one or other was the seat of cancer.* This frequent concurrence of phlegmasia alba dolens with an appreciable cancerous tumor, led me to the inquiry whether a relationship of cause and effect did not exist between the two, and whether the phlegmasia was not the consequence of the cancerous cachexia. I have since that period had an opportunity of observing other cases of painful œdema, in which, at the autopsy, I found visceral cancer, but in which, during life, there was no apprciable cancerous tumor; and in which there existed a cachexia referable neither to the tubercular diathesis, the puerperal state, nor chlorosis. I have thus been led to the conclusion, that when there is a cachectic state not attributable to the tuberculous diathesis, nor to the puerperal state, there is most probably a cancerous tumor in some organ.

Many of you remember the case of a patient in the wards of Legroux, my late lamented colleague. The man to whom I refer was fifty-nine years of age. Without any known cause, he was seized with phlegmasia alba dolens of the left leg. The case presented all the characters of phlebitis: there were acute deepseated pains in the calf, beginning about the lower third of the leg, and extending up to the hamstring. The patient was extremely pale, and had a general cachectic hue. Legroux was inclined to think that the patient had leucocythæmia. When consulted, I said: "Perhaps this patient has leucocythæmia; but he has phlegmasia alba dolens, and consequently deepseated, concealed cancer."

I sought for this cancer with the greatest care: and during the six weeks which the patient remained in the wards of Legroux, that physician searched for it with his habitual scrupulous attention, and yet was unable to detect the signs of a cancerous affection. At the autopsy, there was found an annular cancer of the pylorus, which, as it allowed the passage of the food into the duodenum, had not given rise to vomiting of any special character.

In other cases, in which the absence of any appreciable tumor made me hesitate as to the nature of a disease of the stomach, my doubts were removed, and I knew the disease to be cancerous, when phlegmasia alba dolens appeared in one of the limbs.

Some years ago, one of the professors of the Faculty of Medicine had symptoms of simple ulcer of the stomach. Several physicians had been consulted; and as they found no tumor in the region of the stomach, they were disposed to regard the vomiting as symptomatic of simple ulcer. Soon after this, I learned that the professor had phlegmasia, whereupon I unhesitatingly declared that he would sink under advancing cancerous disease: the rapid progress and fatal issue of the case proved my diagnosis to be correct.

In 1860 a man, about forty years of age, consulted me, as a private patient, regarding pain and a feeling of weight in his left leg. On questioning him, I learned, that at a previous period, he had experienced similar pain in the right leg; and that he had, subsequently, been operated on by M. Maisonneuve for a tumor of the testicle. To me these facts were of the greatest importance: from them I concluded that the patient had had previously phlegmasia, symptomatic of a cancerous tumor of the testicle, and that at the time when I was consulted, the œdema of the left leg was caused by a deepseated cancerous affection: in point of fact, by palpation, I detected within the abdomen, tumors, of the cancerous nature of which I had not the least doubt.

We must not suppose that painful œdema of the inferior extremities in cases of cancer of the testicle, uterus, or rectum, results from the inflammation of the veins of the primarily diseased parts being propagated to the deepseated veins; nor are we to believe that the œdema is the mechanical consequence of pressure exerted on the abdominal veins by tumors or diseased glands. Such opinions are untenable, for whoever carefully analyzes cases will find, that cancerous tumors of the stomach or breast give rise to this kind of phlegmasia. I might cite many cases in confirmation of this statement, but will be satisfied by referring to the case of a man aged forty-six, mentioned by Virchow, who had carcinoma of the stomach coexisting with double phlegmasia of the lower extremities, and painful œdema of the left arm.

So great, in my opinion, is the semeiotic value of phlegmasia in the cancerous cachexia, that I regard this phlegmasia as a sign of the cancerous diathesis as certain as sanguinolent effusion into the serous cavities.*

In the cachexiæ, as I have told you, there exists a special crasis of the blood, which, irrespective of inflammation, favors intra-venous coagulation. This state of the blood is likewise met with in chlorosis properly so called, and in the puerperal state.

Painful œdema, as an epiphenomenon of chlorosis, is, however, a rare affection. I must describe a case which occurred in my wards, and was reported in 1860 by Dr. J. Werner in his inaugural thesis. That young physician particularly calls attention to phlegmasia as an epiphenomenon of cancerous cachexia.

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* See Lecture XXXII, on Paracentesis of the Chest, vol. i, p. 609.

A young woman, aged twenty-five, was admitted to my wards with all the signs of chlorosis—extreme pallor, blowing sound in the vessels of the neck, palpitation of the heart, intercostal neuralgia, dyspepsia, and amenorrhœa. She was forthwith treated, a little actively, when, all at once, she experienced pain in the left inguinal region; and on the same day, there was detected phlegmasia of the right inferior extremity, characterized by œdema of the limb and intra-venous coagulation. After continuing for three weeks, the œdema disappeared.

Painful œdema was formerly looked upon as an affection peculiar to lying-in women: hence, it received many names referring to the state of the recently delivered woman. Mauriceau, Puzos, Callisen, and White have devoted special chapters to the swelled leg of lying-in women, to lacteal engorgement of the inferior extremities, and to the *phlegmasia alba dolens puerperarum;* but Robert Lee and White were the first to describe the lesions of the veins which accompany painful œdema. Subsequently, MM. Bouillaud and Velpeau, in works published in 1823 and 1824, had the merit of showing the part which obliteration of the veins has in producing partial dropsies. Later researches confirmatory of the statements of these savants, have demonstrated that obliteration of veins may occur spontaneously, or, in other words, may depend upon a crasis of the blood, and not upon inflammation of a vein.

The remarks which I have already made upon the frequent occurrence of phlegmasia in phthisical and cancerous patients, render it unnecessary for me now to prove to you that this kind of œdema is not peculiar to lying-in women; and that there exists in the cachexiæ, as well as in the puerperal state, a particular condition of the blood which predisposes it to spontaneous coagulation, that is to say, a condition in which there is an excess of fibrin, a diminution of blood-globules, an increase of water, and an increase of white globules.

I have told you, that the chlorosis of pregnant women is very seldom complicated with phlegmasia; and that the œdema of pregnant women is not due to the coagulation of the blood, but results from a double condition, the aqueous state of the blood, and the embarrassment of the circulation in the inferior extremities, mechanically produced by the augmented volume of the uterus. When anasarca occurs in a pregnant woman, we have to fear another complication—albuminuria.

Though painful œdema is never observed in the pregnant, it is often seen in the recently delivered woman; but its determining cause cannot always be discovered. We may meet with this œdema in cases in which neither the duration of labor, nor the presentation have been abnormal; and in which there has been no need of any important obstetrical manipulations. On the other hand, in cases in which partial œdema results from phlebitis, we can often trace back the affection to the local cause in which it had its origin. Uterine phlebitis may be propagated from the uterine veins to one of the hypogastric veins, and from the latter to the common or the external iliac vein. At autopsies, I have frequently recognized this causation, have seen obliterative phlebitis, and have traced the obliterative clot into the iliac veins, and sometimes even into the inferior vena cava. So long ago as 1826, Velpeau published cases demonstrating the propagation of the inflammatory process from the uterine to the iliac veins.

In these cases, there is sometimes real inflammation, which becomes the cause of the œdema. We then discover that the uterine sinuses and veins contain pus, and have thickened walls: but besides the pus, we find that these vessels contain clots which present an obstacle to the production of purulent poisoning. These fibrinous clots become covered with new layers of fibrin, which, like ceaselessly formed strata furnished by the fibrin of the circulation, become so added to, and superimposed upon one another, that the clots are lengthened out to the junction of the hypogastric with the iliac vein. At the junction, the clot projects, and is, at that point, covered with a new stratum of fibrin, which augments little by little, till the iliac vein itself is soon partially or wholly obliterated. Adhesions form between the walls of the vessel and the newly formed clot: this is the time at which œdema of the entire right or left inferior extremity shows itself. It almost never happens that both limbs are simultaneously affected, that is to say, they do not both become œdematous on the same day, but first the one and then the other. This sequence of phenomena is fully explained by clinical study and necroscopic examination: either inflammation of the uterine sinuses is propagated to both iliac veins at an interval of some days occurring between the date at which each limb is attacked; or, the clot formed in the common iliac vein of one side is prolonged into the vena cava inferior, and from it, into the common iliac vein of the opposite side. The greater frequency of simple phlegmasia on the left side, and of double phlegmasia beginning on the left side, has been attributed by anatomists to the mutual relations of the arteries and veins at the sacro-vertebral angle. You are aware that in that region, the arterial system is situated on a plane anterior to the venous system, so that both common iliac arteries pass in front of the veins of similar name, dividing them at right angles; again, before reaching the inferior vena cava, the left common iliac vein is divided, almost transversely, by the right iliac artery: from these relations, it results, that, on the dead body, the arteries leave their print on the subjacent veins; and it is not unusual, when these veins are filled with clots, to find these clots strongly depressed at the spot at which the veins are crossed by the arteries. This compression is specially manifest in the left iliac vein, and anatomists ascribe to the more frequent compression of the left iliac vein, the more frequent occurrence of phlegmasia on the left side. On the other hand, accoucheurs have supposed that, as left anterior occipito-iliac presentation is the most common, the greater frequency of phlegmasia in the left limb is perhaps owing to the pressure of the head during labor on the left iliac vessels. I do not reject this reasoning; but I only accept the anatomical conditions as a determining cause. The same conditions exist in nearly every recently delivered woman, or in all cachectic persons; and yet phlegmasia is not a necessary complication in either class of patients. Consequently, there exists a special cause, which is unknown.

Painful œdema generally shows itself in a sudden manner: without appreciable cause, the patients complain of pain in a limb, which is at the same time observed to be œdematous. The pain is not always of the same kind: it is sometimes a feeling of weight and distressing numbness of the entire affected limb: at other times, it is a constant pain, intensified at a particular place, which, in the inferior extremity, is generally the calf, the groin, the lower part of the thigh, or the popliteal space: in the superior extremity, it is the axilla. If pressure with the finger be made over the seat of pain, or if the muscular masses in which pain is felt be grasped with the hand, an increase of pain is produced, which is often so great as to cause the patient to cry out. Sometimes, the sensibility of the skin of the whole limb is obtunded: while, at other times, touching or rubbing it lightly will occasion great suffering. I have often observed this cutaneous hyperæsthesia, which, strange to say, is less under strong than under slight pressure. Sometimes, the pain and numbness are accompanied by inability to perform the least voluntary movement: for example, the patients can neither extend nor flex the toes, nor move the leg or thigh. Though in some cases there are articular pains to account for this immobility of the limbs, in others, in which pressure causes no articular pains, all movement is as impossible as if muscular paralysis existed.

An attempt has been made to give a special description of the œdema: it has been alleged that it shows itself first at the upper part of the limb, and then appears secondarily in the remoter parts. Gentlemen, I confess that I have never observed the affection to have that beginning or course: on the contrary, I have always seen partial œdema begin in the most remote and depending parts, subsequently gaining the upper parts of the limb. To be convinced of the truth of this remark, it is only necessary to observe with care the progress of the œdema in cases of double phlegmasia. Directing our attention to the possibility of the occurrence of double œdema, and continuing to observe daily the left limb still healthy, we will soon observe the œdema begin at the malleoli and instep: the skin will be seen to have acquired a dull color, and the subcutaneous cellular tissue will be seen to retain the print of the finger: then by slow degrees, the œdema will ascend, and soon the intra-cellular effusion will give a rounded form to the whole limb. The limb generally acquires the form of an elongated cone, the base of which is the root of the limb. This description applies to œdema consequent upon obliteration of the principal vein of the affected limb.

I have also seen cases of partial œdema resulting from the obliteration of secondary veins, a class of cases in which the œdema may be limited to the region served by the obliterated vein.

As soon as the œdema is appreciable at the malleoli, and before it has gained the root of the limb, there may sometimes be detected, by carrying the finger over the course of the crural vessels, a hard, resisting cord, which can be traced to the ring of the adductor muscles; at the same time, pressure occasions deepseated pain in the course of the vessels, in the popliteal space, and often in the muscular mass constituting the calf of the leg. Sometimes the crural portion of the internal saphena vein communicates to the exploring finger the sensation of a knotty cord.

The current of venous blood is then nearly quite obstructed, and upon the dull colored surface of the entire limb, bluish arborescence becomes visible, the indication of an existing tendency to form a collateral circulation. The arborizations sometimes become hard little cords; and then the circulation is soon obstructed by clots of recent formation. There is observed at a later period, at several points, little red or bluish isolated patches of capillaries, which do not escape the process of coagulation taking place throughout the entire limb.

It is a fact deserving of notice that the temperature of the affected limb does not seem reduced, the hand applied to the surface of the affected limb not detecting any appreciable modification of temperature. The skin continues to present a dull white appearance throughout the entire surface of the limb; it is only at the last, when the affection is near its inevitably fatal issue, that we see the toes and then the instep take on a diffuse bluish color, the temperature of these parts being then decidedly reduced.

Absence of any modification of temperature, while it excludes the idea of gangrene, also excludes the idea of phlegmasia of the cellular tissue. It is equally unusual to observe any red lines similar to those observed in cases of lymphangitis: the glands are only in exceptional cases the seat of abnormal tumefaction, and in some cases it is only at the autopsy that we can discover that the deep-lying glands which accompany the vessels are a little enlarged; and on section we find that they present a slight color. Never since my attention was arrested by this subject, have I observed inflammation of the glands, or peri-glandular cellular tissue.

These facts, then, justify me in rejecting both the existence of lymphangitis and of adenitis in the phlegmasia; while the general rule is a profound modification of the venous system, a modification clinically disclosed by the venous cords, regarding the nature of which pathological anatomy does not leave us in any doubt.

I have said that the phlegmasia is almost never double from the first onset; when both limbs have been seized simultaneously, the obstacle to the venous circulation is seen first in only one side, and that side is generally the left. Sometimes, however, the venous coagulation may be simultaneously observed in the four extremities, but each occurs in succession. The first case I described to you is one of those rare cases in which the study of the phlegmasia may be made at one and the same time in all the four extremities.

It likewise sometimes happens that the phlegmasia does not make its appearance on one side till it has entirely disappeared from that which was first attacked.

The average duration of an attack of phlegmasia is about three weeks; that is to say, in a phlegmasia supervening in a recently delivered woman, or in a case of cachexia, at the end of three weeks the œdema has almost entirely disappeared, the pain has ceased, and the patients have regained the use of the limb. It is necessary, however, to point out the differences in respect of the duration and termination of attacks of phlegmasia which arise according to the different conditions which exist in the individual patient. The œdema hardly ever commences till the tenth day after delivery. It has been sometimes known not to supervene till the end of the third or fourth week; but in these cases, the determining cause is often undue exercise of the limbs, or some other cause capable of keeping up the uterine fluxion, or some pathological state which often exists in the veins of the uterus. Under normal circumstances, about the third week, when there are no peri-uterine complications, there is little ground for fearing phlegmasia, as by that time the general state of the woman is favorably modified; and particularly if she do not suckle her infant, the *inopexia* has a great tendency to disappear.

The issue of the malady is favorable when there is no phlebitis, properly so called. The œdema then diminishes little by little; the tissues regain their elasticity; the collateral circulation becomes less appreciable; the capillary vessels are less apparent; pain is no longer felt in the calf of the leg and in the course of the vessels. It is also found that the vessels, superficial and deep, are less tense; they no longer roll under the finger, and after a longer or shorter period, they regain their normal elasticity, and are again traversed by blood. Sometimes, however, the internal saphena still remains obliterated in a greater or less extent of its course; some nodosities can still be felt in the course of the femoral vein; and it is not till long after the commencement of the malady that all trace of vascular lesion disappears. At other times the affected limbs remain œdematous although the malady be no longer in the œdematous stage; the remaining œdema is then due to obliteration of the veins originally the seat of the process of coagulation. This persistence of œdema may continue for several years; every accoucheur has met with cases of this description. In these cases, the circulation is imperfectly re-established in collateral channels, and the cellular tissue is thickened rather than œdematous, as is shown by its having regained its elasticity to a great extent, and by its not retaining the print of the finger when pressed. The least fatigue, however, occasions pain, and induces embarrassment in the circulation of the affected limb.

Pulmonary Embolism.—Van Swieten and Virchow.—Symptoms of Pulmonary Embolism; Extreme Dyspnœa; Apnœa; Thirst for Air; Sudden Death.—Death takes place from Syncope or Asphyxia.—Œdema of the Lungs, Pneumonia, Gangrene of the Lungs, Hydro-Pneumothorax.—Embolism, pulmonary or cardiac, originating in Uterine or Peripheral Phlebitis.

The phlegmasia may be the cause of a most formidable accident—the breaking up in fragments of the obliterating clots. In some cases, the clot is carried on by the current of blood to the heart, and thence to the pulmonary artery. This grave occurrence has been particularly observed in the phlegmasia which follows delivery. The works of Virchow, the memoir of Ball and Charcot,* and the researches of Lancereaux, have directed attention to it. Venous embolism has, however, been studied in other morbid states: though rare in cachectic cases, it has been observed as a sequel of phlebitis, as is established by the writings of Velpeau, Briquet, and Azam of Bordeaux.

When speaking to you of softening of the brain, I referred to arterial embolism depending upon structural change of the heart, or serious lesions of the pulmonary veins. I propose to-day to treat of venous embolism. Its very great frequency in the phlegmasia of lying-in women justifies my going fully into this subject.

Cases of lying-in women dying suddenly two or three weeks after delivery, are known to every physician; and although the dyspnœal symptoms which immediately precede real death are very different from those observed in syncope, there has been a disposition to attribute death to sudden and persistent stoppage of the heart. Those who have read Cullen's admirable chapter on syncope, and recollect that he recognizes pulmonary syncope, must have been led to the conclusion, that sudden death preceded by extreme dyspnœa has its seat in the lungs, and must have been thus led to discover clots in the pulmonary artery, which explained to them the cause of death.

Van Swieten has recorded the results of experiments which he performed on dogs: he showed that blood coagulated by acids in the peripheral veins can be transported in the torrent of the circulation in the form of plugs to the pulmonary artery, there to determine certain phenomena which prove suddenly fatal. He says: "Tentavi similia experimenta in canibus sæpius, vidique semper sanguinem inde grumescere, et per venas semper latiores in suo decursu, ad cor dextrum deferri: dein in pulmones, ibi autem hærebat: et post *summas anxietates*, animalia hæc moriebantur, citius vel serius, prout major minorve talium coagulantium quantitas venis injecta et diversa foret horum efficacia. Poterit ergo talibus causis subito peripneumonia induci."†

Dr. Ball rightly remarks, that no better description could be given of that which we now call venous embolism than this description by Van Swieten.

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* BALL et CHARCOT: Sur la Mort Subite et la Mort Rapide à la suite de l'Obturation de l'Artère Pulmonaire par des Caillots Sanguins, dans les cas de Phlegmatia Alba Dolens et de Phlébite Oblitérante en général. [*Gazette Hebdomadaire de Médecine et de Chirurgie*, 1858.]

† VAN SWIETEN: T. ii, p. 654, Aph. 824. Paris edition, 1771.

Observe, gentlemen, that Van Swieten said that death was preceded by very great anxiety—*post summas anxietates*—and if you attentively peruse the works published on this subject during the last few years, you will see that in nearly every case it was noted, that the patients were seized very suddenly with dyspnœa and orthopnœa, and were a prey to the most terrible anxiety; whereas, death by syncope, as you know, is not generally preceded by embarrassed breathing. The patients feel their strength failing almost before they have time to call for help, and die without any apparent struggle. But in cases of embolism, the death-agony testifies to the existence of impeded respiration, the patients thirst for air—*ont soif d'air*—the anxiety which they experience being similar to that observed in cases of rapid asphyxia from any cause, whether from an aneurism bursting into the bronchi, or a stunning stroke of apoplexy.

The facts adduced by Van Swieten had been forgotten; and to Virchow belongs the honor of having established by numerous works—the first of which appeared in 1847—that clots formed in the peripheral venous system may be carried into the circulation and occasion speedy death by their arrest in the pulmonary artery.

Therefore, when you see a recently delivered woman suddenly seized with symptoms indicating great disturbance of the function of hæmatosis—symptoms the chief of which are pain in the chest, and great respiratory anxiety, you may conclude that possibly there is pulmonary embolism, and look for signs of coagulation in the peripheral veins. In cases of phlegmasia, the search will not be very difficult; a thrombus of the uterine veins is sufficient to produce an embolism. I recollect a young woman affected with periuterine phlegmon, in whom digital exploration *per vaginam* (performed perhaps somewhat brusquely) gave rise to all the symptoms of pulmonary embolism.

Sudden death is not always the consequence of pulmonary embolism: death will not take place rapidly unless the seat of the embolism be one of the principal branches of the pulmonary artery. In the cases in which the migratory clot has been able to reach subdivisions of the second or third order, the sudden dyspnœa which is induced may be succeeded by peripneumonia and pulmonary œdema, from which the patients may recover. Gangrene may result from pulmonary embolism; and should the gangrene involve one of the peripheral lobules, there is reason to dread a mortal issue, from perforation and consecutive hydropneumothorax. The first case of pulmonary embolism which I saw was of this description.

Gentlemen, I am aware that experimentalists have written that large foreign bodies may be introduced into the pulmonary artery of dogs and horses without producing the slightest dyspnœa. I cannot deny the accuracy of this statement, but I may be allowed to say that I think it requires to be tested by new experiments.

I am also aware that it is difficult to understand how obliteration of one of the branches of the pulmonary artery—which is not an artery of nutrition—should determine gangrene of the lung; but if it be borne in mind that, in the experiments of Virchow, pneumonia was a frequent consequence of pulmonary embolism, it may be granted that, as the embolism can produce inflammation, it may likewise produce gangrene.

The embolus, according to its size, according to the point in the pulmonary artery at which it has been arrested, and according to the individual peculiarities of the patients, will produce certain symptoms, varying in gravity, the mildest being transient dyspnœa and the worst being sudden death. Let me now state how I explain the dyspnœa and sudden death by the migration of a clot to the pulmonary artery. The patients die from a particular variety of asphyxia, to which attention has not been sufficiently directed.

If you forget the etymology of the word *asphyxia*, and only bear in mind the state of which it is the name, you will not see with physiologists, either a complete or an incomplete asphyxia, merely a diminution or suppression of pulmonary hæmatosis. Two things are indispensable for the performance of this function—respirable air and blood—that is to say, the oxygenating fluid, and the fluid which requires to be oxygenated. If air be wanting, there is asphyxia, the degree being in accordance with the extent to which the air is deficient: and if a greater or less quantity of blood do not reach the pulmonary vesicles, to be there vivified by the air, a state of asphyxia will supervene from the obstacle to the arrival of blood. Wherever the obliterated clot may be situated, the sudden suppression of function in part of the lung causes anxiety and dyspnœa. Should the obstacle be sufficiently great to prevent at once, or in a few minutes, the arrival of blood in the lungs, rapid asphyxia, and death will occur. The obstacle is seldom sufficiently great to stop so rapidly the arrival of any blood. When the obstacle is situated in one of the principal divisions of the pulmonary artery, there exist on the distal side of the obstruction the conditions favorable to coagulation *a tergo*, so that the pulmonary circulation soon becomes compromised, and at last impossible. In these cases, the asphyxia may be compared, from the effects it produces, to that caused by closing the windpipe or the principal divisions of the bronchial tubes. If to that state be added the nervous disturbance and the shock felt by the whole system, an explanation is given of the great and rapid disorder caused by a migratory clot.

According to the view I have now set before you, the patients die by the lungs, and from a particular variety of asphyxia. According to other observers, death is the result of syncope. From this point of view, the circulation being obstructed in the pulmonary artery, the right side of the heart soon becomes filled with blood, and is, in consequence, unable to contract, while the left side ceases to act from no longer receiving hæmatosed blood, its required excitant. It is sufficient to remark that in syncope, properly so called, death begins at the heart, whereas in embolism death begins at the lungs. Moreover, the anxious dyspnœa, the craving desire for air, and the purple visage, demonstrate the existence of asphyxia.

When the clots are primarily formed within the heart, it is not unusual to see them prolonged into the pulmonary artery and its principal divisions: in these cases there is a certain slowness in the progress of the symptoms, and the sudden manifestation of symptoms which occurs at the beginning of the attack in embolism is not observed. On auscultation, it is found that the heart beats feebly, and that its sounds are irregular; and often there are morbid sounds.

In certain very unusual circumstances, when peripheral clots become arrested in the heart, and are sufficiently large to obstruct the passage of the blood into the lungs, dyspnœal symptoms may show themselves, similar to those occasioned by pulmonary embolism. In fact, the right side of the heart may be considered as the commencement of the pulmonary artery; but in addition to the dyspnœa and anxiety, physical signs, detected by auscultation, will be present from the commencment of the symptoms.

It would be a mistake to deny the presence of pulmonary embolism, simply because the symptoms of that affection had disappeared. Many cases are on record, in which the symptoms have ceased little by little. Dr. Jacquemier has published a case in which recovery took place. In cases of recovery, it is probable that the clot was small, and capable of being absorbed. When the patients die some days after the commencement of the symptoms, pathological anatomy shows that the transported pulmonary clots, as well as the indigenous clots, may undergo changes tending to softening or cellular organization. When there is softening of the clot, there is disintegration of the fibrin; and the circulation may become re-established without there being necessarily observed any of the lesions usually caused by capillary embolism. In cases in which the clots become organized, adhesions form between the walls of the vessel and the connective tissue of the clot; these cellular adhesions may themselves disappear after the lapse of some time.

Such, however, is not the usual course of pulmonary embolism; it usually carries off the patients in some hours or days after the commencement of the symptoms.

In the beginning of this lecture, I said that phlegmasia might be the consequence of phlebitis; but hitherto I have only been speaking to you about spontaneous phlegmasia. I must now, therefore, say something on the subject of phlegmasia consequent upon inflammation of a vein.

It is not necessary that I should give you a description of phlebitis: I am only desirous to show you how inflammation of a uterine vein, or of any pelvic vein, may become the mechanical determining cause of phlegmasia, properly so called.

If uterine phlebitis be suppurative, purulent infection is observed: when it is adhesive or obliterative, a barrier is set up against purulent infection, and the obliterative clots may extend even into the trunk of the hypogastric vein.

If you believe—and the fact has already been demonstrated by pathological anatomy—that the obliterative clots ascend to the junction of the hypogastric and common iliac veins, it will be seen that in consequence of the inopexia of cachectic subjects, or recently delivered women, a condition which favors a deposit of new fibrinous layers, the head of the hypogastric clot will be sufficiently large to protrude into the common iliac. You then have the mechanical condition of a phlegmasia on that side; for the clot, always increasing in bulk, will ultimately obliterate the circulation in the iliac vein; and that coagulation may extend from the external iliac to the femoral vein. It is a remarkable fact that frequently, as soon as there is an obstacle to the venous circulation, fibrin is precipitated, so to speak, into the valvular pouches, giving rise to the knots in the course of the veins, of which I have already spoken.

You can thus understand how an attack of coagulative phlebitis in the true pelvis may give rise to phlegmasia. Velpeau* has published cases which strongly support this interpretation of phlegmasia, as a consequence of phlebitis in a neighboring part.

Whenever you have reason to believe that there is non-suppurative phlebitis of the true pelvis, you may anticipate the occurrence of consecutive phlegmasia. On the other hand, pulmonary embolism may be observed as a sequel of phlebitis of the true pelvis, without there having been any phlegmasia as an intermediate pathological occurrence: for that to occur, it is sufficient that a fibrinous clot, originally attached to the uterine or hypogastric veins, should become detached. Consequently, when symptoms of pulmonary embolism have been observed after delivery, it is necessary, should no phlegmasia exist in one of the lower extremities, to search in the uterine and hypogastric veins for the point whence the migratory clot has started.

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* VELPEAU: Recherches et Observations sur la Phlegmatia Alba Dolens. [*Archives Générales de Médecine*, 1824, t. vi, p. 220.]

My opinion, then, is, gentlemen, that painful white œdema, or phlegmasia, cannot be mistaken for any other kind of œdema. I have just reperused the memoir of Dr. Bouillaud, and have pleasure in stating that the work of my colleague is as complete as if it had been written yesterday.* It contains eight cases in which phlegmasia was observed in cancerous and tuberculous cachexia, and as a sequel to delivery. In all these cases, obliteration of a vein was observed and described. Bouillaud mentions that there were clots in the veins, obliterative clots, obstructing the venous circulation, and so producing partial dropsies. Bouillaud's work is the basis of all the anatomical researches which have been made in France, since 1823, into the obstruction of veins.

The conditions under which phlegmasia shows itself, its commencement, its progress, and the vascular lesions by which it is accompanied, are all calculated to eliminate sources of error from the diagnosis. When the œdema shows itself, at a time when it is usually limited to the lower limbs, the patient's general state of health has already presented a group of symptoms suggesting that there may be coagulation within a vein. That general state is cachexia, whatever its cause may be. Forewarned of the possible occurrence of venous coagulation, apprised of the coagulation by a feeling of pain in some part of the limb, struck by the rapid development and limited extent of this œdema, the physician will proceed to discover whether, in the course of the superficial and deep veins, there be at any point a hard cord, or the knots which are produced by intra-venous congestion, and deposits of fibrin in the valvular pouches. At these different points, pressure occasions pain, and particularly in those situations where there exist natural obstacles to the venous circulation, as in the muscular masses, and in situations where there is a confluence of several veins in one chief trunk, such as the popliteal, inguinal, and axillary regions.

These remarks will enable you to avoid mistakes. In the cases in which the œdema is irrespective of any cachexia, and the consequence of inflamed varicose veins, it is not so considerable as in cases of spontaneous phlegmasia, and a varicose condition of the limb exists which can always be easily traced back to its cause.

I should not think it necessary to insist on this point were it not that varicose phlebitis may give rise to pulmonary embolism. In two cases of varicose inflammation, observed by M. Velpeau and by M. Briquet, the symptoms of pulmonary embolism were produced by a portion of the venous clot getting into the stream of blood.

To determine with precision whether there has been spontaneous coagulation, or coagulation resulting from inflammation, is sometimes difficult, and is never of clinical interest.

I have still to speak of those very limited coagulations which may become spontaneously developed in the continuity of a vein, and which, from their small size and slight adhesion to an uninflamed vein, may be apt to occasion pulmonary embolism. Perhaps, in such cases, the physician ought, following White's recommendation in respect of purulent infection, to try to interpose a barrier between the clot and the large veins.†

I believe that spontaneous coagulations may be developed in the saphena, crural, or any other vein, and remain limited to a very small extent of the vessel. In that case the œdema and pain only exist in those parts of the venous circulation which were served by the obliterated vein. As regards the pathological anatomy of the affection, it may be stated, that we sometimes see fibrinous clots deposited only where there are valves, while cruoric clots are ultimately deposited in the portions of vein situated between two valvular pouches. If the opinion which I am endeavoring to establish has any value, you can understand how important it will be for the physician to recognize the possibility of these partial coagulations existing; for if he cannot prevent their migration, he may be prepared for the occurrence of sudden death from pulmonary embolism.

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* Bouillaud: Oblitération de Veines et de l'Influence de cette Oblitération sur la formation des Hydropisies Partielles. [*Archives Générales de Médecine*, t. ii, p. 188.]

† White: Inquiry into the Nature and Cause of that Swelling in one or both of the Lower Extremities which sometimes happens to Lying-in Women. Warrington, 1784.

Here, gentlemen, I must make a modification in my statement in respect of the manner in which death takes place in cases of embolism. Generally, the migratory clot reaches the lung, causing dyspnœa and rapid death by asphyxia; but we can understand that in certain exceptional cases, the clot may be arrested in the right auricle or ventricle. Then, in accordance with the predisposition of the patient and the volume of the clot, the phenomena which belong to syncope will be observed; the heart, surprised, so to speak, by the arrival of the migratory clot, will at once cease to beat with regularity and power, and ere long contractions will entirely cease. In these cases, death will take place by syncope, by arrest of the heart: in fact, the prolonged syncope leads to death. Thus, a particular patient who has had repeated fainting fits, may be carried off by syncope, of which the determining cause has been embolism.

A cardiac embolus will then have produced syncope, just as a pulmonary embolus induces dyspnœa and asphyxia.

I look on these latter reflections as purely speculative: I have not observed a single case calculated to convince you of their truth; but a case, communicated to me by my lamented colleague Dr. Thirial, tells in favor of my views regarding syncope caused by embolism. The details are long; but this you will excuse from the interest which attaches to the case:

"M. X., fifty-six years of age, the head of one of the principal commercial houses of Paris, of nervo-lymphatic temperament, robust, and of a sound constitution, had almost always enjoyed excellent health. During more than thirteen years, I had been his physician, and had only had to treat him for unimportant maladies, such as rheumatic pains, and slight gastric disturbance connected with habitual constipation. He was subject to hæmorrhoids; and had long suffered great inconvenience from a very obstinate eczema podicis.

"On 20th December, 1861, M. X. sent for me. I was told, that for the five or six preceding days, he had begun, without any known cause, to experience a painful condition of the left calf, producing a certain amount of inconvenience in walking or standing. I detected neither redness, swelling, nor hardness in the affected part: the patient was in a feverish state, suffered from pains in the whole muscular system; and one or two points, rather difficult to localize with precision, were more particularly painful on pressure. From the antecedents of the patient, from his having been, as I have said, somewhat subject to arthritic pains, I was led to look on the affection as simple rheumatism.

"I was, however, not altogether free from doubt; and I even entertained a vague apprehension that the patient might be suffering from some obscure, latent lesion of the vascular system. But at the end of a few days, having seen no new symptom to confirm that suspicion, I adopted with confidence the opinion that the affection was rheumatic.

"I consequently ordered rest, different calmative and narcotic applications, and latterly, as these means produced no decided effect, I resorted to a succession of small blisters dusted with morphia.

"This treatment was continued till about the 10th of January. During the whole of that interval, the patient, in the hope of a speedy cure, hardly ever left his bed, and when out of bed for a short time he was careful to place the left limb extended on an arm-chair. After three weeks of this careful management, the pain in the calf seeming to be gone, I recommended M. X. to remain out of bed for a portion of the day, and to walk about in his room so as to prepare himself for resuming in a few days his former habits and occupations.

"Great was my surprise to be sent for on the same day that I gave these directions, and to find very decided œdema extending from the malleoli to the extremities of the toes.

"The appearance of this œdema could leave no uncertainty as to the true diagnosis. After a new examination, I soon detected, about the middle of the calf, a small hard knotty cord, which was about four or five centimetres in extent, and very slightly sensitive. Here, I ought to state that, on both legs there were slight subcutaneous and capillary varicose dilatations. The case was clearly one of very circumscribed phlebitis, the seat of which was a branch of the saphena vein; and it evidently was to this small venous inflammation that the pain in the calf, which I had considered rheumatic, was attributable. It is probable, moreover, that the nodosity was at first very slight, as, notwithstanding repeated explorations, it had escaped notice, both by me and the patient. Then again, the change of position, and particularly the walking, had favored the development of the œdema, which had no doubt been previously kept in abeyance by rest in bed and maintaining the leg in the horizontal position.

"Next day, 11th January, my accomplished colleague, M. Richet, met me in consultation. He detected the existence of a phlebitis of very limited extent, and also an œdematous swelling, which, however, from the left limb having been kept in an elevated position, was less in size than on the previous evening.

"I carefully sought for a cause, general or local, to which I could attribute the affection. In succession, I passed before me in review the habits of life and peculiarities in the health of the patient likely to throw any light on the subject; but after long inquiry, and careful analysis, I found nothing to show even the probable not to say certain character of the cause. My examination was next directed to the heart, and to the origin of the great vessels. I at once observed that there was slight irregularity, and even intermittence in the pulsations of the heart, and in the pulse; but I soon ascertained that this irregularity was only accidental, and quite transient. I saw that it was due to the mental emotion of the patient, who was very nervous, and was exceedingly disturbed by the examination. I finally satisfied myself that the heart was in a perfectly normal state. I came to the same conclusion in respect of the organs of respiration.

"The treatment prescribed consisted in the employment of topical solvents, particularly mercurial inunction. I specially recommended rest in bed or on the sofa; and advised the affected limb to be kept as much as possible in an elevated position. The tendency to constipation was directed to be kept in abeyance by injections and laxative medicines; and a restorative diet was ordered.

"Under this treatment, which was scrupulously followed for three weeks, the small venous cord gradually disappeared. By February 1st, scarcely any traces of it were left: another small branch of the saphena then became affected, and presented a slight induration a little above the point previously affected, and towards the outside of the calf. M. Richet was again called in. With the exception of difference in situation, this limited phlebitis, that is, the obliteration of the vein, presented exactly the same characters as the preceding obliteration: it consisted in a very limited and very slightly painful nodosity. It is important to note, that the œdema disappeared at the end of some days, and was not in any degree reproduced. Similar treatment was resumed, with the addition of tonics, particularly quinine in Malaga wine, recourse being had ultimately to alkaline and sulphurous baths. The second attack lasted only a short time: from motives of prudence, however, the patient kept to his bed, or bedroom, for a fortnight.

"At the end of that period, when the patient felt assured that no return of the malady resulted from his leaving his bed and walking about his rooms—not even the slightest appearance of œdema—he began to drive out in a carriage; and soon he made some excursions on foot. Under the beneficial influence of this new mode of life the appetite, which had been long much impaired, was soon restored. Strength and plumpness speedily returned. Gloomy forebodings gave place to restored cheerfulness and confidence.

"I must mention, however, that notwithstanding progressive restoration, there still remained a certain degree of weakness in the left limb, which was not accompanied by the slightest pain at the points where the vein was obliterated.

"M. X. had, for nearly a month, resumed his ordinary habits of life; and every one was eager to congratulate him upon his recovery, which, long waited for, had come at last with every appearance of secure permanence.

"But, unfortunately, all was not yet terminated. On the 15th March, I had the mortification to be recalled to the patient in consequence of his having had a new relapse. The malady, on this occasion, in place of creeping about its starting-point, went ahead at a bound. It thus advanced to about the middle of the inside of the left thigh. There was a small, hard, knotty, and almost indolent cord, of not more than from four to five centimetres in extent, situated in the course of one of the rather superficial veins.

"It was impossible for M. Richet and me not to feel some anxiety regarding this case, seeing that successive relapses had occurred, when apparently there was reason to believe that a cure had been obtained; and seeing also the rapid ascent of the malady, which had quickly mounted so high as to threaten the great venous trunks with phlebitis. These relapses, all of which occurred without any external appreciable morbid manifestation, revealed the existence of a persistent internal cause, whatever it might be; and we could foresee neither when nor where this cause would cease.

"In addition to topical remedies, I prescribed, with the view of preventing the internal coagulations, the use of iodide of potassium and Vichy water, the latter to be taken with meals. Likewise, as it was evident that the habitual constipation of the patient must cause an impediment to the venous circulation, and so favor coagulations, I ordered a dose of castor oil to be administered every two or three days. Rest as before was again enjoined.

"This treatment was most exactly followed. After eight days, the absorption of the intra-venous clot seemed to be already considerably advanced: at this age, the little nodosity, on account of the slight extent to which it projected, and its slight degree of sensibility, could not be found without a certain amount of attention.

"It was decided that M. X. was to get up on the 23d March for the first time since his last relapse; but not having found him so well on the morning of that day, I told him that it was necessary to wait. Feeling better, he rose towards evening, sat at table to dinner, and ate with a good appetite. He passed the evening cheerfully with his family.

"At ten o'clock, he went to bed. In making the necessary movement to stretch himself in bed, he was suddenly seized, in the precordial region, with an acute sensation of pain, which only lasted for a short time. He attributed it to nervous spasm; and mentioned it to no one. Two hours later, he awoke. With a view to place, according to custom, a pillow under the affected leg, he stooped, rather abruptly, to take up the pillow which had fallen at his bedside: that exertion was followed by a momentary return of the pang. At midnight, he awoke with a feeling of general discomfort, accompanied by slight shivering, which gradually subsided in light sleep.

"On the morning of the 24th March I saw the patient, who then told me with an anxious air of what had occurred since the previous evening. The statement, I confess, seemed to me rather unusual, and led me to apprehend some new incident. I auscultated the heart rapidly, and to a certain extent stealthily, to avoid alarming the patient, who followed all my movements with a troubled expression. I thought I heard, at the base of the heart, a slight morbid sound, the nature of which I could not determine with precision. The pulsations were one hundred in the minute, unequal in strength, and, apparently, a little confused. I was, however, disposed to attribute these disturbances of the circulatory system to the extreme emotion of my patient, who, I perceived, was engrossed with the incidents of the night, and with my examination.

"I prescribed an antispasmodic draught, did my best to reassure him, advised him to rest quietly in bed, especially telling him not to get up in the evening, unless he felt perfectly well.

"From prudential reasons, the patient only took a basin of broth to his breakfast. He passed a good day: in the afternoon, he received some visits. At half-past four, feeling himself comfortable, he resolved to rise, and dine with the family.

"He got out of bed, and sat in an arm-chair to dress himself. He had hardly pulled on his drawers, when he was seized with an indescribable pang at the heart, and felt that he was fainting: he had only time to call his wife to his aid when he became insensible.

"I was sent for in all haste. By a fortunate chance, I arrived at the house at that very moment. I found the patient seated in his arm-chair: he had recovered consciousness, but was icy cold, livid in the face, sunken in the eye, almost pulseless, and making efforts to vomit. With all possible expedition, I laid him on his bed, placing his head somewhat low, and causing him to swallow a few drops of a cordial: I subsequently had recourse to external and internal stimulants of every kind. For more than an hour, during which time I was a prey to the greatest anxiety, I fought against the syncope, against the state of profound collapse, which lasted so long that I dreaded every moment a fatal issue. Of all my efforts, my greatest was to give courage to the unfortunate man, who believed himself to be hopelessly lost.

"My endeavors were in the end successful; little by little reaction set in, and ultimately became pretty powerful: the pulse rose to 108; but the pulsations were for a long time small and concentrated.

"During the evening, M. Richet met me in consultation on the case. After describing to him all that had occurred since the previous evening, we proceeded together to make a careful inquiry, with a view to discover, if possible, the reason of the serious incidents. Let me here remark, that during the whole of the crisis, I observed neither notable disturbance of the respiratory functions, nor any true dyspnœa.

"Our examination by auscultation and percussion informed us, that there was nothing abnormal in the respiratory functions. It was otherwise in respect of the circulatory system. The pulse, as I have already said, was pretty high, being 108, but did not present any notable irregularity. At the base of the heart, Dr. Richet and I heard a slight morbid sound, which recalled the sound I had heard in the morning. This sound, rather difficult to describe, was less like a bellows-sound than like a dry clapping. M. Richet thought it proceeded from one of the auricles. That I may omit no fact useful to mention, I may state, that there was considerable gaseous distension of the stomach, which somewhat pressed upon the heart. We on several occasions noticed this state of meteorism, and remarked, that it was not associated with the slightest dyspnœa. Although immediate danger seemed averted, it was evident that the malady was entering upon a new phase, which was one of some gravity and great obscurity.

"Had we had to do with a case of syncope, pure and simple? What was the cause of the syncope? To solve these questions, we reviewed all the probable circumstances, great and small, which could have produced the syncope; such as the state of debility to which the patient had been reduced by his disease and its treatment, the transition from his for some time customary horizontal position to the upright and sitting attitudes, and the exertion of dressing. But we felt that had the fainting depended on such slight causes, it was not likely to have been either so intense or so protracted.

"We asked ourselves, whether so profound an attack and one of so threatening a character, preceded, the previous evening, by functional disturbance of so unusual a character, might not indicate miasmatic syncope?

"And again, might not collapse, so sudden and so prolonged, arise from embolism?

"We considered that we were bound to give prominence to this question, which must evidently raise many objections and difficulties.

"To sum up: As the syncope might be dependent on debility, or might be the manifestation of a 'pernicious' fever, we were decidedly of opinion that quinine was indicated in virtue of its neurosthenic and antiperiodic properties: we consequently prescribed a potion containing 60 centigrammes (9 grains) of quinine, and 4 grammes (62 grains) of extract of cinchona: and we likewise advised the patient to abstain from sudden movement and violent exertion.

"25th March. The night passed without any noteworthy occurrence. I found that he had had some hours of tolerably quiet sleep. In the morning, the pulse was 96; and I noted that the patient had had a natural stool without the assistance of an injection: I use the term *natural*, so as to eliminate from the description anything calculated to cause a suspicion that intestinal hemorrhage was the cause of the syncope. Broth and the quinine potion were ordered to be continued.

"26th March. After a quiet day, he passed a night even better than the preceding: he had several hours of sound sleep. In the morning, the pulse was 54, and quite regular. There did not remain the slightest morbid sound of the heart. Broth and soup were ordered.

"The family, naturally alarmed by the scene which had occurred two nights previously, expressed a wish that Dr. Bouillaud should join us in consultation. The consultation was fixed for the same evening, when, to our great regret, Dr. Richet was unable to attend.

"Dr. Bouillaud having made himself acquainted with the different phases of the malady, and particularly with the recent crisis, proceeded to the diagnosis with all that care which belongs to him. In the middle of the thigh, he detected a venous cord in process of resolution, and then reduced to a rather thin knotty thread. The respiratory organs were found in a perfectly normal state. He examined the organs of circulation with very great attention. All at once, he detected a certain irregularity, and even a little intermittence in the pulsations of the heart and in the pulse at the wrist: but he soon ascertained that this disturbance was transient, and entirely due to the emotion of the patient, as I had previously observed more than once. Dr. Bouillaud satisfied himself that there was no abnormal sound audible in the heart or great vessels; and he announced that, with the exception of a certain degree of nervousness, there was nothing appreciably wrong with the central organ of circulation.

"I next wished to know Dr. Bouillaud's opinion as to the nature of this crisis which had occurred on the evening before last. I disclosed the uncomfortable impression left on my mind by that spectacle. I mentioned to him my doubts, my different conjectures in relation to that syncope so unusual in its characters; and, in particular, I did not conceal from him my apprehensions of embolism which might or not be well founded.

"Dr. Bouillaud replied that he understood my doubts, and, to a certain extent my fears, suggested by the circumstances of the malady; but, relying upon the very satisfactory result of his examination, upon the good appearance of the patient, and particularly upon the absence of all untoward symptoms since the last crisis, he looked upon the syncope as an accidental occurrence of a purely nervous character. His opinion was, that, in all probability, the case would have a favorable issue.

"Dr. Bouillaud did not hesitate to reassure the family by announcing this conviction, who were only too happy to hear so comforting an opinion from so great an authority. He recommended that the patient should be at once strengthened by tonic medicines and restorative diet; and likewise advised that he should be sent to the country as soon as possible.

"Notwithstanding my great confidence in the enlightened experience of Dr. Bouillaud, notwithstanding the excellent reasons in favor of his prognosis, it was impossible for me to share in his view of the case. The syncope did not seem to me to have been ordinary syncope: certain insidious preliminary phenomena, and the whole group of symptoms, led me to believe that it was of a suspicious nature. Whether it was my solicitude for a patient to whom I was attached by an old and deep affection, or a secret invincible presentiment which possessed me, I know not; but so it was, that I felt that the disease had not yet said its last word, and in spite of myself, I dreaded a new and early attack.

"My apprehensions were so decided, that I deemed it my duty to disclose them to a near relation of the family, so that, while he kept his motives a secret, he might, in advance, point out to the family all the precautions necessary to be taken in the event of a fresh alarm.

"The consultation, however, had produced a happy effect on the patient. Since the last crisis, he had been in a state of extreme anxiety: he had been living under the constant dread of a return of the syncope, which, he said, having come without warning, or any appreciable cause, might any moment unexpectedly return in similar fashion, and carry him off. But the reassuring, convincing opinion of Dr. Bouillaud coming to strengthen my own encouraging expressions, his misgivings were shaken, and he soon even attained to some extent a state of serenity.

"He passed, then, the night of the 27th very quietly. In the morning, the pulse was 80, and perfectly natural. With a return of confidence, there was also a tendency to a return of appetite. During the day, the patient several times took broth and chicken. The cinchona wine was continued at meals. To meet his wishes, the sulphate of quinine, which he had hitherto taken in potion, was prescribed in pills.

"On the 28th March, he had as good a night as on the 27th. He ate with appetite a mutton cutlet at breakfast. As all was going on well, it was agreed, that he should, for the first time since the fainting, be allowed to change his bed, to sit on a chair, and to make his toilet.

"Everybody, particularly the patient, had misgivings as to this trial. I resolved, therefore, to be present, to inspire confidence, and to give succor in the event of its being required. Fortunately, all passed off to a wish, the patient not feeling the slightest discomfort nor the least disturbance of respiration or circulation. During the day, he received some visits with an air of greater satisfaction than he had shown for a long time. He dined on soup and chicken. He passed the evening after dinner with the family. He joined willingly in conversation, and even read aloud the newspaper.

"At ten o'clock, the family retired to rest. At eleven, M. X. was asleep. After sleeping quietly till two in the morning, he awoke. His wife, who lay in an adjoining bed, rose to administer to him his usual cinchona pill. He sat up to take the pill, swallowed it, and then drank a mouthful of water. Having done so, without the least complaint of discomfort or suffering, he again lay down.

"Madame X. had hardly returned to bed, when she heard her husband groaning as if suffocated. She called him, and asked him if he were suffering. He replied in the negative. She lay down at the foot of his bed; and then saw that he was pale, exhausted, unconscious, and motionless.

"I was sent for in haste. As I lived in the same building, I was with my patient in less than ten minutes. What a spectacle! Some hours previously, I had had left him full of life and hope: I now found him icy cold with death stamped on his face: he was pulseless, there were no appreciable movements of the heart, and only some respirations at long intervals. In vain I tried to restore him by the use of stimulants. In five or six minutes he expired.

"As there was no autopsy, some degree of uncertainty must remain as to the cause of death. Nevertheless, I do not hesitate to attribute it to embolism: the phases of the malady, the order in which the symptoms occurred, the most characteristic phenomena considered by themselves, all the facts seem to concur in favor of this interpretation.

"It has appeared to me that a complete and faithful picture of the disease could alone supply, up to a certain point, the want of a post-mostem examination. This is my reason for giving the complete details. The reader, having been present, as it were, at all the catastrophes of the case, and taken part in all my impressions, doubts, and divergences of opinion, is in a position to judge for himself as to the nature of this affection, which was so irregular, obscure, and insidious in its course.

"Among cases of the same class, this case presents a special, and I may say exceptional, character: for a long time, there existed certain indications leading to the suspicion of embolism, to a certain extent enabling us to foresee, but, unfortunately, not to prevent, the fatal catastrophe.

"In the first phase of the affection, we followed the development of the phlebitis, the expression of a probably general but very obscure cause; and we saw this phlebitis, at intervals of varying duration, give rise to very circumscribed coagulations in the lower extremities.

"So long as the malady, which was then moreover slight, did not go beyond the leg, there was nothing to create much anxiety. But I began to be anxious when the phlebitis went on without interruption, to gain the middle of the thigh, and approach the neighborhood of the great vessels; for from that moment, it was impossible to say where the morbid process might terminate, and what might be its consequences.

"The second phase soon made its appearance; and with it came the worst consequences which could have been present: this phase was completed in two periods separated by an interval of only a few days.

"Its commencement was marked by the great crisis of 23d March: as the sequel of some preliminary symptoms, it burst forth in the form of a frightful fit of syncope, occurring when the patient was getting up for the first time, and just as he was beginning to dress. As the crisis, notwithstanding its violence and duration, had no evil consequences, doubt as to its real nature might, for a moment, be entertained; but the progress of the disease left no room for uncertainty on that point; and it would be difficult, after the fatal issue, not to recognize the effects of embolism in the first seizure.

"To explain the case, I conclude that, from the coagulum seated in the thigh, and in full process of being absorbed, or perhaps from some unobserved coagulum, a small fibrinous or cruoric fragment had been detached, at the moment when the effort was made; and I likewise suppose that this fragment, becoming quickly involved in the current of the circulation, reached the right side of the heart: thence arose, in my opinion, the sudden syncope—the collapse so profound and so threatening.

"The heart, however, under the influence of stimulants, became roused, and recommenced its movements. The clot—supposed to be small and soft—taken up, and beaten as it were by the wave of blood—though, perhaps, not dissolved, was broken down into grumous particles sufficiently small to be lost in the ramifications of the pulmonary artery. It has been seen, moreover, that the heart, for some time after the accident, continued in an agitated, disturbed state: it may be remembered that several hours after that occurrence, I detected an abnormal sound in the heart: it is no doubt quite possible that this sound was simply dynamic, but it is also possible that it had some relation to a fibrinous concretion adherent to one of the orifices or to the columnæ carneæ of one of the cavities of the heart.

"Be the explanation what it may, the greatest crisis, notwithstanding all my fears, had not a disastrous issue. During the two days which followed it, there was a tendency to a restoration of calm; the patient gradually regained confidence, appetite, and good sleep: all the functions were performed in a natural manner: and the state of the heart was quite normal. Nothing presaged immediate nor even remote danger. On the contrary, everything seemed to justify a favorable prognosis, and two days elapsed during which no sign of contrary import manifested itself.

"This calm, however, was a deceitful calm. At a moment when it was least to be expected, in the silence of the night, on awaking from a tranquil sleep, the patient was, all at once, after a slight effort, struck as if by a thunderbolt: he was heard to groan, and in less than an hour he was dead.

"The first seizure was only a menace: the second led up to the fatal issue: but in my opinion, both seizures proceeded from the same cause. It is very probable, that a clot, already free, or only slightly adherent, was shaken about and put in motion by the effort which the patient made to sit up; and that within a very short time, it reached the right auricle.

"This clot, whether from its size, or from some other cause difficult to define, became engaged in one of the cavities of the heart, and immediately determined syncope with profound collapse, from which there was no rally."

Gentlemen, if a post-mortem examination had been made, it is probable that it would have demonstrated the cause of the syncope to have been in the right side of the heart. The cause was not an organic disease of the heart, for neither general nor local signs of such an affection had ever been discovered. On the other hand, when it is considered, that upon two different occasions, with a long interval of time between them, when making the necessary exertion to dress, he was seized—suddenly, on both occasions—with an indescribable pang at the heart—that he fainted and lost consciousness, is not one led to conclude, that a migratory clot, detached from a vein by the exertion, took the heart by surprise and impeded its action, causing forthwith pain, heart-pang, and mortal syncope?

Pathological Anatomy of Phlegmasia.—Œdema of the Subcutaneous and Deep Cellular Tissue of the Affected Limbs.—Coagulation of the Blood in the Superficial and Deep Veins.—Fibrinous and Cruoric Clots.—Fibrinous Clots in the Valvular Pouches.—Absorption of Intra-venous Clots.—Tendency in these Clots to become organized.—Cellular organization of these Clots, and the Permeability of the New Tissue.—Persistent Fibrous Obstruction of the Veins: Collateral Circulation.—Pseudo-purulent Softening of the Clots.—Organic Causes seemingly Favorable to Intravenous Coagulation at particular points.—Absence of Lymphangitis and Adenitis.

Let us now consider the pathological anatomy of phlegmasia alba dolens. In the present day, there are only two views on this subject which merit discussion. In painful œdema, is there a lesion of the lymphatic system? Ought we not rather to believe, that a structural change takes place in the venous trunks and branches?

I have already remarked, that the œdema of phlegmasia shows itself at the extreme and most dependent parts of the body. Thus, it begins in the feet and ankles, then shows itself higher up, while, at the same time, it is chiefly conspicuous in the lower extremities and most depending parts; that is to say, at the posterior part of the calves, thighs, and trunk, when the patient is lying on the back. The skin is tense, the dermic meshes are enlarged; and there are perceived, on the dead as well as on the living body, a blue marbling, arising from a fretted state of the dermis, and its greater transparency in the situations where it is fretted. The limb is deformed and rounded, and presents at the joints an appearance of being strangled.

When incisions are made in œdematous limbs of this description, there pours forth a large quantity of colorless or slightly yellow serosity, in which float numerous globules of fat. Sometimes, the serosity retained in the meshes of the cellular tissue under the aponeuroses and in the intermuscular spaces, seems coagulated, and pits on pressure. The muscles, isolated in the midst of the serosity, do not seem to be penetrated by it; but they are pale, and very flaccid. Vessels and nerves are either dissected, or are in such a state as to be very easily dissected. In situations where the sheaths of the vessels are provided with cellular tissue, the serosity is very abundant.

Deeper incisions show that many veins of variable size are distended by black coagulated blood, or by small yellow fibrinous concretions. If, then, proceeding in our examination, we ascend from the foot to the origin of the affected limb, and dissect the superficial and deep veins, we observe, that the principal trunks are hard, that they roll under the finger, and that sometimes the circumvascular cellular tissue creaks under the scalpel; but invariably, throughout a greater or less extent, the vessels feel resistant to pressure: their walls seem distended, here and there, knots, corresponding to the valves of the veins, being visible. Sometimes, the walls of the veins retain a great amount of transparency, so that the vessels can be seen to be filled with black coagulated blood. At other times, a vein has a dull appearance; and when laid open by the knife throughout its whole course, it is seen that the clots vary in different places in color and consistence. The blackest clots are generally found at the most distant parts and at the periphery of the limbs; and the nearer the clots are to the trunk, the more fibrinous and yellow do they appear. However, it is not unusual, in the deep veins of the calf and thigh, to meet with very resistant clots in which the fibrinous exceeds the cruoric portion.

The clots in which the fibrinous part predominates are probably the oldest, that is to say, those which first obstructed the circulation. Indeed, the situations in which pain is first felt are those in which the fibrin is found to predominate; as, for example, at the point where the saphena opens into the crural, and in the deep veins of the calf.

There is still greater reason to believe that the cruoric clots, by far the most extensive, are formed consecutively. It is important to remark, that it is at the places where the fibrin predominates, that one can study those modifications which cause certain parts of softened clots to be carried into the current of the circulation to become migratory clots, emboli, the consecutive anatomical phenomena produced by which we shall have to determine.

The fibrin, in certain cases, may have a tendency to become organized: the clot then presents interstitial cellular partitions placed obliquely, transversely, or parallel to the axis of the vessel: some of these partitions or trabeculæ are inserted in, and incorporated with, the internal coat of the vein. Fibrin, yellow or nearly white, is found in the spaces between the trabeculæ. The organization of the trabeculæ is not of a doubtful character: in the lamellæ which are most resistant, and evidently oldest, the microscope discloses a fibro-cellular structure, a considerable deposit of amorphous matter, and numerous fat-globules: in the lamellæ in course of formation, a similar structure is found, except that the fibre-cells are fewer in number and less distinct.* The fibrin within the partitions is granular, and contains some more or less altered blood-globules.

All these anatomical details were verified in a patient treated in my wards, whose case is reported by Dr. Dumontpallier, who, aided by M. Charcot, studied the appearances under the microscope.

We have seen that the obliterative clot consists of two very distinct portions—a cruoric, by far the largest, and a fibrinous portion. The latter is the most interesting to study in relation to its ulterior modifications. One of four things may occur: the clot may break up, and be slowly absorbed; it may burrow parallel with the axis of the vessel; it may hollow out irregular cavities, and present an appearance similar to cavernous tissue; or it may become transformed into a fibrous cord. In the first three cases, the circulation is re-established: in the last-mentioned case, it finally ceases. When the clot has undergone the fibrous transformation, it adheres strongly to the walls of the vessel, which at these points is contracted on itself. There is no doubt as to the fibrous character of these clots: and sometimes they are even infiltrated with calcareous matter, which renders them very resistant. At the points where the clot is thus transformed, it is not unusual to see the *vasa vasorum* of the vein increased in number, and sometimes in diameter.

Every venous obliterative clot may undergo connective transformation; and that transformation may lead to a persistent obliteration of the vessel converting it into a fibrous cord, or producing a cavernous, trabecular, and permeable condition of the vein, through which the circulation is re-established.

These different modes of transformation are the result of a process of organization seated in the clot, and probably originating in its fibrinous portion. In these cases, the fibrin has an organizing power, the last term of which is the production of connective tissue. At other times, the fibrin becomes softened throughout its whole extent, and then nothing is found in the vessels except more or less diffluent masses, of a brown or yellow color, and mingled with perfectly recognizable blood. Again, at other times, these soft masses are encysted, so to speak, in portions of hard clot adherent to the walls of the vessel, and in these cystous cavities we occasionally meet with a fluid of purulent appearance, which, when examined under the microscope, is found to be only amorphous granular fibrin, and a quantity, variable (sometimes very large), of white globules, which, by being mistaken for pus-globules, have led to the conclusion that the clot may be transformed into pus. This opinion may appear tenable, particularly when there is thickening of the walls of the vessel, and when they present the traces of phlebitis. But in cases of phlegmasia, suppuration, properly so called, is exceedingly rare; and the truth is, that the purulent appearance of the clot is usually nothing more than a modification of its fibrinous portion.

But although the clot in softening does not give rise to purulent infection, it is frequently the origin of the exceedingly interesting phenomena upon which Virchow has based the theory of venous embolism.

Before I say more on the subject of embolism, I wish to address some words to you upon the clots observed at the valves of the veins, and on the part which the valves play in causing the coagulation of the blood. The formation of the clots is consecutive to the obliteration of an upper part of the venous system, and is induced by the eddy and stasis of the blood when they are formed in the inferior portion of that system. If the iliac or femoral vein be obliterated, the venous blood, prevented from taking its normal course, makes a way for itself through the collateral veins; the result is a temporary impediment to the entire column of blood below the obstruction, an eddy and finally a stasis, particularly at the valves, which are then depressed by the recurrent stream. This last condition is specially favorable to coagulation of the blood, which then takes place under the double influence of the inopexia which has produced the primary coagulation in the iliac or femoral vein, and of the anatomical conditions which the valves present to the too fibrinous blood. When the primary coagulation is not dependent upon an affection of the uterus, the obliterated veins of which have gradually caused an extension of the obliteration to the hypogastric venous system, when, for example, in the subject of tuberculous or cancerous cachexia, the anatomical condition added to the inopexia, is stricture of the veins at the points where they traverse the aponeuroses, this is what takes place in the axillary and popliteal hollows and in the fold of the groin. That the formation of the valvular clots is consecutive to an obliteration of an upper portion of the venous system, is shown by their being fibrous, by their structure being lamellar and much less perfect than that of the clots situated higher up, which are much more intimately adherent to the walls of the vein. This was well seen in an anatomical preparation presented by Dr. Duguet to the Société de Biologie.

Pathological Anatomy of Pulmonary Embolism.—Serpent-head appearance of the Cardiac Extremity of Intra-venous Coagulation.—Softening of the Head of the Clot.—Its Rupture.—Pulmonary Embolism of Various Dimensions and Forms.—Occupying Infundibulum of Pulmonary Artery.—Generally arrested at a spur of the Artery.—Obliterating completely or incompletely one of the principal divisions of the Artery.—Embolism sometimes continuous with newly-formed Clots.—Embolism recognizable by its Structure, Valvular Débris, and Special Prolongations.—Embolism of the Principal Divisions of the Pulmonary Artery causing Pneumonia, Gangrene, and consecutive Hydropneumothorax.—Embolism occasioning sometimes numerous Pulmonary Abscesses.

Let me lay before you the report by Dr. Dumontpallier of a case which occurred in my wards, which will serve to establish the doctrine of pulmonary embolism.

A young woman, delivered at the Maternity, during October, 1858, left that hospital, nine days after her confinement, in a state which seemed very unsatisfactory.

At the beginning of November, she asked to be admitted to the Hôtel-Dieu, that she might there have her infant cared for. At the middle of November she was seized with pain and œdema of the left lower extremity, *phlegmasia alba dolens*, characterized by pain, œdema, and a venous cord, extending into the popliteal hollow. The femoral vein contained an extensive and very obvious coagulum.

The venous circulation seemed gradually to become re-established in the left lower limb, and the œdema had almost entirely disappeared, although the venous cord remained; but, suddenly, the patient felt pain in the right iliac fossa and calf of the right leg, which pain soon disappeared, and was not followed by œdema of the right limb.

During the first days of December she was suffering more from general discomfort than from definite disease; but on the 8th of that month she was suddenly seized with pains in the right side of the chest, and difficulty of breathing. The inspirations were short and frequent. On auscultation, moist râles were heard; then a blowing sound and pectoriloquy were detected in the upper and posterior part of the chest: at a later period, in the lower part of the chest, a bellows-sound and ægophony were heard. The sanguinolent expectoration was not like that seen in acute pneumonia: from the second day of the thoracic symptoms, the sputa resembled those of pulmonary apoplexy; from the fourth day, they had an increasingly gangrenous odor; and on the seventh day the patient died, respiration having become more and more rapid before death. The pulse ranged between 130 and 140 in the minute. The tongue was dry, and the gums were covered with sordes. There was no diarrhœa. During the last twenty-four hours of life, the body was covered with a profuse cold sweat.

I had enunciated the idea that the gangrene of the lung might be due to transport of a clot—part of the clot of the phlegmasia—to one of the divisions of the pulmonary artery. In support of that opinion, I called attention to the abrupt manner in which the pulmonary affection commenced, the frequency of the respiration, the difference of the expectoration from that of pneumonia, and the rapidity with which pulmonary gangrene was manifested in a woman delivered two months previously, and who had

had phlegmasia alba dolens. My attention had been directed to this subject by the researches of Virchow and a recently published memoir of Dr. Charcot.

Let me now give you an account of the appearances observed at the autopsy. The crural, saphena, and popliteal veins, and the veins of the calf, were full of colored clots of varying consistence and structure, which throughout the greater part of their extent were free, and only adhered at some points to the walls of the veins.

The femoral vein in its upper part, at the crural arch, contained a fibrinous clot from four to five centimetres in length, of a pink color, perfectly organized, hard to the touch, composed of longitudinal striæ, which in its whole extent adhered to the walls of the vessel. The internal surface of the vein had no downy smoothness, but was so intimately united to the clot by lamellæ and filaments of connective tissue, that it could not be removed without tearing this tissue. The cellular tissue surrounding the vein was indurated, œdematous, and creaked when cut.

The femoral clot was continuous inferiorly with a fibrinous clot mixed with much of the red portion of the blood: it was brown, and this color was of a deeper shade the nearer it was to the popliteal vein. The same femoral clot was continuous superiorly with a semi-fibrinous, semi-sanguineous clot, which was highly organized, but non-adherent, in the common iliac and external iliac veins and in the vena cava inferior.

There was a fibrinous clot in the vena cava inferior, to a certain extent obstructing that vessel. This clot was of a pink color, fibrinous, resistant on pressure, composed of longitudinal striæ, and non-adherent. Its length was five centimetres, and its diameter one centimetre; it was flattened from before backwards, and terminated a little below the mouths of the emulgent veins, in the form of a soft, slashed stump, to which were attached five or six small pedicles of fibrinous clot resembling portions of a lumbricus; some clots of similar nature and form appeared to be unattached. It is well to remark that the autopsy was performed with every possible precaution, the dissection being made whilst the parts were *in sitû* and in their normal relations. The clot in the vena cava inferior terminated, as I have said, in the form of a stump, and was continuous posteriorly with an exceedingly small membranous fibrinous clot, which was joined to a large fibrinous clot above the emulgent veins. This latter, which was non-adherent, occupied almost the entire cavity of the vena cava, in its hepatic portion, where it received many other fibrinous clots of different sizes belonging to the subhepatic veins. It reached the right auricle, and then extended into the ventricle, after sending off a fibrinous prolongation into the vena cava superior and vena innominata. Within the heart, the clot was yellow, fibrinous, and seemingly adherent, from its numerous prolongations extending into the interstices of the columnæ carneæ.

The entirely fibrinous cardiac clot was continuous with a clot, partly fibrinous and partly cruoric, situated in the pulmonary artery and its two principal divisions, extending likewise into divisions of the second and third order of that vessel. These semi-fibrinous, semi-cruoric clots had evidently been formed during the latter moments of life and after death, judging from their structure and slight consistence. I shall afterwards have to speak of other clots, some of which were found in the principal divisions of the pulmonary artery. At present, I proceed to describe the anatomico-pathological examination of the parenchyma of the lungs, more particularly of the right lung. The entire examination, I repeat, was made with the organs remaining in their natural situation.

The right pleura contained a sero-purulent effusion, and purulent false membranes; the lobes of the right lung adhered to one another by cellular adhesions and false membranes.

The surface of the inferior posterior portion of the upper lobe of the right lung had a dark-brown color for about from four to five centimetres square. In that situation, the pulmonary tissue was exceedingly soft: and, on insufflation, it was found that the lung was perforated at the place where it was gangrenous. The perforation had not taken place till the last moments of life; or perhaps, not even till after death; for the patient never had any signs of pneumothorax.

The perforation of the lung led to a gangrenous anfractuosity, large enough to contain a pigeon's egg. The appearance of the pulmonary tissue, and the odor of the affected parts, left no doubt as to the gangrenous character of the local lesion.

That having been ascertained, I resumed the dissection of the pulmonary artery. I observed that the large branch which serves the superior lobe of the lung contained a fibrinous clot adherent to the walls of the vessel: it was of a pink color, had very well-marked longitudinal fibres, and was in all respects similar in structure and appearance to the clot found in the vena cava inferior. It was three centimetres in length, and was posteriorly continuous with a much less organized fibrinous clot, and continuous anteriorly—that is to say, towards the divisions of the third and fourth order—with softened cruoric clots.

The principal clot of the pulmonary artery was not in any degree softened, and was, in all parts, of uniform consistence.

The portion of lung which presented signs of hepatization and gangrene corresponded with the division of the pulmonary artery obliterated by the fibrinous clot.

I am not going to endeavor to explain how obliteration of a division of the pulmonary artery may occasion gangrene of the lung: I only wish to remind you that Virchow, by his experiments on dogs, was led to believe this possible.

I must add, that in the case now under consideration, I found no obliteration of the bronchial arteries and veins to account for the gangrene of the lungs. I found obliteration of the pulmonary artery coexisting with gangrene of the pulmonary tissue. It is probable, that the embolus which caused the obliteration also caused the gangrene.

There was no lesion of the left lung.

The examination of the uterus and its appendages presented nothing requiring mention. There were no traces of inflammation in the utero-ovarian and hypogastric veins.

It may be concluded, from the facts now stated, that the phlegmasia alba dolens was due to the obliteration of the crural vein; that the crural clot was prolonged, by juxtaposition of fibrin, into the vena cava inferior; that the fibrinous clots (both those which were free and those which were adherent to the surface of the clot of the vena cava) might have given rise to venous embolism; that at the time when chest-symptoms supervened, an embolus was carried into the pulmonary artery, and arrested in the superior division of that vessel in the right lung; and that gangrene was the result of this obstacle to the pulmonary circulation.

The fibrinous coagulation met with in the other divisions of the pulmonary artery, vena cava superior, heart, and hepatic portion of the vena cava inferior, seemed to have been produced progressively by the obstacle to the pulmonary circulation during the latter hours of life.

To sum up: A recently delivered young woman was affected with phlegmasia alba dolens; and three weeks after the commencement of that malady, when she believed that she was cured, she all at once felt acute pain in the right side of the chest. Very soon, the expectoration of pulmonary apoplexy was observed, which expectoration afterwards acquired a gangrenous odor—and the patient died. The lung was found in a gangrenous state, in that portion served by a branch of the pulmonary artery obliterated by an old fibrinous clot, similar in all respects to another peripheric clot found in the vena cava inferior, which at its free portion had a jagged appearance and marks of being torn. It is reasonable to suppose that during life, probably under the influence of an exertion, and of a strong wave coming from the emulgent veins, a portion of the clot of the vena cava, softened at its superior extremity, was broken up, detached, and then carried to the lung, where it determined the symptoms which have been described. This migratory clot, by becoming arrested in the pulmonary artery, first occasoned a hemorrhage, and then gangrene limited to that portion of the lung to which the obliterated branches of the vessel were distributed.

Now that you are acquainted with the conditions in which embolism may occur, and the phenomena to which it may give rise, I proceed to describe that anatomical state of the clot which favors the formation of venous emboli.

All observers agree, that the superior extremity of the venous clot has a special form. The venous clot has a tendency to become prolonged towards the heart, and generally goes on extending till it reaches the mouth of some considerable branch. That you may quite understand this disposition, let us suppose a clot spontaneously formed in the crural vein: it has been observed that such a clot will generally become prolonged into the external iliac to the mouth of the hypogastric vein: there, new layers of fibrin, furnished by the hypogastric circulation, will give particular form to the clot. In that situation, the superior extremity of the clot often takes the shape of a slightly flattened serpent's head, free from adhesions and continuous with the original clot.

The head of the clot is formed of two parts, viz., the cortical, which is resistant and fibrinous, and the central, which is softened: it is to this central softening that the head of the clot owes its flattened form. The softening also leads to other and much more important consequences: it first of all extends to the peripheral parts of the clot, and there induces disintegration, so that it becomes easily torn: then, the portion of the clot set free in this way may be carried by the circulation, and give rise to the phenomena of cardiac or pulmonary embolism.

In fact, the head of the clot constantly receives the shock of the blood carried by the nearest afferent vein; and if the clot be softened at any point, we can understand that a portion may become detached, and be carried on to the vena cava, the heart, and the lung. It will there produce symptoms exactly similar to those which Virchow produced at pleasure, in his experiments, by introducing foreign bodies into the jugular vein. The portion of clot liberated from its attachment, in the manner I have described, is simply a foreign body, which will advance towards the heart till stopped at the angle formed by a branch of the pulmonary vein, or by some subdivision of that vessel too narrow for its further passage.

The remaining portion of the peripheric clot then presents a slashed fibrinous and cruoric extremity, floating like the fibres of the root of a tree; or it may be entwined, so to speak, in new fibrinous clots—clots formed in the last struggle of life—which may, as in the case I described, become so prolonged, as to extend with apparent continuity from the original clot to the pulmonary embolus.

This apparent continuity of clot has furnished the opponents of the theory of embolism with an objection. They say that in the case the details of which I have now laid before you, and in other similar cases, what is seen is only the continuance and extension of the femoral clot into the vena cava inferior, right auricle, and pulmonary artery. In reply to this objection it is sufficient to state, that the structure of the clots is quite different. Though the structure was the same, the clot in the vena cava inferior was of as old a date as that in the pulmonary artery: it was quite otherwise in the space intervening between these two extreme clots: this was a fact not admitting of any dispute, as the interposed clot was fibrinous, gorged with serosity, a veritable fibrinous sponge similar to the clots observed in the right side of the heart consequent upon the final struggle. The clots situated at the extremes, although formed of fibrin, were resistant, the fibrin having undergone retrogressive changes. Moreover, it has been ascertained by clinical observation, that there had been œdema of the left lower extremity at a particular period, suddenly followed by urgent dyspnœa, and pathological phenomena attributable to the lungs. Bear in mind this important fact, that in our patient, the contractions of the heart had regained their natural frequency and amplitude: it was not till after the lapse of some time, that all terminated in the pulse becoming small and irregular, and the breathing exceedingly disturbed.

It being admitted, that there has been an interval of variable duration between the phenomena attributable to pulmonary embolism and to death: it being, moreover, admitted, that the intermediate clot was of most recent date, the opponents of the theory of embolism object, that the old pulmonary clot might have been indigenous—that is to say, that spontaneous coagulation had taken place in the pulmonary artery, just as it might have occurred in a peripheric vein, the crural vein, or any other vein. To this objection, clinical observation replies forthwith, that the thoracic phenomena have not occurred till a period long after the existence of the phlegmasia; that the commencement of these thoracic phenomena did not take place till the phlegmasia had been in existence for some time; that the first manifestation of the phenomena supervened with, and was accompanied by, sudden maximum dyspnœa, and that it is impossible to say how rapidly there might be formed in one of the branches of the pulmonary artery, an obliterative clot capable of originating formidable dyspnœal phenomena, or even sudden death. Finally, pathological anatomy has shown, that the clots which produce embolism have a special form and structure, sometimes presenting distinct, indubitable marks of their peripheric origin.

These clots, generally stopped at a spur of the pulmonary artery, are only adherent by one part of their surface to the vessel; they are split into two parts, of which the central is soft, and the peripheral a fibrinous envelope. They sometimes retain the form of the serpent's head belonging to the peripheric clot, and also the jagged extremity which united it to the latter. The clot, become an embolus, is continuous, above and below, with new fibrinous and cruoric clots. When we recollect, that in cases of spontaneous intra-vascular coagulation, the blood has a special constitution, we can easily understand how the embolus should become a centre around which a new fibrinous formation is formed. It often happens, that the embolus is lost in the midst of these new clots, and that some trouble is necessary to find it.

So much for the embolus, its relations, and its continuity with secondary clots: its structure is identical with that of the peripheral clot: the solvent power of ether, and, still more, the solvent power of sulphuret of carbon, show, that the embolus and the peripheric fibrinous clots have the same composition; which renders it exceedingly probable, that the embolus has been detached from one of the clots.

For the sake of those who may be but little inclined to accord conclusive value to the arguments I have now adduced, let me add, that there are emboli which bear indubitably the stamp of their origin: they present marks which leave no doubt as to the place whence they came. Dr. Lancereaux, in a communication to the Société de Biologie, has stated, that he has seen a clot bearing the print of a venous valve. It has likewise been stated, that some clots have been found to have carried with them valvular débris discoverable in the substance of the clot. I am indebted to Dr. B. Ball for the account of a very remarkable case of pulmonary embolism, in which the clot presented three lateral prolongations. After minute and protracted research, the starting-point of the migratory clot was found in the vena azygos. At the upper part of that vessel, there had been rupture of a clot, and immediately above that rupture, the vessel presented three lateral orifices, which, in situation and calibre, perfectly corresponded with the disposition of the three lateral prolongations of the migratory clot.

It is unnecessary to say, that such facts give powerful support to the theory of embolism: they, in fact, are demonstrations of its truth.

Emboli may be capillary, and numerous: that is to say, very minute fragments of disintegrated fibrin may be carried by the circulating stream, and deposited in the capillary ramifications of the pulmonary artery. In these cases, there are observed, on the surface of the lung, numerous small ecchymoses, identical with those which M. Sedillot has described as occurring in certain forms of metastatic abscesses in cases of purulent infection.* In the centre of these ecchymoses, the fibrinous nucleus can sometimes be detected: at other times, after remaining for a long period in the capillary branches of the pulmonary artery, these minute deposits of fibrin become softened, and assume a purulent appearance. Some of the ultimate ramifications of the pulmonary artery may become injected with fibrin; and this network of fibrinous deposit may occasion lobular pneumonia. I ought, however, to remark, with Virchow, that these forms of embolus are observed chiefly in the capillaries of the liver, spleen, and kidneys, and consequently originate in structural changes of the ventricles of the heart, and in the large arteries. This fact has been established by the publications of Virchow, and of MM. Charcot and Lancereaux on ulcerous endocarditis.†

Let us now consider together, what becomes of the migratory clots, and what changes they undergo when arrested in the pulmonary artery.

If they are of large size, like those observed in the cases described by M. Briquet and M. Velpeau (in which the immediate result was rapid death), they do not undergo any modification of texture. They only recall, by their form and aspect, the vein from which they came.

Though the migratory clots are less considerable, they may reach branches of the second, third, fourth, and even fifth order of the pulmonary artery, remain there for a certain time, and acquire from their new relations a condition of existence and new modifications.

Though Virchow and Cohn‡ were the first to study these modifications, I have pleasure in here mentioning the published researches of MM. Charcot, Lancereaux, and B. Ball on this subject. They have all asked the same questions. What, say they, becomes of these clots? What changes do they undergo in the pulmonary artery? What is their action on the pulmonary artery, and parenchymæ of the lungs?

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* SEDILLOT: De l'Infection Purulente, ou Pyohémie, p. 476. Paris, 1849.

† Comptes Rendus des Séances et Mémoires de la Société de Biologie, 1862.

‡ COHN: Klinik der Embolichen Gefässkrankheiten. Berlin, 1860.

The changes which the clots may undergo in the pulmonary artery are of infinite variety, and may infinitely vary with each case. One may, nevertheless, describe in general terms their relations to the artery and their ultimate modifications.

Emboli vary in size according to the situation in which they have originated; and they are arrested in the pulmonary artery, either on one of its spurs, or in its divisions of the second, third, and fourth order.

The modifications which emboli undergo in the pulmonary artery are proportionate to the period they have been there. Generally adherent by a greater or less extent of surface to the interior of the vessel, they are continuous with the fibrinous or cruoric clots: they are often covered, to a greater or less extent, by newly formed clots; but they can be easily recognized by the modifications which they have previously undergone; thus, their central position is softened, and is composed of a pseudo-purulent yellowish-red substance, containing amorphous fibrin and the rudiments of connective tissue, while their extremities are rounded or jagged.

This form and structure, identical with the form and structure of the clots of the peripheric venous system, enable us to recognize their origin. When once the clots are definitively arrested in the pulmonary artery, they there undergo new modifications. The fewer their number, the greater is the chance of their softening. Should they, from their size, become fixed in divisions of the second, third, and fourth order, they may be detected, after a sojourn of longer or shorter duration, either to have undergone no important change of structure, or to have formed adhesions with the pulmonary artery. At other times, like a real foreign body, they determine morbid action, the result of which, according to Cohn and Lancereaux, is the production of a membrane which will form a cup, and then an envelope for the migratory clot, "in such a manner," says M. Lancereaux, "that after no long time, the arterial coagulum is completely surrounded by a perfectly organized membrane." I confess that I have not been so fortunate as M. Lancereaux, having never found capillaries in the clot nor in its membranous sheath; nor have I ever met with that new membrane to which Lancereaux attributes special properties in relation to the absorption of the embolic clot. I must here, however, recall to your recollection the fact that Virchow, in his memoir on embolism, after describing the connective organization of the pulmonary clot, has demonstrated the existence of a membranous sheath around triangular fragments of caoutchouc introduced into the branches of the pulmonary artery. He recognized the existence of capillary vessels in this membrane; and remarked, that the maximum of vascularity corresponded to each of the angles by which the caoutchouc came in contact with the serous coat of the vessel: he likewise pointed out, that there was an unusual development of the vasa vasorum at these points. Virchow also says that he has observed the formation of vessels at the extremities of the clots, when, apparently, there was no continuity between those vessels and the vasa vasorum, a circumstance which leads us to infer that a vascularizing power resides in the substance of the clots. If it be so, the organization of a thrombus or an embolus may be independent of the vascular walls.

We see, then, that an embolus may become organized. What afterwards becomes of it? Probably, it undergoes connective, cellular, pseudo-membranous transformation; and, after having been rarefied, may disappear, in the same way that the new membranes of pleurisy disappear. Probably, the complete disappearance of the clot is usually due to absorption, and this absorption can only be effected by the new vessels continuous with the vasa vasorum. Here, I ought to state, that progressive disappearance of the clot is often observed during life; and that at the autopsy performed at an epoch very near to that at which the obliteration was noticed, it has happened that no trace of the obliterative clot could be found, no abnormal development of vasa vasorum, and no change in the serous coat of the vein. There is, consequently, reason to conclude, that the disintegrated fibrin had returned directly into the circulation.

We have still to study the possible action of the embolus on the pulmonary artery and on the parenchyma of the lungs. I have already spoken at some length regarding the dyspnœa and extreme anxiety of the patients; and I have stated that rapid death, from syncope, occurs in some cases. The first effects produced by the embolus arrested in the infundibulum, in the trunk of the pulmonary artery, or in one of its principal divisions, are embarrassment of the cardiac circulation, and temporary or permanent arrest of the contractions of the heart. In these cases, cardiac syncope is the peril to be dreaded: but though sudden death, or to speak more correctly, rapid death has been observed several times, it has generally happened that the dyspnœa diminished some seconds or some minutes after the clot reached the pulmonary artery. In such cases, the heart, which had not ceased to beat, gradually regains its regularity and power: it is the lungs which undergo secondary changes in function and structure.

The organic matter, and the fibrin of the emboli, may have a threefold action,—an *obstructive* mechanical action, an *irritant* action as a foreign body, and a *special* action (perhaps putrid), which alters the tissues with which it comes in contact.

It appears from the researches of Virchow, Cohn, Charcot, B. Ball, and Lancereaux, that the most usual lesion is œdema of the lung, the direct consequence of the mechanical obstruction; then, next in frequency, comes hypostatic pneumonia, the result of irritant action; sometimes, though seldom, apoplectic extravasated clots; and still more rarely gangrene of the lung.

It is important to bear in mind, that the special nature of the obliterating body may exert an equally special action on the lung: this fact is established by the experiments of Virchow, and by post-mortem examinations, such as some of those which I have described to you. Such cases, however, are of rare occurrence: Dr. B. Ball only records two which can be placed alongside of my case. I am inclined to think that cases of gangrene from pulmonary obstruction are most apt to occur when there has been morbid alteration of the humors, peripheric gangrenous affection, or putrid infection, whatever may have been the original cause. In such cases, the obliterating clot possesses special properties.

The details into which I have entered, in relation to pulmonary embolism, have led me far away from my starting-point,—phlegmasia alba dolens. But I could not pass over in silence so frequent a complication of phlegmasia alba dolens, a complication, the result of which, though only very recently explored, has furnished a great amount of instruction.

Let me now terminate my lectures on phlegmasia alba dolens, by stating certain conclusions which may be considered as a summary of my remarks on that subject.

Obstruction of a vein is the cause of phlegmasia alba dolens. This obstruction is generally the result of spontaneous coagulation of the fibrin of the blood, and sometimes, but much more seldom, of coagulative phlebitis. Spontaneous coagulation is specially met with in patients under cachectic influence and in lying-in women. The obliterative clots, contain a great quantity of thready molecular fibrin, red or white globules, fat, and hæmatin. These clots becoming disintegrated may disappear without leaving any traces of their past presence, and generally without causing appreciable phenomena. They may undergo, *in situ*, a process of cellular organization: the interior of the vessel may assume the appearance of a cavernous trabecular tissue, and the circulation may be carried on by these altered vessels, or the connective organization may become fibrous, may be accompanied by contraction of the walls of the vessel, which may be transformed into a fibrous or fibro-calcareous cord.

Venous clots have two extremities, the peripheric and the cardiac: the former ramifies in the origin of the veins; the latter often has a resemblance to a serpent's head, and is frequently cystic, in which case, the envelope of the cyst is composed of stratified fibrin, and in the interior of the pouch there is found softened, disintegrated fibrin, having the appearance of pus. The head of the clots is usually non-adherent, and is situated at the mouth of an afferent vein.

Supported by a more or less resistant pedicle, this extremity may be carried away by the circulatory torrent, arrested in one of the branches of the pulmonary artery, and give rise to phenomena of pulmonary embolism. The secondary phenomena vary with the general state of the patient, and with the extent, volume, and composition of the emboli.

Cardiac and pulmonary emboli may occasion sudden death. Sudden death is the immediate result of the arrival of the embolus in the heart, or in the trunk or principal branches of the pulmonary artery. The embolus may become fixed in the divisions of the pulmonary artery, and there undergo various changes. The clot may be absorbed at the place where it was disintegrated, or it may obliterate the canal of the vessel. Persistent obliteration will generally produce local anæmia, œdema, and pneumonia; less frequently, gangrene of the lung and pneumothorax.

LECTURE XCVI.

PERINEPHRIC ABSCESS.

Insidious Beginning and Slow Progress of Perinephric Inflammation.—Etiology of Perinephritis: Fatigue, Muscular Exertions, Contusions, Repeated Blows over the Kidney.—Renal Calculus.—Typhoid, Purulent and Puerperal Fevers.—Perinephritis causing Sympathetic Pain in the Bladder and Spermatic Cord.—Perinephric Abscess consecutive to Iliac Abscess, Typhlitis, and Hepatic Colic.—General Symptoms.—Local Symptoms.—Intra-abdominal Tumor in the Side.—Iliac Abscess.—Spontaneous Opening of the Abscess into the Lumbar Region, the Intestine, Bladder, Vagina, and (very rarely) into the Peritoneum.—Lumbar Fistulæ.—Relative Gravity of Perinephritic Abscesses.—Treatment: Opening by bistoury in the Iliac and Lumbar Regions.

Gentlemen: You may, for many months, assiduously follow an active clinical service, without having a single opportunity of observing a case of perinephric abscess. For this, there are two reasons: the first is, because the affection is relatively rare; and the second is, because the nature of these cases is liable to escape notice, attention being only directed to the concomitant and consecutive phenomena. The beginning of the disease is often as insidious as its progress is sometimes slow: for a considerable time, the local symptoms may be absent; and sometimes, the general symptoms are so predominant, that little attention is paid to the patient's complaint of pain in the side. When the pain cannot be attributed to lesion of the stomach, liver, lung, pleura, uterus, or annexes of the uterus, there is reason to suspect that the tissue which envelops the kidney is diseased, particularly when nothing morbid can be detected in the urine. In the beginning of the affection, the patients may present such lesions in the iliac fossæ and broad ligaments that there seems good ground for ascribing all the morbid phenomena to the lesions.

There is sometimes a very great difficulty in recognizing perinephric abscess; and this difficulty continues, till the local symptoms become so marked as to render it impossible not to suspect the cause of the malady. Analyses of the different cases which I am about to lay before you will prove that this is a real difficulty, and put you on your guard against chances of error.

A woman, 32 years of age, was the occupant of bed No. 2, St. Bernard Ward. For ten days, she had been complaining of pains in the kidneys, and of feverish symptoms. Every afternoon, from the day on which she began to experience these pains, she was seized with fever and shivering: the fever lasted several hours on each occasion, and prevented the woman from going to sleep till one or two o'clock in the morning. During the continuance of the fever, she had lancinating pains in the right side.

At the middle of June, that is to say, five days after her admission to the Hôtel-Dieu, the fever became continuous with paroxysms which returned every evening from four to five. The paroxysms frequently began with a great shivering fit, and sometimes there was slight shivering in the afternoon.

There was almost total want of appetite, great thirst, nausea, and vomiting. She was rapidly getting thinner, and there was prostration of strength without any stupor. There was neither diarrhœa nor pink lenticular spots. On several occasions, purgatives were prescribed. Three weeks after admission to the hospital, the patient had regained her appetite, and was decidedly improved. Twenty-eight days later, however—on the 10th July—having been exposed to cold, she had a return of the fever, accompanied by rigors, and it was stronger than ever: new and very acute pains were complained of in the abdomen, on the right side; while at the same time, there was flexion of the thigh on the pelvis. Soon afterwards, a very decided swelling declared itself in the lumbar region: the costo-iliac hollow was effaced.

On grasping the loins with both hands, and exerting slight pressure, one could distinctly feel that there was deepseated boggy distension in that region. Day by day, the local pain became more acute on pressure: it lancinated several times during the day; and in the afternoon, the patient had rigors followed by fever.

The fever was continuous, and the rigors recurred at intervals: the pain became more and more acute in the lumbar region, where it could hardly be doubted that pus was forming. Some days later, fluctuation having become evident, M. Jobert (de Lamballe) afforded an exit to the pus by making an incision, some centimetres in length, in the lumbar region. Before penetrating into the purulent collection, the bistoury cut through a thick layer of œdematous indurated tissues. Two small lumbar arteries were tied. The pus, which issued copiously from the incision, was greenish-white, and contained streaks of black blood. To stop the flow of blood from the wound, small strips of agaric were introduced between its lips. Before making the dressing, I introduced the index finger into the abscess, and found that the kidney was pushed forwards, and that the cavity of the abscess contained débris of cellular tissue adherent to the surface of the kidney.

Immediately after the operation the patient felt relief: she had three hours of sleep during the day. In the evening, the strips of agaric were removed to afford issue for the pus. The pus still contained small filiform clots. The lips of the wound had ceased to bleed. A pledget of lint was introduced into the abscess.

Next day, the patient was almost without fever: the pulse was rather frequent, but it was quite regular. The patient took a basin of broth with relish. Day by day, the febrile state decreased: the walls of the abscess closed on themselves, and the purulent discharge went on diminishing. The patient ate a ration of food, and made visible progress towards recovery. At last, the fever quite ceased: the appetite improved: a small quantity of purulent serosity, devoid of fetor, exuded from the incision. The distended boggy feel of the abscess no longer existed. Three weeks after the operation, the incision was cicatrized. Some months after leaving the hospital, the patient returned to see us: she was then quite well, and had regained a certain degree of plumpness. She could walk easily, and without being tired. From the time of her leaving the hospital she had not felt any pain in the lumbar region.

This case, gentlemen, may seem to you simplicity itself; and in now hearing my rapid sketch of it, you may possibly be surprised that after the complaints latterly made by the patient, the least hesitation should have remained as to the diagnosis of the disease. Those of you, however, who have had some clinical experience, and have listened attentively to the history I have laid before you, will have grasped all the importance of the case. For ten days this woman had been suffering from pains in the loins, and from a general feverish state: she had been having fever and rigors every afternoon, accompanied (during the febrile paroxysm) by shooting pains in the right side. Along with the fever, she had nausea and loss of appetite. Her strength soon failed so much that she applied for admission to the hospital. When I interrogated her for the first time, she mentioned, in addition to the circumstances which I have now described, that the jolting of the carriage which conveyed her to the hospital had occasioned pains in the abdomen, particularly in the hypogastrium. I found that she had been having every afternoon a febrile paroxysm and rigors.

Any one not taking into account the lancinating pain in the right side, along with the general symptoms, and the hypogastric pain, might have supposed that the case was an incipient attack of mild continued fever; that the hypogastric pains excited by the jolting of the carriage arose from congestion of the uterus and its appendages, a condition often observed at the beginning of fevers. This supposition seemed the more probable, that after some days of expectant treatment, the feverish state perceptibly diminished, the rigors and paroxysm of fever did not recur, and there was, moreover, a return of appetite.

But this was only a pause in the progress of the disease. After exposure to a chill, the patient had a renewed attack of fever, attended by pain in the right side: there soon appeared tumefaction of the right lumbar region, where boggy distension became manifest: the rigors recurred daily: there then supervened a difficulty, and subsequently an impossibility to stretch the thigh, which from that time became flexed on the pelvis. Henceforth, no diagnostic doubt remained: it was evident that there was an abscess in the renal region; and that the psoas muscle was involved in the inflammatory process. I do not insist upon the termination in this case: I shall afterwards have occasion to revert to the quantity and nature of the pus of perinephric abscesses, and to all the symptoms which follow their being opened. At present, I only wish to impress on you the fact that the beginning of a perinephric abscess is often insidious: attention is not always sufficiently directed to the lumbar pain, which is apt to be neglected in consequence of a greater pain existing in another part of the body, and the general symptoms being sufficient to divert observation from the local pain. I beg you also to remark, that in this case the formation of pus took place slowly, and, so to speak, at two periods.

In the first case, I was unable to discover the cause of the phlegmasia; and I have told you that the abscess was *primary*, to distinguish it from perinephric abscesses consequent upon a lesion of the genito-urinary organs, or a serious general derangement of the economy.

Before discussing the various causes of perinephric abscesses, I must rapidly sketch the anatomy of the region in which they are developed, and the relations of the kidneys to neighboring organs. The kidneys are situated on each side of the vertebral column, and surrounded by a large quantity of cellulo-adipose tissue. The fatty capsule of the kidney has relations posteriorly with the pillars of the diaphragm, and with the deep fold of transverse aponeurosis. It has, also, relations anteriorly with the ascending or descending colon. It is unnecessary to describe the connections of the kidneys with the liver or spleen. The fatty capsule is continuous with layers of cellular tissue, which are continuous with the cellular tissue of all the organs of the perinephric regions. But the continuity most important to note is that which exists between the perinephric cellular tissue of the iliac fossæ. The iliac aponeurosis, to which M. Cloquet has given the name of *fascia iliaca*, is usually only formed, in the two upper thirds of the iliac fossa, by loose cellular tissue which is rarely continuous with fibrous tissue. From this disposition of parts, it results, that the pus surrounding the kidney will find its way equally easily into the cellular tissue which forms the immediate covering of the pscas muscle, or into the subperitoneal or subaponeurotic cellular tissue. This continuity of the cellular tissue of the perinephric region with that of the iliac fossa is the anatomical explanation of the facility with which perinephric abscesses, following the iliac and crural vessels, open into the triangle of Scarpa, or at the trochanter minor, following the psoas muscle to its lower insertion.

The perinephric adipose tissue is continuous with the cellular tissue of the lumbar region beyond the quadratus lumborum, between the margins of the latissimus dorsi and obliquus externus abdominis; that is to say, in the situation where J. L. Petit and Jules Cloquet have observed lumbar herniæ, and where it has been recommended to make the incision in nephrotomy. The continuity of the cellular tissue shows us the course taken by the pus in cases of perinephric abscess, when it is poured out into the subcutaneous cellular tissue of the lumbar region, to be there either localized, or spread out in the dorsal and gluteal regions. The lower surface and the substance of the quadratus lumborum are traversed by the lumbar arteries and veins, vessels of sufficient size to be sources of mortal hemorrhages when several of them are divided by the surgeon's bistoury.

The relation of the perinephric cellular tissue to the iliac fossæ, true pelvis, colon, diaphragm, and psoas muscle, enable you to understand the peregrination of these abscesses.

I now resume their consideration:

Before Rayer* wrote, little attention had been paid to perinephric abscesses. His researches were followed by those of Parmentier,† Féron,‡ Lemoine,§ and Ch. Hallé,‖ who collected a large number of cases from the lectures and practice of their teachers, among whom I would specially name Demarquay, Vigla, Gueneau de Mussy, and Chassaignac.

Perinephric abscesses arise from various causes, some of which are of a complex nature. Wounds in the renal region may occasion the formation of abscesses around the kidneys: Baudens mentions a case of this kind. Contusions in the lumbar region are still more common causes of perinephric abscesses. M. Bergounhioux (of Clermont) and M. Bienfait (of Rheims) each report a case showing the effect of a direct contusion in producing these abscesses.

In M. Bergounhioux's case, the patient was a peasant, who in falling from a tree, received a severe contusion in the right lumbar region. Extensive ecchymoses in that region, and hæmaturia lasting for some days, were the immediate results. The patient soon experienced deepseated pain; and fever set in. The hæmaturia ceased; but the bruised region became swollen, there were frequently recurrent rigors, and ere long, fluctuation could be distinctly recognized. An excision made external to the sacro-lumbar mass, gave exit to a very considerable quantity of phlegmonous pus. In a few weeks, the patient, being quite cured, left the Hôpital Clermont-Ferrand.

In M. Bienfait's case, the patient was a nurse who fell down eight steps of the stair, striking the edge of a pail. She was severely contused; but it was not till two days after the accident that she took to bed with fever and vomiting. When M. Bienfait saw the patient, he observed that the decubitus was dorsal: the face was pale, anxious, and sickly looking: the pulse was small and quick. The hypochondrium and the right side were swollen, tense, and painful. The urine passed since the previous evening was observed to have deposited a small quantity of blood. For three weeks there was a continuance of fever, excitement, and night-delirium: there was also diarrhœa. At that time, the right side of the abdomen was much increased in size: there was great tumefaction of the lumbar region, and complete obliteration of the costo-iliac hollow; these parts were œdematous. The intra-abdominal tumor was so large as to be compared by M. Bienfait to the uterus at the sixth month of gestation: it was situated in the side, stretching into the hypochondrium. It had every indication of having an alcoholic character at the inferior surface of the liver, which organ it pushed upwards and forwards: it extended to the left beyond the umbilicus, and downwards it reached to the upper part of the iliac fossa; and it transmitted to the flat hand, when placed over the loins, the impulse communicated to its anterior portion. Very obscure fluctuation was detected. Caustic potash was applied over the opening in the aponeurosis through which lumbar hernia takes place. Three weeks elapsed without there being any discharge of pus, the constitutional symptoms continuing as before. M. Bienfait made a puncture through the slough, when only a very small quantity of pus flowed out; but in a forty-eight hours afterwards the discharge became very profuse, and the tumor collapsed. The case terminated favorably. I may mention that in this case, paralysis of the right leg existed for four or five days when the tumor was at its maximum.

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* Rayer: Traité des Maladies des Reins. Paris, 1839.

† Parmentier: Union Médicale, vol. xv, année, 1862.

‡ Féron: Thèse sur la Perinéphrite Primitive. Paris, 1860.

§ Lemoine: Union Médicale, 20 Juin, 1863: t. xviii, p. 551.

‖ Halle: Des Phlegmons Perinéphrétiques, Thèse soutenue le 13 Août, 1863.

There are other cases in which no direct contusion, no blow on the loins has been received—cases in which violent exercise, such as a long journey on horseback, or the jolting of a badly hung carriage, have been sufficient to cause the formation of abscesses round the kidneys. This etiology can hardly be explained, except by admitting that the shocks imparted to the kidney by the trot of the horse, or the jolting of the carriage, have irritated the perinephric cellular tissue; but Dr. Hallé, while he admits that these causes in part determine the formation of the abscesses, correctly remarks, that at the same time the patients have been exposed to cold when in a state of perspiration. The majority of authors concur in recognizing a chill as a cause which may at any moment act as the determining cause of perinephric abscess.

Violent strains seem sometimes to occasion perinephric abscesses. For the history of a case of this kind, we are indebted to Professor Tardieu and his interne, Dr. Aug. Ollivier. A workman employed in the plaster quarries, when lifting a heavy load, felt acute pain in the left lumbar region. The pain having diminished, he continued to work; but twelve days later, was obliged to take to his bed, and seek admission in the Hôpital Lariboisière. At that period, there was an even tumefaction of the lumbar region, unaccompanied by redness of the skin: the tumefaction was greatest on the left side: the swollen parts seemed œdematous. There were severe lancinating spontaneous pains shooting through the chest and abdomen. These pains made the respiratory movements painful, and occasioned very acute colics. Slight pressure made posteriorly hardly increased the pain, whilst it was aggravated by hard pressure. Fluctuation, though carefully sought for, could not be detected. There was no albumen in the urine. The skin was burning, the pulse was 110, and the thirst very great: there was loss of appetite, constipation, and no vomiting. Six days after admission to the hospital, this patient had fluctuation in the left lumbar region: from a deep incision made into the abscess, there flowed about a tumbler of greenish, creamy pus, in which were seen some sanguinolent striæ, but no muscular débris. The opening of the abscess gave almost immediate relief from pain. By introducing a probe into the wound, the exact seat of the abscess was ascertained to be behind the left kidney, neither extending above nor below the organ. It was necessary, some days later, to enlarge the incision, on account of a dread of purulent absorption from there not being a very free exit for the pus: gradually, the source of the discharge dried up; and within six weeks after admission to the hospital, the man had completely recovered. Gentlemen, it is important to remark, that in this case of primary perinephritis, the strain was enough—without any lesion of the kidney—to have produced the local inflammation: the patient was a man of good constitution, who never had had any serious illness, nor any urinary affection.

I have the details of another case similar to that now described, in which the influence of a strain in producing perinephric abscess is clearly established. The son of one of our most celebrated painters, a youth of twenty, felt acute pain in the loins, when straining to haul up a boat upon the bank of a river. The pain soon abated: but in some days became so acute that the young man had to take to his bed. The physicians and surgeons consulted were all of opinion that there existed perinephritis, which would be likely to terminate in suppuration.

I am indebted to the ordinary medical attendant, Dr. Bonin (of Poissy), for important information regarding the termination of this case; and as it supports a doctrine in general pathology to which I have often called the attention of my auditors, I ought not to neglect to lay it before you. I was informed that the perinephritis did not, in this case, terminate in suppuration; and that diminution of the pain, the predominating morbid phenomenon, sufficed to dispel all the other symptoms. The acute character of the pain caused the patient to utter piercing cries: in these circumstances, some drops of the solution of the neutral sulphate of atropine, injected into the cellular tissue of the lumbar region, promptly relieved the pain, whereupon the patient was cured as if by enchantment. Does that mean, gentlemen, that the diagnosis was a mistake, and that perinephritis never existed? That is not my opinion. The surgeons who had been called in, being experienced in such affections, could not have been mistaken: the beginning, the progress, and the cause of the affection presented characters too distinctive to admit of any doubt on the subject. The pain having been put an end to, all the other symptoms yielded, and the progress of the inflammation was arrested. You know, gentlemen, the part which pain has in the inflammatory fluxion: how often have I demonstrated this to you at the bedside of the patient, particularly in cases of suborbital neuralgia. Have you not seen that when the pain ceased, all the other morbid phenomena disappeared in a few hours? It is very probable, then, that in the young patient whose case I have just now narrated, the cessation of all the symptoms of perinephritis is attributable to the cessation of the pain. You will, I think, be the more prepared to accept this interpretation of the course of the disease from my having previously pointed out to you that there is sometimes a spontaneous arrest, transient or permanent, of the symptoms of perinephritis.

It appears, then, that a strain may cause perinephritis: the two cases which I have just described, will enable you under similar circumstances, to foresee, from the commencement, the symptoms to be dreaded. When your attention is once directed to the existence of a deepseated abscess, you ought carefully to seek, day by day, for fluctuation, and all the signs, local and general, of the formation of pus. You will thus be able to seize the opportune moment for opening the abscess, and preventing the pus from working its way into the iliac fossæ, and setting up very formidable symptoms.

I am now going to lay before you, gentlemen, several cases in which the formation of the abscess was consequent upon a very slight cause; but that is not so always, and sometimes the existence of perinephric abscesses are not discovered till long after their beginning. MM. Cusco and Chassaignac have seen abscesses supervene several months, and even several years, after the action of the probable cause. In the cases to which I refer, the patients had received serious contusions in the lumbar region: the pain had disappeared: and it was not till a much later date, that, under the influence of a chill, or without any appreciable determining cause, the abscess showed itself. I am inclined to think—no new contusion having been received—that fatigue, a strain, or a chill, aroused irritation, till then latent. In such cases, there are two periods in the formation of the abscess. There takes place, in the first period, as the result of the contusion, a slow, latent modification of the perinephric cellulo-adipose tissue; and in the second period, we have the determining cause in operation—the fatigue, strain or chill—when pus is formed, and the local and general symptoms attendant on suppuration show themselves.

Perinephric abscesses very often have as their starting-point nephritis or calculous pyelo-nephritis. In such cases, the inflammation may extend, by çontiguity, from the kidney to the surrounding cellulo-adipose tissue: at other times, calculi impacted in the calices, the pelvis of the kidney, or the ureters, produce inflammation and ulceration of the different parts of the urinary apparatus, and when fistula is formed, they give rise to urinary abscesses which ought to be opened with the least possible delay. In these cases, the abscess is generally preceded by nephritic colics, and disturbance of the urinary function: it is not unusual to discover, on exploration with a probe, the presence of calculi in the abscess itself: at other times, the calculi remain imprisoned in the kidney, and it is not till after some time has elapsed, that the calculi present themselves at a fistulous opening of the abscess. Some have recommended search for the calculi. Dr. Miguel has even applied lithotrity to imprisoned calculi of too large a size to traverse easily the urinary fistula. At the present date, however, surgery takes a less active part in the extraction of renal calculi: the present practice is to wait till they present themselves at the mouth of the abscess.

Perinephritis is also, you see, a symptomatic affection, due to the presence of foreign bodies tending towards elimination: in some rare cases, the foreign bodies are hydatids situated in the perinephric cellulo-adipose tissue, in which they cause suppuration. Cases of this kind have been observed by Dr. Rayer, and Professor Denonvilliers, in patients in whom (as clinical examination had not given any reason to suspect hydatids in the liver, lungs, pleuræ, or other parts of the body), it was impossible to diagnose their presence in the perinephric region.

As the purulent diathesis may affect any part of the organism, it is very natural to suppose, that it may cause abscesses to form in the cellulo-adipose capsule of the kidney. In the Edinburgh Medical and Surgical Journal there is reported a case of perinephric abscess in a sailor, suffering from the affection popularly known at Plymouth as the "disease of the docks." This disease, according to Butler, is a fever, which may lead to purulent formations in different parts of the cellular tissue. Dr. Duplay reports a case of perinephric abscess, consecutive to an attack of typhoid fever, treated in Dr. Pelletan's wards in the Hôpital de la Charité. Dr. Desruelles and Destouches discovered a perinephric abscess in a woman of sixty, convalescent from gangrenous pneumonia.

In these cases, there is probably a special alteration of the humors leading to the formation of numerous abscesses, as is so frequently observed after small-pox.

Again, the *puerperal state* predisposes to the formation of pus, even when no puerperal epidemic is prevailing. As you know; mammary and iliac abscesses are of very frequent occurrence during the first months after delivery. You also recollect, how often I have called your attention to the pain which recently delivered women experience in the lumbar regions: this pain, and the pain in the uterine appendages, is not felt by patients unless pressure be made over the loins: you saw two cases of this kind in St. Bernard Ward; and in them, after some days, I discovered perinephric abscess. Bear in mind, that these abscesses around the kidney are not always the consequences of propagation of an abscess situated in the broad ligament or the iliac fossæ. The first of the two patients to whom I have now referred, who occupied bed 25 *bis*, St. Bernard Ward, had a chill on the fourth day after delivery, the symptoms being rigors, fever, and pains in the abdomen. All these symptoms subsided under the influence of rest: fifteen days later, this woman, having committed imprudences, was seized with shivering and fever, and a renewal of abdominal pain in the sub-umbilical region. The pain extended to the left iliac fossa; and some days later, by digital examination, I was able to detect an abscess of the broad ligament, which might open any day into the vagina, and so completely disappear. But when my attention was occupied in following the progress of the abscess in the broad ligament, the patient was seized with pain in the right side: pressure in the lumbar region increased the pain: in the renal region, there existed a decided doughy fulness, most perceived when the region was grasped between the two hands. The liver was not painful on percussion, and did not extend beyond the false ribs: the right iliac region was not swollen, nor was it painful on palpation. It appeared, then, that the lesion was limited to the right renal region; but, gradually, the pain and fulness diminished, the fever moderated, and there was no recurrence of the rigors: ere long, by palpation and percussion, I ascertained that the inflammation was undergoing resolution. Two months after admission to the hospital, this patient left it perfectly restored in health.

This termination of perinephric abscess by resolution is unusual; and the case of the patient, bed 4 St. Bernard Ward, is one proof more that suppuration is the rule. Though this woman had a natural labor, the uterus remained painful and very bulky: the uterine appendages were much inflamed, and the pus formed in the right broad ligament had two outlets, one into the bladder, and the other into the vagina. For fifteen days after the spontaneous opening of the abscess, everything went on to a wish; but at the end of that period, the patient was seized with rigors, fever, and pain in the right side. The liver was bulky, and descended within three fingerbreadths of the crest of the ilium. The mobility of the liver was ascertained by making the patient respire. In the lateral parts of the abdomen, and in the loins, there was felt an engorgement, which could only be referred to the renal region. The swelling occupied the whole of the costo-iliac hollow. By placing one hand behind, and another in front of the tumor, the movements of the liver during inspiration and expiration could be felt: and at the same time, a tumor was perceived which did not move during inspiration. There was no reason to suppose that this tumor was stercoral: no sign of occlusion of the intestine existed: the iliac fossa was free, and was not the seat of pain. The initiatory rigors, fever and lumbar pain suggested the probable existence of a perinephric abscess. As the fever was still going on, it seemed best to wait: by and by, detecting fluctuation, I opened the abscess external to the sacro-lumbar muscular mass: as soon as I reached the deep layer of muscles, I laid aside the bistoury for a hollow sound, with which I tore through the tissues: forthwith, there came a gush of non-fetid pus from the aperture I had made. On introducing the finger into the abscess, I could feel the kidney. Poultices were applied to the loins, whereupon the flow of pus was easy and abundant. With the view of drying up the abscess, and modifying the action of the suppurating surfaces, I several times employed injections containing iodine: little by little, the flow of pus diminished, and the incision began to cicatrize, when the patient was again seized with rigors, and pains in the right iliac fossa, thigh, and knee. Soon afterwards, an abscess was detected in the iliac fossa. Finding that this abscess had a tendency to pass below the crural arch, I begged Professor Jobert (of Lamballe) to open it. He made an incision one finger's breadth above the arch, and in a line parallel to it. A great quantity of greenish pus issued from the opening: the cavity of the abscess was washed out with water containing tincture of iodine: the pain of the psoïtis continued for a long time, the inferior extremity, nevertheless, resuming its normal position. There was reason to hope for a permanent amelioration, when the patient was seized with diarrhœa, and then with hectic fever: but she died some weeks after the opening of the iliac abscess. It was impossible for me to obtain a post-mortem examination of the body. This is much to be regretted; for we could then have ascertained whether the perinephric abscess (as is exceedingly probable) had worked its way to the iliac fossa. You can understand how the pus collected round the kidney, by following the psoas muscle, might reach the cellular tissue of the iliac fossa below the aponeurosis.

At other times, the pus may filter through the layers of aponeurosis, invading the cellular tissue which lines the peritoneum of the renal and iliac regions: in these cases, the abscess is not in immediate contact with the psoas muscle. Anatomy fully explains the infra-aponeurotic and supra-aponeurotic position of abscesses in this region. It must, however, be remarked, that the numerous aponeurotic openings allow the pus situated below the iliac aponeurosis to invade the subperitoneal cellular tissue.

Before leaving this part of my subject, I must remark, that in those cases in which perinephric abscesses have been complicated with pleurisy or pleuropneumonia, the thoracic affection and the perinephric abscess have always been on the same side. It was so in the cases observed by Desruelles, Cazalis, Demarquay, and Bernutz: in these cases, the pleurisy and pleuropneumonia evidently depended on vicinity.

There are other causes, besides those I have mentioned, which lead to the formation of abscesses around the kidney. Pain, as you are aware, which is most commonly the consequence of inflammation, may likewise be the cause of an inflammatory fluxion. Paludal suborbital neuralgia is often accompanied by hyperæmia of the conjunctiva and a profuse secretion of tears. If the orbital congestion continue for some time, a copious secretion of mucus is induced from the palpebral glands; and should the congestion last still longer, a certain amount of mucopurulent secretion will be seen at the angle of the affected eye. The pain may, under such circumstances, although there exist no primary local lesion, occasion inflammation, which will disappear as soon as the paludal poisoning of the system has been modified by general specific treatment. It is very evident then, that in such cases pain may induce inflammation of the eye. Toothache, also, often determines a fluxion to neighboring tissues. Some neuralgic affections of the cervix uteri, not attributable to any organic lesion of the uterus, determine, during each paroxysm of pain, a secretion from the follicles of the cervix, and chronic inflammation of the mucous membrane of the cervix. We have seen how paludal poisoning, a general cause, may induce inflammation, and have also seen how neuralgia, originating in a local cause, may lead to a similar result. You will find other cases recorded, in which pain has induced inflammation at a distance from the seat of pain. Dupuytren mentioned in his clinical lectures, that having included one of the branches of the brachial plexus in ligature of the axillary artery, he found, on making the post-mortem examination of the patient, that there was an abscess of the brain; and he was inclined, in that particular case, to think that the constant acute pain endured by the patient during the continuance of arterial ligature, was the determining cause of the cerebral abscess. To me, however, this explanation seems rather hypothetical.

Pain may similarly explain the formation of some perinephric abscesses. After attacks of severe nephritic colic, it is not unusual to meet with perinephric abscesses, the opening of which shows that the formation of pus could not have been the consequence of a urinary fistula; while the examination of the urine has proved the non-existence of purulent nephritis. In these cases, therefore, there is no ground for supposing that inflammation had been propagated from the kidney to the surrounding cellular tissue, and we certainly must in these cases attribute to pain a large share in the formation of these abscesses. Again, acute pain of the bladder may induce the formation of abscesses around the kidney. At the close of the year

1862, I was called in, in consultation, by Dr. MacCarthy, my very honorable colleague, to visit a lady who had complained for more than two years of a very inconvenient irritability of the bladder. An irresistible desire to urinate was excited by the presence of even a few drops of urine in the bladder. On October 4th, 1862, this lady wore, for a few hours, a very tight dress. On the following day she took to her bed, and complained of acute pain in her right side; the pain extended into the lumbar region. She had had no stool for three days. She had no fever. On October 8th, the menses appeared, and continued for twenty-four hours. The pain in the right side continued. For eight days the local pain went on increasing. She had rigors and fever, increasing in severity day by day. No relief was obtained from poulticing, and two applications of leeches. Every day, care was taken that the bowels should be opened by the use of an injection, or aperient pills; but notwithstanding there was no abatement of the pain and fever. On October 15th, for the first time, my honorable colleague, when making pressure on the seat of pain, detected a hard round tumor. The pulse was rapid, the skin hot; and the shivering constantly recurrent. It was at this period that Dr. MacCarthy consulted me in the case. We were both satisfied that there was a lumbar tumor: by palpation, I ascertained that the movements of the liver during respiration were independent of the tumor, which remained immovable. The tumor was large, and very painful. Dr. MacCarthy and I took exactly the same view of the case, and were of opinion that the patient had a perinephric abscess. Although the patient had had hepatitis in Bombay, there was no ground for supposing that she had a tumor of the liver. That she had perityphlitis, was an equally untenable hypothesis, because that is an inflammatory affection generally seated in the iliac fossa; moreover, there was no pain in the large intestine, and the stools presented no important alteration. In consideration of the fever and the size of the abscess, our prognosis was given under reserve; but we held that the patient had perinephritis, independent of any lesion of the kidney, and depending, probably, on sympathy with a constantly irritable bladder. The immediate cause of the affection might have been a chill, or excessive compression of the lumbar region by a too tight corset or gown.

The tumor rapidly increased in size, the rigors recurred, there was complete loss of appetite, and a twofold severity of the fever. Under these circumstances, Dr. MacCarthy, considering the time had arrived for opening the abscess, again called me in to consult on the case, and, along with me, summoned my honorable hospital colleague, M. Alphonse Guérin. When we met, the tumor occupied the whole of the right side, extending to the umbilicus. The fluctuation was very obscure; and the slightest pressure occasioned extreme pain. There was well-marked œdema in the right lumbar region. Hesitation as to the course to pursue was imposible: it was evidently necessary, without delay, to afford an outlet to the pus, so that the abscess might not have time to open into the intestine, or work its way into the iliac fossa. M. Alphonse Guérin made an incision ten centimetres in length near the external margin of the sacro-lumbalis muscle. Having reached a depth of four centimetres, he laid aside the bistoury, and with the index finger of the right hand penetrated the abscess, whence issued a torrent of fetid thick pus. For four following days the patient had a continuance of slight fever with shivering. So that there might be no doubt as to the pus, which was still somewhat fetid, having a free outlet, the opening into the abscess was enlarged by the finger. The discharge gradually decreased, and lost its odor. Eight days after the opening of the abscess there was a notable amendment in all the symptoms: the suppuration was inconsiderable, the wound was cicatrizing, and the appetite was restored. No check occurred in the progress towards recovery. On the eighteenth day after the operation, the wound was perfectly closed. At that date, there was no trace of the tumor; and some days later, the cure may be said to have been complete.

Is it necessary, gentlemen, that I should expatiate upon the leading symptoms in this case? Habitual irritability of bladder, persistent and increasing pain in the lumbar region, then fever, soon afterwards pain in the renal region, and finally, a tumor and abscess, were the phenomena.

In conjunction with this case, let me call your attention to another, also bearing testimony to the influence of pain and irritability of the bladder in producing perinephric abscesses. In the latter months of 1863, a great personage was operated on for vesical calculus by Civiale. Lithotrity was successfully performed, and the patient was able to leave Paris to recruit his strength in the country. But some days after the operation, the illustrious patient began to feel pain in the renal region, in one side only. Fever, rigors, and loss of appetite supervened. Several surgeons were assembled in consultation: after ascertaining that there was no symptom of lesion of the urethra, bladder, or kidney, they were disposed to attribute the lumbar pain to ilio-lumbar neuralgia consequent upon the lithotriptic manipulations. The pain continued for several weeks in the renal region, and extended into the iliac fossa of the same side; but in the latter situation, there was neither tumor nor any sign of psoïtis. The fever and rigors, nevertheless, continued, and the patient went on losing strength. M. Nélaton was now asked to join the other surgeons in consultation. Informed of all the symptoms which I have now related to you, and of the conditions under which they showed themselves, the learned professor very attentively explored the renal region. Palpation occasioned pain; and there was a slight fulness in the lumbar region, but (as the matter was still very deepseated) fluctuation could not be perceived. M. Nélaton had no hesitation in stating that there was a perinephric abscess. A large incision was made at the margin of the quadratus lumborum muscle, and immediately there was a gush of healthy pus without special odor, and not containing any clots of blood. By introducing the finger deep into the wound, it was ascertained that the abscess was situated in the circumrenal cellulo-adipose tissue. From the day of the operation both the fever and pain disappeared. There was no recurrence of the rigors: day by day the appetite returned: and at the present date recovery may be said to be complete, although for some weeks the patient had had a rather obstinate diarrhœa. There was neither renal nor vesical lesion. It is, therefore, very probable, that the abscess was caused by the irritation of the bladder, acting by sympathy upon the perinephric cellulo-adipose tissue.

Let me now quote from Chopart's treatise a case which I consider very interesting, inasmuch as it is one more fact to prove the part which pain has in producing perinephric abscess:

"I saw a man," says Chopart, "who had had his right testicle amputated for cancer. All went on favorably till the thirty-second day after the operation, when he had considerable rigors, and complained (for the first time) of heat and lancinating pain in the kidneys. The wound—the cicatrization of which was complete—became pale and dry. The fever continued. Next day the abdomen was tense. During the night, the patient had nausea and restless excitement. On the following day he died. I was present at the post-mortem examination. There was an abscess in the adipose tissue of the left kidney; the pus was serous and fetid; the cellular tissue of the spermatic vessels was infiltrated by the same matter;

and there were also two small abscesses in the pelvis, on the same side. As the whole of the spermatic cord had been included in the ligature, in place of only the spermatic artery, it was supposed that the ligature might have occasioned the suppuration by the irritation it had excited in the cellular tissue of the pelvis and loins of that side, and of which irritation the patient had given signs at the moment the ligature was tightened, for he then complained of an acute pain in the region of the left kidney which continued for several hours. All other parts of the body were in a healthy state."

It would certainly be difficult to find in the annals of science a case in which the influence of pain was more distinctly marked. There was not the slightest grounds for supposing that there was either phlebitis or purulent infection; for, on the one hand, the pus found in the course of the cord was not in the veins, and on the other hand, the patient never had had a metastatic abscess. It appears to me that in this case pain caused irritation of the cord and was re-echoed, if I may so speak, by the cellular tissue of the kidney, just as gonorrhœal irritation of the urethra may make itself known in the joints, and produce blenorrhagic arthritis. It is, therefore, most important to remember that there is a special irritation in blenorrhagia which may cause blenorrhagic arthritis; and also that irritation of the bladder or spermatic cord may act sympathetically upon the perinephric cellulo-adipose tissue.

I have now to tell you that there are cases in which the perinephritis cannot be attributed to any of the causes we have been considering. In the patient who occupied bed 2, St. Bernard Ward, the cause of the perinephritis could not be ascertained. The same impossibility to discover the cause of the affection existed in a case to which I was called in consultation with Dr. Cavasse in October, 1861, and of which the following is a summary of the details: M. X., a man aged thirty-five, since an attack of typhoid fever, had been in rather feebler general health than previously, but was able, nevertheless, actively to attend to his business. On his return one day from hunting, when he had fatigued himself by overwalking, he complained for the first time of pains in the left lumbar region. The affection, supposed to be lumbago, was attributed to damp weather. Every time the patient bent forwards, pain was felt. He had had neither fever nor rigors, and his appetite was unimpaired. He continued to go about his ordinary occupations. The pain, however, was constant. Eight days after his first visit, Dr. Cavasse made a renewed examination of the seat of pain, when, not without surprise, he found a tumor in the left lumbar region. In that situation there was slight redness of the skin and doughiness of the cellular tissue. On endeavoring to make out the limits of the tumor by the finger, a hardness of from seven to eight centimetres was discovered. The tumor was as large as a hen's egg, and was prominent under the skin. It was five or six centimetres from the vertebral column. Fifteen days later, the tumor became more prominent; both the redness and doughiness increased. The patient had lancinating pains; and by palpation deep, obscure fluctuation was detected. Observe, gentlemen, that up to this date there was neither fever nor loss of appetite. These were the circumstances in which Dr. Cavasse asked my advice. I made out all the signs of a deepseated abscess in the lumbar region. The patient's age, his usual state of health, the absence of any osseous lesion in the ribs, vertebral column, or pelvis, excluded the idea of there being any indolent abscess, the result of congestion. The pain in the region, the redness of the skin, and the fulness of the cellular tissue indicated the existence of acute inflammation; the deep seat of the abscess was the cause of the tardiness with which fluctuation became appreciable. What were the original seat and cause of the inflammation? There had been neither intra-abdominal pain, nephritic colic, nor any noteworthy alteration in the urine. The kidney, consequently, could not be the cause of the condition I have now described; the patient had not received a blow in that region. It was therefore very difficult to determine the cause of the abscess. But the pain at the first, its persistence in the renal region, and the swelling and heat in that situation, were sufficient indications that the evil had originated there. Though the kidney was not in any way the cause of the affection, and though no direct traumatic cause was in operation, there was every reason to believe that the perinephric cellulo-adipose tissue was the seat of the suppuration, and that there existed a primary perinephric abscess. That was my opinion when I first saw the patient with my honorable colleague, Dr. Cavasse. I, however, recommended my colleague to delay opening the abscess till the matter became more superficial. On an early day of November, that is to say, five or six weeks from the beginning of the affection, M. Cavasse opened the abscess; there flowed from the incision a tumbler of non-fetid pus, mixed with blood. The walls of the abscess were hard; and its cavity presented numerous anfractuosities. The opening was kept patent by the introduction of a dossil of charpie, so that there was an easy flow of pus. Injections containing tincture of iodine were employed with a view to modify the surface of the interior of the abscess. The cure was not completed till the middle of January, 1862.

The case is interesting for more reasons than one. First of all, it is interesting, because it proves that a primary perinephric abscess may exist without any other appreciable cause than excessive walking, or damp weather: and in the second place, it shows, that a deepseated perinephric abscess may be slowly developed without causing the constitutional symptoms which usually occur in similar circumstances.

Other causes, besides those which I have mentioned, give rise to perinephric abscesses.

In a previous lecture, I remarked, that pleurisy or hepatitis is a very frequent sequel of severe hepatic colic; and that cellular adhesions between the liver and diaphragm are very common in persons who have recently had severe hepatic colic. In the cases in which I pointed out to you that these colics were succeeded by pleurisy of the right side, I explained the phenomenon by telling you, that the peritoneal inflammation was communicated to the diaphragm, and from the diaphragm to the pleura. I also reminded you of a very usual anatomical disposition, a separation of the muscular fibres of the diaphragm, by which the peritoneum and diaphragmatic pleura are brought into contact, and are adherent, being separated only by a very thin layer of cellular tissue. It is easy to understand, that when such conditions exist, inflammation of the peritoneum may very easily be propagated to the pleura.

You can now understand how inflammation of the gall-bladder, so common in hepatic colic, may be propagated to the peritoneum covering the right kidney, and how that inflammation may be the starting-point of a perinephric abscess.

Perforation of the bladder by a calculus, which usually produces rapidly mortal general peritonitis, may (as in a case I described to you, which we had an opportunity of observing in St. Bernard Ward) determine adhesions between the bladder and neighboring parts; and then, the calculus escaping from its position, falls into adventitous cellular tissue, which will patiently support its presence till the abnormal contact excites inflammation of the perinephric cellular tissue. It was probably a case of this kind which I had an opportunity of observing in the practice of my colleague Dr. Millard. We saw together an old lady suffering from attacks of hepatic colic. After one of these attacks of unusual duration and severity, she had all the symptoms of acute hepatitis, accompanied by inflammation of the gall-bladder. There was very intense pain in the subhepatic region: there was fever and severe general disturbance of the system, when all at once the pain extended to the right renal region: a large tumor then made its appearance, accompanied by rigors, and soon afterwards, by undoubted signs of perinephric abscess. The contents were evacuated by an opening and counter-opening made by Dr. Trélat. The symptoms rapidly subsided after the operation.

We believed, that inflammation of the gall-gladder was the cause of adhesions forming between the cholo-cystic peritoneum, and the peritoneum which covers the intestines and extends above the kidney: that a calculus had escaped into the adventitious cellular tissue: and that the inflammation had been propagated to the perinephric tissue. This, of course, was only an hypothesis: but the statement is not hypothetical, that the hepatic colic and consecutive inflammation of the gall-bladder and peritoneum were the cause of an abscess forming in the circumrenal cellular tissue.

The symptoms of perinephric abscesses vary with the cause—according as they follow disease of the kidney, or supervene under the influence of some other cause. In the first instance, the symptoms of perinephritis are preceded by the peculiar pain of nephritic colic: there may have been calculous nephritis; and sometimes gravel and calculi are found in the urine. Hæmaturia may have existed in some cases; and should inflammation have invaded the calices and pelvis of the kidney, more or less pus will be deposited by the urine. Should pain, swelling, redness, and doughiness supervene in the lumbar region, it will be natural to ascribe the perinephritis to lesion of the kidney.

Generally, however, the perinephritis occurs irrespective of any renal lesion. Generally, quite suddenly, and as the result of very various causes, the patient complains of a deepseated, diffuse pain, which may be acute or dull, in the lumbar region. This spontaneously originating pain, which is sometimes shooting, is always increased on pressure, particularly when the seat of pain is pressed between the two hands. The pain is sometimes absent for weeks or months, not returning till a new determining cause arises. Generally, however, the pain is persistent, and goes on increasing till the pus is evacuated. The pain is always a very important symptom, because for several days, or weeks, no other local symptom may exist. The general disturbance of the system, however, shows that the pain depends on an organic cause: the patients have continued fever, with fits of shivering in the evening. Every day, the patients have rigors, followed by hot and sweating stages. They soon lose appetite, and rapidly become thin. Sometimes, the febrile paroxysm sets in with vomiting; and it is almost always attended by obstinate constipation.

Within a period, then, varying from eight to fifteen days, the patients present no symptoms except local pain, general debility, and quotidian ague. Afterwards, other local signs of deepseated inflammation show themselves: the pain becomes more and more decided on making pressure over the region, in which also there is more or less doughiness: the costo-iliac hollow is effaced; and if (the patient lying on his back) the physician press his hand deeply into the lumbar region, he perceives by the touch, as well as by the eye, that a more or less marked projection exists; and if, at the same time, he place the other hand upon the corresponding anterior region, he recognizes, between his hands, a deepseated tumor continuous with the subcutaneous cellular tissue. This tumor remains fixed during full inspiration and expiration, performed at the request of the physician—a fact which establishes with certainty, that the tumor is independent of the liver, which rises and falls with each inspiratory and expiratory movement. The doughiness of the lumbar region is often accompanied by œdema, and this œdema may extend to the dorsal region and hips. There is sometimes also, a little redness of the skin. This redness is erysipelatous in cases in which the abscess extends into the cellular tissue of the region. From the very commencement of the local signs of inflammation, fluctuation can be distinctly felt. As, however, it is very deepseated, it requires great aptitude on the part of the examiner to detect it with certainty: its existence may sometimes only be suspected from the complications of œdema, doughiness of the region, and from the general symptoms. As soon as the formation of pus commences, there is an exacerbation of the fever: the pulse acquires a certain degree of fulness, and becomes harder and more resistant, while, at the same time, the patient complains of frequent shiverings. Under these circumstances, the indication to evacuate the pus is quite clear: there must be no hesitation in opening the abscess, for if this be delayed, the pus may work its way into the iliac fossa and coxo-femoral articulation, thereby endangering the life of the patient.

Though, sometimes, the result of perinephric inflammation is the formation of a partially encysted abscess—the abscess remaining limited to the perinephric layer of fat, and showing no disposition to extension beyond the lumbar region—yet, at other times, the inflammation gains the cellular tissue of the neighboring parts, invading perhaps the subdiaphragmatic cellular tissue, occasionally even passing that barrier and reaching the pleura or lung, ending there in pleurisy or pneumonia. These are the terminations and complications which result from too long delaying surgical intervention. This has been observed by MM. Demarquay, Cusco, Cazalis, and Bernutz. The pus sometimes penetrates even into the bronchial tubes. A case is reported by Dr. Rayer, in which there was a vomica in the lung attributable to no other cause than a perinephric abscess.

The inflammation often invades the iliac fossa, the patients then complaining of pain in that region; and if an outlet be not afforded to the pus, a tumor is soon perceived projecting above Poupart's ligament, and passing below that ligament to show itself at the base of the triangle of Scarpa. In the latter case, the pus has followed the sheaths of the iliac and femoral vessels: at other times, it takes the course of the psoas muscle to the trochanter minor, and may, as we have seen, invade the coxo-femoral articulation.

I have already told you, gentlemen, that tke pelvic cellular tissue may be invaded by inflammation which had the renal region as its starting-point. In one of our patients in St. Agnes Ward you saw that the pus had worked its way from the renal region to the cavity of the pelvis, and had then been evacuated into the bladder and vagina. The same patient had had double perinephric abscess: the abscess on the right side had terminated in resolution. In cases in which the pus has worked its way from a distance, it causes great structural damage, and gives rise to very long-protracted suppurations, so that death is almost always the result of these migratory abscesses. The right practice, therefore, is to open perinephric abscesses as soon as the local and general signs of their existence are undoubted. You have seen, gentlemen, that the spontaneous evacuation of the pus by the vagina or bladder may be a favorable termination of the case; but that mode of evacuation does not always occur: when the spontaneous evacuation takes place by the colon, the result may be unfavorable, as was seen at the autopsy of a patient who was treated in Professor Cruveilhier's wards, the details of whose case have been reported by Dr. Parmentier.

I know of only one case in which there was probably a spontaneous opening of the perinephric abscess into the peritoneum: perhaps some other cases may have been recorded. The rarity of such cases will be at once explained, when you consider the relations of the adipose envelope of the kidney to neighboring organs and to the peritoneum. Perinephric abscess is generally situated behind the kidney: it is then separated from the peritoneum by the kidney; and the colon, in contact with the anterior surface of the kidney, also increases the distance between the inflamed cellular tissue and the peritoneum. Should the inflammation have a tendency to extend to the peritoneum, peritonitis will be induced, the result of which will be the formation of false membranes increasing the thickness of the peritoneum. The autopsies show that under these circumstances the pus burrows under the serous membrane, and does not perforate it.

Allow me to make some further remarks upon the progress of perinephric abscesses, before I discuss the differential diagnosis between them and other affections. I have generally observed that the abscess has a tendency to make its way to the lumbar region, the inflammation gaining the different tissues little by little, till at last it reaches the subcutaneous cellular tissue. But should there be delay in opening the abscess, it will dissect the subcutaneous cellular tissue, and extend to the hips.

In 1861, bed 8, St. Agnes Ward, was occupied by a man forty-four years of age, who, when admitted to the hospital, had an abscess occupying the dorsal and lumbar regions of the left side. The man had fever, and an absolute loss of appetite: an erysipelatous redness covered the whole of the region invaded by the abscess. The patient, who for six weeks had been suffering from lumbar pain, stated that he had been three months of the previous year under treatment for a similar affection in the Hôpital Saint-Antoine under the care of my lamented colleague the late Dr. Aran. In reply to repeated questioning, he affirmed that no incision had ever been made. He left the hospital without being cured, and for four months afterwards was unable to resume his work. He said that he did not know how the tumor had disappeared: he only knew that the improvement in his condition had occurred very gradually. He maintained that he never had had nephritic colic, and had never passed gravel or calculi with his urine. In the absence of positive facts as to his previous history, we had to rest satisfied by ascertaining the condition he was in when admitted to St. Agnes Ward. I have said that the pain had continued for six weeks: from the very first, there was fever; by degrees, an enormous phlegmonous tumor had formed, occupying the lumbar region, and subsequently extending to the dorsal region and hips. Palpation occasioned great pain; fluctuation could be detected in a salient point in the lumbar region. There was one point at which the tumor projected into the abdominal cavity, and extended in one direction from the liver to the iliac fossa, and in another to the umbilicus. The tumor was evidently a large collection of pus. By making an opening in the lumbar region, there was evacuated a large quantity of greenish-yellow horribly fetid pus, mingled with a fluid consisting of mixed pus and blood. About two litres of this fetid pus were collected; and for several days after the operation there was a great additional discharge of similar character seen on the poultices in which the region of the tumor was kept enveloped. Great relief followed the opening of the abscess: the fever abated: from the third day, the erysipelatous redness disappeared:

the walls of the abscess progressively and rapidly contracted. In the external iliac region, there was another abscess which opened spontaneously: it discharged pus of the same character as the lumbo-abdominal abscess. By cautiously introducing a probe into the second abscess, it was ascertained that there was no morbid state of the iliac bone. By degrees, all the affected parts became cleansed, the fever disappeared, the appetite returned; and, to the great satisfaction of everybody who had ascertained the extent of the affection, the patient was restored to perfect health in three weeks.

Lessons may be deduced from this narrative, notwithstanding its enormous gaps. What are they? We learn in the first place, that the abscess may extend exceedingly in the abdominal cavity without bursting the peritoneum, without reaching the iliac fossa, or without emptying itself into the large intestine. When by invading the tissues step by step, the abscess seeks to evacuate itself externally, it may detach these tissues, layer by layer, and so produce a subcutaneous or submuscular abscess, as happened in the case of our patient. That case also afforded an example of two perinephric abscesses occurring in the same person, and in the same side, at an interval of some months. This recurrence of the affection on the same side would indicate the continuance of some local cause, such as has been noted by observers. In our patient, however, we could not ascertain the existence of calculi in the kidney: moreover, the patient affirmed that he had never perceived any important change in the urine, and had never had nephritic colic.

It must be remembered, however, that calculi may remain a long time in the parenchyma of the kidney without occasioning acute pain. Pozzi, quoted by Rayer,* mentions the case of a man in whom the right kidney (as large as the head of a two years' old child, and weighing two pounds and a half), contained a calculus, the point of which had come through the renal parietes, occasioned gangrene, and led to the formation of a deep-seated abscess. The other kidney contained at least a hundred other calculi. "Sed quod mirum est," says Pozzi, "toto tempore vitæ nunquam conquestus est de doloribus nephreticis, calculis, urinis, sabulosis aut difficulter vel diminute fluentibus." Taking into consideration cases of this kind, we are justified in concluding, that our patient had renal calculi which gave rise to the perinephric abscesses.

I have sometimes seen a perinephric abscess occasion a very extensive lumbar phlegmon: it also, sometimes, happens that while these purulent collections detach the lumbar tissues from one another, emphysema of the entire dorsal region is produced. Twice I have seen this take place: the abscesses were opened; and exit was afforded to pus and fetid gas. In one of the cases, the abscess communicated with the intestine: the patient passed pus with the stools, and from the incision there was a discharge of yellow matter, which certainly came from the intestine. The relation of the colon to the perinephric abscesses explains these occurrences.

We now come, gentlemen, to consider the important question of the diagnosis of perinephric abscesses. There are three morbid conditions which may serve as a basis of diagnosis: I refer to pain, tumefaction of the lumbar region, and fever. At the beginning of the formation of perinephric abscesses, the only symptoms are pain in the loins and fever. When the pain is on the right side, and when, associated with it, there is continued fever, having a quotidian exacerbation, and prostration of strength, one may for a moment regard the case as one of typhoid fever: but the progress of the disease, and the absence of other symptoms peculiar to dothinenteritis, soon rectify such a mistake.

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* RAYER: Traité des Maladies des Reins, t. iii, p. 35.

Simple neuralgia does not generally cause fever: nor is fever induced by the pain of lumbago, which usually has its seat in the two sacro-lumbar muscular masses. On the contrary, the duration of the pain, its characteristics, and its being excited by pressure when there is perinephric inflammation, and also the continuance of the fever with its quotidian paroxysms, render it sometimes possible to diagnose the probable existence of perinephric abscesses, even in their first stage.

Nephritis and calculous pyelo-nephritis are generally preceded by nephritic colic: they may be accompanied by fever, and a saburral condition accompanied by vomiting and lumbar pains exasperated by pressure; but examination of the urine, which is frequently albuminous during the crises, and the immediate relief experienced on the calculus reaching the bladder, demonstrate, that the lesion is limited to the kidney and the excretory urinary organs. The diagnosis, however, will be more difficult in a case of pyelo-nephritis accompanied by a tumor in the lumbar region, if repeated examination of the urine disclose neither the permanent nor the temporary presence of more or less pus in the urine. There are cases, however, in which there is still greater difficulty in diagnosing pyelo-nephritis accompanied by a tumor: I refer to those cases in which, on examination after death, a calculus has been found impacted in the ureter obstructing the passage of pus into the bladder. In those cases, the examination of the urine gives a negative result: but it is necessary to remark, that the distension of the pelvis and calices of the kidney may cause perinephric abscess, in consequence of the inflammation being propagated to the surrounding cellulo-adipose tissue, or from its forming a blind fistula leading to the effusion of pus and urine into the fatty covering of the kidney after which soon appear all the signs of genuine perinephric abscess.

Let me now describe to you, gentlemen, a case of chronic perinephric abscess with pyelo-nephritis, which gives support to the remarks I have now made. For the history of this case, I am indebted to the courtesy of Dr. Demarquay.

The patient was a man, of about thirty years of age, previously subject to nephritic colic, who had passed small urinary calculi, and experienced pain in the right lumbar region for four or five years. When my honorable colleague saw him the first time, about the end of July, 1864, he ascertained the presence in the right hypochondrium of an enormous tumor, having for its boundaries the liver, the iliac fossa, and the linea alba. This tumor was specially prominent on the anterior abdominal wall; the costo-iliac hollow was effaced, but the lumbar region presented neither deformity nor œdema. The liver did not appear to be implicated: no pain had ever been experienced in the hepatic region: nor had there ever been jaundice. There was fluctuation in the tumor. The urine deposited a considerable quantity of purulent mucus; and attacks of nephritic colic were followed by excretion of urinary calculi. From all these facts, the surgeon was led to believe, that the fluctuating tumor was an abscess in the circumrenal tissues. Dr. Demarquay opened the abscess by applying caustic potash to the most prominent point of the tumor. For several days, a great quantity of pus flowed from the opening: then, by degrees, the tumor diminished in size, its walls contracted, and there was reason to hope for a speedy cure, when the patient, after certain imprudences on the occasion of the fêtes of 15th August, succumbed under symptoms of very acute peritonitis. At the autopsy, the tumor was found to be really situated around the kidney:

on the surface of the kidney, there were traces of chronic inflammation. The calices, pelvis of the kidney, and the urethra were full of pus; but it was impossible to discover any trace of urinary fistula: the pus could pass drop by drop into the bladder: no calculus could be detected in any part of the urinary apparatus. The walls of the abscess were constituted by all the organs in contact with the purulent collection, and by the peritoneal lining of the products of the inflammation. The adhesions between the walls of the abscess and the walls of the abdomen, which had been established by the action of the caustic, were unruptured. Peritonitis certainly existed: but it was impossible to discover any communication between the abscess and the peritoneal cavity.

The case now described is a beautiful example of chronic perinephric abscess, most probably consecutive to a pyelo-nephritis which itself had had calculous inflammation as its cause.

I do not think it necessary, gentlemen, to dwell at any length upon the differential diagnosis of perinephric abscess, hydronephrosis, and cancer of the kidney. In the two latter diseases, although there is a tumor situated in the lumbar and abdominal regions, the progress of both affections is essentially chronic, and neither are accompanied by fever. There is fluctuation in hydronephrosis, but the lumpy state of the kidney may be sometimes detected. Though pain be a symptom in cancer of the kidney, the hardness of the cancerous tumor, and the frequent hæmaturia, will enable you to avoid any error in diagnosis.

I have already told you how to distinguish tumors of the liver from tumors of the right kidney by palpation performed during very deep inspiration; the liver moves, moving with it the tumors seated in its parenchyma, while, during similar inspiratory movements, renal tumors remain immovable. Tumors of the spleen project in so marked a manner, that it is hardly possible, even when they are very large, to mistake them for lumbar tumors. Perityphlitis, fecal tumors of the large intestine, and fecal abscesses, can only, I think, occasion temporary mistakes: indeed, inflammation of the cæcum or appendix vermiformis, terminating in abscess of the iliac fossa or true pelvis, has so limited a seat, that error is impossible, unless it be in cases of latent perityphlitis, and in cases in which the inflammation, by propagating itself to the iliac fossa and lumbar region, slowly induces signs of perinephritis: in these cases, the abscess will not only yield pus having a stercoral smell, but will likewise emit a certain quantity of intestinal gases. Fecal tumors of the large intestine have their seat in the ascending or descending colon, or they are formed as the result of long atony of the bowel: by palpation, a certain degree of softness can generally be recognized disclosing their nature: moreover, as they are removed by purgatives, all diagnostic hesitation is thus terminated.

I do not require to dwell on the diagnosis of the complications of perinephric abscess: it will be sufficient to remind you, that these abscesses may terminate by extending from above downwards, or from below upwards: in the former case, if they burrow towards the iliac fossa and true pelvis, they may cause psoïtis, and may open spontaneously into the bladder or vagina: in the latter case, they may give rise to diaphragmitis, pleurisy, or pneumonia. In conclusion, I advise you, whenever you have to diagnose the nature of a lumbar tumor to bear in mind that, at the same point where a deepseated lumbar abscess is prominent under the skin, we may have that form of intestinal hernia with which Jean-Louis Petit has connected his name. Lately, a mistake was very nearly committed in a case of this kind, had not the surgeon before proceeding to open the supposed abscess, endeavored to reduce the tumor.

Were it not that physicians are more frequently consulted than surgeons regarding lumbar pains, I should not have said so much about perinephric abscesses; it was important that I should give you a detailed description of deepseated renal abscesses, sufficient to enable you in their beginning to suspect their existence, foresee their progress, and discover their etiology. I have specially brought under your notice the cases of perinephric abscesses on account of the importance of determining from the first, whether an abscess be primary, or whether it be secondary to lesion of the kidney. In the former case, the prognosis will be nearly always favorable, particularly if you early recognize the pus, and evacuate it from the lumbar region. But the prognosis is very unfavorable if the perinephric inflammation has been allowed, by temporizing treatment, to advance to the iliac fossa or the diaphragm. The prognosis is also very grave, when the perinephric abscess is the sequel of calculous pyelonephritis. There are cases which show that renal calculi may, after a period of varying duration, make a way out for themselves by determining an opening in the abscess: in such cases, urinary fistulæ may remain for many years, which must not be closed, lest by so doing, the patient be exposed to the risk of new inflammatory attacks.

I have told you, gentlemen, why perinephric abscesses are as much within the domain of the physician as of the surgeon. It is therefore my duty to give to all of you, but particularly to those of you who will have to practice both medicine and surgery, the results of my experience in the treatment of perinephric phlegmons and abscesses.

You have seen that a phlegmon may terminate in resolution. When the affection is incipient, you must endeavor to obtain that result. First of all, use every effort to calm the pain by frictions with the preparations of opium and belladonna, or by subcutaneous injection of solutions of atropia or morphia. Cupping and large flying blisters may be applied with benefit to the seat of pain. The bowels must at the same time be kept open by the daily use of saline purgatives and injections. Purgatives have a twofold beneficial action in these cases: they remove the constipation, and prevent the pain which would be caused by straining at stool. They also promote the resolution of the phlegmon by their antiphlogistic action.

Should these various measures fail to arrest the progress of the inflammation, and should an increase of fever with frequent rigors show that the phlegmon is suppurating, the utmost care must be taken to recognize, as soon as possible, the physical signs of suppuration. You will soon detect a doughiness in the whole of the affected region; and pressure with the hand, or the slightest movement of the body, will increase the pain. The tumor will soon become more prominent in the lumbar region; and though there may be no redness observable in that situation, you will perceive a local œdema which will convince you of the presence of pus. You will then be able to detect deepseated fluctuation, which will be rendered more manifest by your grasping the tumor in both hands and submitting it to a brusque concussion-stroke. There must then be no hesitation as to evacuating the purulent fluid. Three methods of accomplishing that object are available.

Chopart, and recently Drs. Denonvilliers and Gueneau de Mussy, have applied caustics, so as to produce adhesions and avoid peritonitis and hemorrhage. There are numerous methods of carrying out this practice. The application of the Vienna caustic paste, repeated once or oftener to the same place, may suffice; for the natural process of eliminating the eschar, provided it be deep, will terminate by making an opening into the abscess: the pus will then flow out slowly; and in some fortunate cases, the abscess will be entirely evacuated, deep cicatrization taking place at the same time that superficial cicatrization is accomplished on the falling off of the eschar. Though this method of proceeding has great advantages, it has often, in my opinion, the great drawback of being very slow, and so affording time for the abscess to extend to the iliac fossa or diaphragm, or open into the intestine. The advocates of the method are quite aware of this objection: MM. Denonvilliers and Gueneau de Mussy have, on the second or third day after applying the caustic, cut out the eschar to get at the abscess. This mixed method —cauterization and incision—has the advantage of diminishing the thickness of the tissues which have to be traversed by the bistoury, and causing the formation of adhesions between the superimposed tissues. It has the disadvantage of risking hemorrhage, by the bistoury cutting into deepseated vessels which had not come under the influence of the caustic. I prefer to make the incision in the first instance, taking care, however, to cut layer by layer, and to ligature all the arteries divided by the bistoury. If this principle be adopted, it is of little consequence whether the incision be longitudinal or transverse. However, when the thickness of the lumbar wall renders it difficult to tie the deep-lying arteries, it will be more prudent, after cutting through the superficial parts, to separate the deep parts by tearing them with a canulated sound. The risk of dividing an artery will thus be obviated. The incision through the superficial must be larger than through the deep parts, so that the pus be not detained in the wound to detach the tissues and burrow between the subcutaneous aponeuroses and muscles. A large pledget of charpie ought to be inserted in the wound so as to reach into the abscess. Any venous or capillary hemorrhage which may arise from the lips of the wound will be easily arrested by the application of some pieces of agaric. When the patient's general state is satisfactory, and when the perinephric abscess is primary, the walls of the abscess will soon contract on themselves, and in a fortnight or three weeks from the date at which the opening was made, complete cicatrization—deep and superficial—may be expected. Sometimes, however, the suppuration continues for a longer period, which may depend on a difficulty in the flow of the pus, or on some special condition of the walls of the abscess. In the former case, the deep opening must be enlarged; and in the latter case, detergent injections must be thrown into the cavity of the abscess: the injections, which ought to be repeated at each morning and evening dressing, may consist of tincture of iodine diluted with two or three times its weight of tepid water. Incision has the advantage of giving an immediate outlet to a great quantity of pus, and allowing a digital exploration to be made of the kidney, so as to ascertain its position and discover whether it be or be not diseased. It is unnecessary to add that, when the finger detects the presence of calculi imprisoned in the kidney, nothing ought to be done to favor cicatrization of the surgical incision: on the contrary, a fistula ought to be maintained to enable urine and pus to find an exit.

The method of drainage, to which M. Chassaignac has several times successfully had recourse for the evacuation of deepseated lumbar abscesses, allows both a continuous flow of pus and the gradual contraction of the cavity and the abscess. It also lessens the risks of hemorrhage, and does not appear to favor purulent infection; but it has the disadvantage of hardly allowing complete exploration of the abscess and the kidney. Moreover, the presence of the tubes frequently maintains a purulent discharge for a long period.

LECTURE XCVII.

PERIHYSTERIC ABSCESS.

Perihysteric Abscess, including Phlegmon of the Broad Ligament and Pelvi-peritonitis or Female Orchitis.—Etiology.—Symptoms and Duration of Pelvi-peritonitis.—Perihysteric Tumors.—Spontaneous Opening of the Abscesses into the Intestine, Bladder, and Vagina.—Complications.—Diagnosis of Perihysteric Abscesses.—Preventive Treatment of Perihysteric Abscesses.—Active Intervention only proper in the Iliac Abscesses.

Gentlemen: You know my dislike to neologisms, and the pain it gives me to become the author of a new word. I have a respect for old names; yet I avow that I feel it very distasteful to employ barbarous terms such as those which result from the alliance of a Greek preposition with a Latin substantive. Hence my objection to the word *periuterine*, employed to designate different affections which have their seat around or in the neighborhood of the uterus. It is a hybrid, badly coined word, for which I propose to substitute *perihysteric*, from περι and ὑστερα. It has the advantage of being composed of two Greek words. I have, however, so little paternal partiality for the term, that if you prefer the word *circum-uterine*, recently introduced into medical nomenclature, I am quite willing to accept it.

Having disburdened myself on that preliminary point, I now enter at once into the subject. In the study of perihysteric abscess upon which we are now entering together, I propose that we do not confine our attention to abscesses situated around the uterus, but that we likewise consider those which have secondarily invaded the iliac fossæ and the sacro-iliac symphysis. It is necessary thus to extend the limits of our study, on account of the impossibility to be always able to determine with precision the starting-point, the primary seat of perihysteric affections.

Gentlemen, I take it for granted, that you recollect the principal details of the anatomical topography of the female pelvis. Allow me, however, to remind you of the great, special features in its anatomy, because unless you possess that knowledge, it will be impossible for you to understand what I have to say on the progress of perihysteric abscesses.

The cavity of the pelvis, which contains and protects the uterus and its annexes, may be divided into two regions,—the anterior, and the posterior. The uterus and the broad ligaments constitute the boundary between these two regions. The uterus, the normal axis of which is the axis of the true pelvis, is suspended in the pelvic cavity and maintained in its normal relations by the broad ligaments and the *retro-uterine* and *ante-uterine* ligaments. These last-named ligaments, you are aware, are formed by folds of the peritoneum, which proceed from the rectum and bladder, to be inserted in the inferior lateral portion of the body of the uterus. At these points, the serous membrane is strengthened by fibres of fibrous and muscular tissues. The broad ligaments have the same fibro-muscular structure as the ante-uterine and retro-uterine ligaments, but they are much wider, and have a quadrilateral form: they present, in apposition, two layers of peritoneum, containing in their upper margin the Fallopian tubes, the round ligament of the uterus, and the ovarian ligament. In that situation in particular, there are numerous fibres of connective and muscular tissue: there, also, converge the utero-ovarian vessels. The inferior margin of the broad ligament is inserted in the floor of the pelvis. The internal or uterine margin is a continuation of the folds of peritoneum which cover the anterior and posterior surfaces of the uterus. Between them, at the point at which they are inserted into the uterus, there is a space, the dimensions of which vary with the degree in which the organ is full or empty; but they are always so firmly adherent to the anterior and posterior surfaces of the uterus by cellular tissue, that it is impossible, by the most patient dissection, to detach the peritoneum at these points, without tearing it. This anatomical fact is very important; because it establishes the impossibility of the peritoneum in that situation being separated by circumuterine suppurative inflammation. The external margin of the broad ligament is the widest: its extreme limits are the iliac fossa and the floor of the pelvis: consequently, by means of the cellular tissue which joins the two peritoneal folds, it is in immediate relation with the subperitoneal cellular tissue of the iliac fossa and pelvic cavity. These insertions, while they keep the empty uterus in its position and normal relations, are not so rigid as not to yield when the organ is increased in size, as it is in certain pathological states, or in cases of tumor of the broad ligament. If you keep in view these anatomical relations, you will easily understand my description of tumors situated before, behind, or in the broad ligament. Again, by recollecting the relations of the round ligament and ovary with the cellular tissue of the iliac fossa and cavity of the pelvis, you will be easily able to follow the advance of purulent collections in these different situations.

In a previous lecture, when treating of perinephric abscess, I explained to you how abscesses originating in the circumrenal cellular tissue might burrow in that tissue, so as to reach the true pelvis and open into the bladder or vagina. When the pus flows into the vagina, we can sometimes see, by the aid of the speculum, the fistula by which it is discharged: before the spontaneous rupture of the abscess, a tumor of greater or less volume on the side of the uterus could, by digital exploration, be recognized projecting into the vagina. I remind you of these facts, because they show beyond the possibility of doubt, that the cellular tissue of the pelvis may be the seat of purulent collections without the existence of any peritoneal inflammation. Here, let me repeat, that it is exceedingly unusual for such abscesses to open spontaneously into the peritoneum. You are thus enabled to anticipate my statement, to the effect, that real abscesses may be formed in the perihysteric cellular tissue, and that they are not always attributable to pelvi-peritonitis.

However, to be convinced of the existence of abscesses in the cellular tissue of the pelvis, it is well to recollect certain facts in pathological anatomy, which can seldom be verified, as the patients do not generally die till after the inflammation has gone on for several weeks or months, and has extended to the peritoneum, rendering the anatomical demonstration quite impossible.

Most of you, no doubt, remember the autopsy of a young woman who died in St. Bernard Ward, consequent upon purulent infection supervening a fortnight after delivery. I was anxious to discover the seat of the purulent infection: no morbid change was visible in the uterine veins, but the walls of the vagina were covered with pustules situated in a vascular network. Moreover, the cellular tissue lining the vagina presented all the characters of inflammation: that is to say, the layers of tissue were engorged with a yellow serosity, in some places having a purulent appearance; while, at the same time, the tissue had acquired a special hardness. The phlegmon extended to the sides of the pelvis. In difficult labors, it is not unusual to observe contusions of the soft parts of the pelvis leading to engorgement of the cellular tissue of the contused parts. The engorgements may terminate in resolution; but they may sometimes also become the origin of pelvic abscesses projecting around the uterus, the peritoneum not being necessarily implicated in the inflammatory action. In Dr. Dumontpallier's inaugural thesis, you will find the case of a young woman who died on the sixth day after delivery from a very acute attack of non-purulent traumatic peritonitis. The cellular tissue of the left iliac fossa contained pus; but it was particularly in the true pelvis that the most serious morbid changes were found. The whole of the cellular tissue attaching the different organs to the osseous parietes was infiltrated with purulent serosity, and presented in some places, particularly on the left side, layers of sanguineous deposit, the manifest evidence of great contusion. It is probable, that had this patient not succumbed under the severe peritonitis, she would ultimately have presented all the signs of iliac and pelvic abscess.

It appears then, that contusion during labor may be the origin of pelvic phlegmons, which, becoming developed and going on to suppuration, produce a perihysteric abscess. This cause of pelvic phlegmon is much less common than inflammation of the uterus and its appendages. When there is suppurative inflammation of the placental surface, uterine phlebitis and lymphangitis are, as you know, very frequently observed. When, at the autopsy, you look for pus in these vessels, you know that the chosen place for it is in the veins at the margin of the uterus: an incision made in these vessels, the true confluents of the uterine veins, nearly always discloses small abscesses situated in the venous tissue itself. Moreover, around these veins, the cellular tissue of the broad ligament is œdematous; and if the patients do not die from purulent infection, it is because there has been adhesive phlebitis further down the stream than the purulent collection: the intravenous abscesses are often the origin of abscesses of the broad ligament. A similar remark is applicable to suppurative lymphangitis. In these cases, the peritoneum does not participate in the inflammatory action; and the abscess, if not absorbed, will tend towards the depending parts, or the inflammation may extend to the iliac fossa of the same side.

In the former case, the abscess, in the form of a tumor, will have its seat in the lateral parts of the uterus; and, sooner or later, will project into the vagina. You then have an abscess of the broad ligament, that is to say, a suppurative inflammation of the cellular tissue which unites the two peritoneal folds, between which lie the inflamed vessels, veins or lymphatics, the starting-point of the phlegmon.

Not only delivery, but any traumatic condition of the mucous membrane of the uterus, may occasion perihysteric abscesses. For example, they have been seen after abortion, and after surgical operations, such as scraping the uterus by Récamier's instrument. The actual cautery, and even the application of the nitrate of silver, have sometimes given rise to similar phlegmons. Perhaps a similar result has been caused by excessive coitus. I am inclined, however, to think that this kind of excess more frequently produces pelvi-peritonitis. Though primary inflammation of the veins or lymphatic vessels is the most frequent cause of an abscess, conditions necessary for its formation are a solution of continuity and inflammation of the mucous membrane of the uterus. These lesions exist after delivery, abortion, and the application of caustic or cautery: but I can hardly understand their being produced by excess of coitus, unless, indeed, uterine catarrh or ulceration of the cervix uteri previously existed.

Be that as it may, once the abscess is formed in the thickness of the broad ligament, what is the usual course of its progress? Most frequently, as I have said, it will project at the side of the uterus, and open into the vagina or bladder. It usually occurs on one side of the uterus only, and does not project either behind or in front of that organ. When an abscess is situated behind or in front of the uterus, the conclusion is that the pus is in the peritoneal cavity; or—to make the same statement in other terms—the abscess is due to pelvi-peritonitis. In point of fact, an abscess cannot invade an ante-uterine or a retro-uterine cellular tissue, inasmuch as neither exist!

When there is a real perihysteric abscess, that is to say, when the purulent collection is situated around the uterus, in the subperitoneal cellular tissue, and not in the cavity of the peritoneum, the pus sometimes reaches the iliac fossa, giving rise to the signs of abscess in that fossa—signs which vary according as the abscess is superficial or deep. It is generally superficial, and uncomplicated with psoïtis. Suppurative inflammation, moreover, must, it is evident, follow the cellular tissue to reach the iliac fossa.

When an iliac abscess is formed, whether consequent upon propagation of the inflammatory process from the broad ligament, or on inflammation primarily seated in the iliac fossa, pain is felt in the place where the abscess is situated. By moderate palpation, doughiness of the region is detected; and there is sometimes slight œdema of the abdominal walls. When the abscess is situated in the right iliac fossa, it may open into the cæcum, but more frequently, it points immediately above the crural arch. At other times, if prompt issue be not given to the pus, it may, following the vessels, work its way to the triangle of Scarpa, or reach the trochanter minor and coxo-femoral articulation.

When the last-described course is taken, the iliac abscess is generally deepseated, subaponeurotic, and complicated with psoïtis. These deepseated iliac phlegmons almost always terminate in death, the patients sinking from exhaustion consequent upon the suppuration. I could by narrating many examples prove these cases to be as serious as I have now stated. I will confine myself, however, to the description of one case, which presents many complications of great clinical interest; and shows the amount of damage produced by purulent collections of this kind. The patient, thirty-five years of age, was delivered on August 30th, 1861, at the Hôpital des Cliniques, where she remained till the 18th September, experiencing pain in the hypogastric region. On leaving the hospital, she kept her bed at her own home till the 5th October, the date at which she was admitted to the Hôtel-Dieu, bed 5, St. Bernard Ward. I then ascertained that there was a phlegmon of the right broad ligament. Soon, there was an abscess; and the inflammation invaded the right iliac fossa. Early in November, the urine was observed to deposit a large quantity of pus. Very probably, a fistulous communication had been established between the abscess of the broad ligament and the bladder.

The inflammation of the iliac fossa nevertheless continued to advance; and about the middle of November, a tumor could be readily recognized, tending towards the crural arch. At the same time, very acute pains supervened in the iliac region, and throughout the whole of the inferior extremity: extreme pain was caused by the least pressure on the tumor, or by the slightest movement of the limb. The pain was constant: and paroxysmal exacerbations extorted cries from the patient.

The iliac tumor pointed immediately above the crural arch, where flucfore, reasonable to suppose, that the symptoms attributed to psoïtis do not belong to any lesion of the muscle, but to lesion of the crural nerve?

In proof of the value of this remark, it is sufficient, on the one hand, to find cases of inflammation of the psoas muscle without symptoms of psoïtis; and on the other, to recognize these symptoms in cases in which the crural nerve has been seriously implicated in the process of inflammation. By a singular chance, I, next day, met with these latter conditions. The deceased was a young man who had just succumbed from typhoid fever in Dr. Horteloup's wards. Dr. Lefeuvre, then interne of the service, invited me to verify the existence of an enormous abscess in the substance of the psoas muscle. The pus was thick and mingled with blood. The muscular fibre was partially disorganized: some portions of it, when examined under the microscope, presented the appearance of pale longitudinal striæ, and the transverse striæ were only visible in some places: the muscular sheath contained a quantity of small shining fat-globules. The nerve-bundle constituting the crural nerve had not been involved in the inflammation. The limb was extended, and parallel with that of the opposite side.

Dr. Lefeuvre informed me, that the patient had never complained of pain, nor of any symptom which could lead to the conclusion that there was psoïtis. You see, therefore, that in this case, there was great lesion of the psoas muscle, without any symptoms which could be attributed to psoïtis.

I thought that the psoas abscess was probably a metastatic abscess resulting from purulent infection, the anatomical cause of which was probably ulceration of the glands of Peyer. Purulent infection is an unusual sequel of typhoid fever: here, however, was a case in which it had occurred, as was proved by the presence of numerous metastatic abscesses in both lungs. Having shown that this case takes its place with other cases of purulent infection, I now return to the principal fact, viz., the absence of the symptoms of psoïtis in a case of undoubted suppuration of the psoas muscle.

In this case, then, there was psoïtis properly so called, that is to say, inflammation of the inter-fibril cellular tissue, along with disorganization of the muscular fibre, yet there had been none of the symptoms attributable to psoïtis, and—let me call your special attention to the fact—there was no lesion of the crural nerve.

From the conjoint consideration of these two cases, we may, I think, draw the following conclusions:

1. That the symptoms of psoïtis specially depend on lesion of the crural nerve.
2. That in certain cases of psoïtis, a great pathological change may take place in the psoas muscle, there being nevertheless an absence of symptoms attributable to psoïtis.
3. That inflammation of the psoas muscle and inflammations of the iliac fossa, may often give rise to the alleged characteristic symptoms of psoïtis when the crural nerve has been secondarily invaded by the surrounding inflammation. [In justice to Professor Grisolle, I must mention, that in his remarkable memoir on iliac abscesses, he attributed a great share to lesion of the lumbar plexus and crural nerve, in explaining the pains which accompany iliac phlegmons and abscesses.*]

We have seen, gentlemen, that deepseated iliac abscesses may produce grave alterations in the coxo-femoral and sacro-iliac articulations: the existence of these lesions is generally unknown till disclosed at the autopsy.

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* GRISOLLE: Archives Générales de Médecine. Third Series (1839): vol. iv.

thickened, and the uterus was almost glued to the right wall of the pelvis by the contraction which had taken place in the diseased tissues subsequent to the evacuation of the pus by the vesical fistula.

During life, I had been led to the conclusion that there was a vesical fistula; and at the autopsy, I had searched for the communication between the broad ligament and the cavity of the bladder. An opening having been made in the bladder, in its anterior superior part, a vesical fistula was observed, which communicated with the primary abscess of the broad ligament. The fistula was situated in the right lateral portion of the fundus of the bladder, three or four centimetres behind the orifice of the right ureter.

The uterus, vagina, and rectum, on careful examination, were found not to communicate with the abscess, and to present no morbid change. The pus was in immediate contact with the psoas magnus; but there was no morbid change in its fibres, except that the superficial fibres had a greenish color, due to contact with the purulent fluid.

The lungs were engorged, but presented no traces of inflammation. Both apices contained some small masses of softened tubercle. The lungs and liver were both free from any trace of metastatic abscesses. On some parts of the surface of the liver, there were small irregularly shaped yellowish spots of the size of a twenty centime piece, due to the presence of numerous fat-globules in the hepatic cells. The spleen was small, and not softened. The kidneys were normal in color, form, and texture.

The pathological physiology of these cases appears to me exceedingly interesting. Here, a recently delivered woman had abscess of the broad ligament, and the inflammation had extended by continuity of the cellular tissue of the true pelvis and iliac fossa. The abscess of the broad ligament had emptied itself into the bladder, whilst the subaponeurotic abscess made great ravages, penetrating into the sacro-iliac symphysis, dissecting the crural nerve, pointing above the crural arch, after having detached the superficial fascia of the anterior abdominal wall: it was at that point that the surgeon introduced his bistoury. The abscess had burrowed below the crural arch, following in one direction the crural nerve, and in the other, the psoas muscle to its trochanterian insertion. It advanced to the coxo-femoral articulation; and denuded the head of the femur of its cartilage.

The connections formed between the uterus and broad ligament and fundus of the bladder, by cellular tissue, explained the opening of the pelvic abscess into the bladder. The progress of the iliac abscess, and its tendency to burrow towards the regions which I have mentioned, may be foreseen: but it is not less remarkable to see the ravages of the inflammation extend to the sacro-iliac and coxo-femoral articulations.

The grave articular changes would be sufficient in themselves to explain the pains which the patient suffered: every movement occasioned frightful torture: and though it was not forgotten that the crural nerve had been dissected by the pus, that many of its branches detached by the inflammation of the nervous bundles were swimming in it, there was found in the articular mischief an explanation of the acute pains which darted through the inferior extremity with that paroxysmal character so remarkable in lesions of the nerves.

Here, gentlemen, allow me to make a short digression, touching the value of the lesion of the crual nerve in explaining the symptoms of psoïtis. This lesion seems to me to explain the symptoms which we have observed, and which are generally attributed to psoïtis. In our patient, the psoas magnus muscle had undergone no pathological change. Is it not, therefore, reasonable to suppose, that the symptoms attributed to psoïtis do not belong to any lesion of the muscle, but to lesion of the crural nerve?

In proof of the value of this remark, it is sufficient, on the one hand, to find cases of inflammation of the psoas muscle without symptoms of psoïtis; and on the other, to recognize these symptoms in cases in which the crural nerve has been seriously implicated in the process of inflammation. By a singular chance, I, next day, met with these latter conditions. The deceased was a young man who had just succumbed from typhoid fever in Dr. Horteloup's wards. Dr. Lefeuvre, then interne of the service, invited me to verify the existence of an enormous abscess in the substance of the psoas muscle. The pus was thick and mingled with blood. The muscular fibre was partially disorganized: some portions of it, when examined under the microscope, presented the appearance of pale longitudinal striæ, and the transverse striæ were only visible in some places: the muscular sheath contained a quantity of small shining fat-globules. The nerve-bundle constituting the crural nerve had not been involved in the inflammation. The limb was extended, and parallel with that of the opposite side.

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* **Grisolle**: Archives Générales de Médecine. Third Series (1839): vol. iv.

In a subsequent lecture, when discussing relaxation of the symphysis of the pelvis, before and after delivery, I will describe two cases in which there was suppuration of the sacro-iliac symphysis. In neither of these cases, was the articular inflammation the consequence of an intra-pelvic abscess.

In the case which I have just related an abscess of the broad ligament seemed to have been the origin of all the symptoms. However, abscesses of the broad ligament are not so frequent as some years ago they were said to be. Dr. Bernutz, and his lamented fellow-worker, Goupil, in their memoir published in 1857,* endeavored to show that pelvi-peritonitis is very common. They showed that in many cases in which the physician had made out all the signs of acute or chronic phlegmon of the broad ligament, it had been demonstrated at the autopsy, that the cellular tissue of that ligament had not been involved in the inflammation, and that the sole seat of the products of inflammation had been the peritoneum of the true pelvis. Hence these physicians, after collecting a great number of cases came to consider the pelvic peritoneum in the female as analogous to the tunica vaginalis in the male subject.

Orchitis, or, to speak more correctly, vaginitis, ought, therefore, to have pelvi-peritonitis as its corresponding affection in the female. Just as lesions of the urethra, prostate gland, and testicle, often originate orchitis, so (it is argued) must lesions of the vagina, uterus, Fallopian tubes, and ovaries, often produce pelvic peritonitis. I am willing to accept this ingenious interpretation of facts, and to recognize with MM. Bernutz and Goupil the existence of blennorrhagic, traumatic, tuberculous, and other kinds of pelvi-peritonitis. This classification has the advantage of obliging the clinician to carry back his investigation to the cause of the inflammation of the pelvic peritoneum. Science has long been in possession of the histories of numerous cases of these different kinds of pelvi-peritonitis; and practitioners in their daily practice are called upon to verify the symptoms of a localized peritonitis in women affected with vaginitis and acute or chronic inflammation of the uterus. All of you must have present to your minds some necroscopic examination in which inflammation of the Fallopian tubes and ovaries was seen to have afforded the starting-point for a more or less extensive peritonitis.

It is particularly as a sequel to normal or artificially induced labor, that we meet with inflammation of the tube and ovary. In these cases the peritoneum surrounding or adjoining the tubes and ovaries soon takes on the inflammatory action. Pain and swelling localized at some particular part of the pelvis, enable us to recognize the seat and nature of the lesion. Adhesions form between the affected parts; and if the peritonitis do not become general, the patients may recover after a certain time, retaining the cellular adhesions, which will make their presence known by occasioning pain, particularly at the menstrual epochs. You will often observe that these attacks of limited peritonitis are connected with some acute lesion of the ovary. The frequent recurrence of the inflammation may occasion numerous adhesions between the pelvic organs, whence may arise errors in diagnosis, if great care be not taken to mark well the progress of the engorgements, and observe the signs furnished by palpation and percussion. These numerous adhesions, uniting in the form of a tumor of the uterine annexes, to portions of the intestinal mass, sometimes extensive, lead to serious consequences; they impede the performance of the functions of organs, and produce marasmus.

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* Archives Générales de Médecine [March and April, 1857]; and Clinique Médicale sur les Maladies des Femmes, t. ii. Paris, 1862.

I have not often observed pelvi-peritonitis as a consequence of inflammation of the vagina; but every year, I frequently meet with peritonitis consequent upon metritis. Bear in mind the pains in the lower part of the abdomen complained of by some of our patients—pains accompanied by nausea, vomiting, fever, and dysuria—in the evening of the day or on the day following that on which we have applied caustic to the neck of the uterus. These pelvi-peritonitic symptoms generally subside in a few days: the inflammation, however, may extend to a great part of the peritoneum, when the patients may be carried off by very acute peritonitis.

Sometimes, mortal peritonitis is occasioned by leaving an instrument in the cavity of the uterus. In the discussion on stem-pessaries, which took place in the Academy of Medicine, several such cases were mentioned. The temporary introduction of the hysterometer has occasioned very alarming peritoneal symptoms. From these statements, there can be no doubt that an apparently slight uterine lesion may give rise to very acute general peritonitis: and, therefore, of course, we may assume, that acute metritis will much more readily occasion peritonitis limited to the true pelvis.

Pelvi-peritonitis, gentlemen, is characterized by effusion of serosity into the true pelvis, and the formation of adhesions which give rise to perihysteric tumors. These tumors, variable in volume, are situated at the sides of the uterus, or in the ante-uterine and retro-uterine cul-de-sacs. Sometimes, the broad ligaments assist in developing the tumor: in such cases, the uterus seems to be firmly nailed to the middle of the tumor. When the retro-uterine cul-de-sac is the seat of the pelvi-peritonitis, the uterus is carried forwards behind the symphysis of the pubes, as happens in retro-uterine hæmatocele. When the pelvi-peritonitis is more severe on one side than the other, lateral deviation of the uterus will be produced. The different deviations of the uterus are only of secondary importance. The great fact which you require to bear in mind is, that these peritoneal inflammations, which often become encysted abscesses, have a tendency, whether they be acute or chronic, to evacuate themselves by the rectum, vagina, or bladder, so that in a single day may disappear a great part of the tumor which occupied the true pelvis.

Pelvi-peritonitis is sometimes very protracted, and occurs in paroxysms. Some years ago, a surgeon who was treating one of my patients for a uterine affection of old standing, proposed to employ Récamier's scraper [*curette de Récamier*] to destroy fungous excrescences on the mucous membrane of the uterus. It is always a serious proceeding, gentlemen, to introduce a foreign body into the uterus, particularly when that is done with a view to remove diseased portions of the mucous membrane: there is always a risk of its producing metritis and its consequences. Under the circumstances, therefore, I proposed that another surgeon, a Professor of the Faculty, should meet us in consultation. My accomplished colleague realizing the great responsibility which he was assuming, wished to examine the affected organs with great care, so as to be able to state his reasons for the advice he gave. The digital examination occupied a long time; and was, the patient said, very painful. Possibly, there already existed latent chronic pelvi-peritonitis: it is important to note that from the day of our consultation, the patient felt almost constant pain in the lower part of the abdomen—pains which several times within the month assumed intense severity. For three years, our patient, obliged to keep her bed or lie on the sofa, passed a large quantity of pus by the anus. I need not say that, on all occasions, during this long period, the necessary digital examinations were made with the utmost caution. Several times I had an opportunity of ascertaining that there was a doughy tumidity [*empâtement*] within the pelvis, particularly in the retro-uterine region. Finally, in this patient, coagulations formed in the pelvic veins. One day, when the acuteness of the pains had rendered an examination necessary, the patient was suddenly seized, during the examination, with the symptoms of pulmonary embolism. The symptoms gradually abated: some months later the extent of the perihysteric engorgement diminished: no more pus flowed from the rectum. The patient may to-day be described as cured.

Very probably, this was a case of pelvi-peritonitis, and not of perihysteric phlegmon. The progress of a phlegmon is different and less protracted; pelvi-peritonitis, on the other hand, as necroscopic examinations prove, is characterized by chronicity; and when the adhesions are well established, it may, under the influence of a variety of causes, be the source of ceaseless suppuration. In saying this, I do not mean to express a positive belief that the cellular tissue lining the peritoneum remains exempt from the inflammation primarily seated in the pelvic peritoneum. In fact, I have difficulty in understanding how a chronic phlegmasia, accompanied by an abscess opening into the rectum, vagina, or bladder, should always remain limited to the peritoneum; I do not believe that that is the rule, although MM. Bernutz and Goupil have given very remarkable cases in support of such a view. In reality, chronic inflammation so thoroughly invades the cellular tissue of the pelvis, Fallopian tubes, and ovaries, that at the autopsy it is sometimes very difficult to distinguish any trace of these organs, and impossible to say the order in which the different organs were attacked, unless account be taken of the progress of the symptoms during life.

My patient's case proves that a chronic lesion of the mucous membrane of the uterus may lead to latent pelvi-peritonitis, and that a determining cause, such as digital examination, may bring on acute symptoms. Moreover, these symptoms become chronic, may have paroxysmal exacerbations, declaring themselves by more or less purulent discharge. Again, the blood may coagulate in the pelvic veins surrounding the inflamed tissues; and clumsy or inopportune manipulations may break up the clots, and so lead to their migration to the heart and lungs. Under such circumstances as I am now referring to, you cannot be too reserved in your prognosis, nor too prudent in your examination of the affected organs.

Pelvi-peritonitis may also lead to other consequences. Not only from its long duration may the suppuration cause great wasting of the body and hectic fever, but it may likewise, though not so frequently, lead to purulent infection. Chronic pelvi-peritonitis may also, in predisposed subjects, by its long continuance lead to tubercular affection of the lungs and peritoneum.

The peritonitis does not always remain limited to the pelvis; I have often seen it spread so widely as to comprise a great portion of the intestines in its adhesions. The extension of the peritonitis is particularly observed after delivery and abortion. Numerous autopsies show that a portion of the small intestine sometimes becomes united in one common mass with the great omentum and the organs contained in the cavity of the true pelvis. When that is the case, and when the patients do not die under the acute symptoms, they soon grow weak. The pus, however, which occupies the inflamed masses, at last makes an issue for itself; and in this way, recovery may take place. You remember the case of the young woman who occupied bed 23, St. Bernard Ward. On three occasions, with some days of interval between each, she presented the signs of peritonitis, which each time made progress; the acute symptoms abated; the tumor became circumscribed, and discharged its pus by opening spontaneously into the vagina. Some weeks afterwards, the woman left my wards quite recovered, except that she continued to have the abdominal pains inseparable from extensive adhesions of the abdominal peritoneum.

As I have already said, inflammation of the Fallopian tubes and ovaries, so common in recently delivered women, may be the source of pelvi-peritonitis and phlegmons of the iliac fossa. But under certain circumstances, when there is no adhesion of the tube or ovary to the surrounding parts, the abscesses of these organs may open into the cavity of the peritoneum, and (as I have twice seen) give rise to rapidly mortal attacks of peritonitis.

I have now, gentlemen, brought under your notice several cases which were phlegmon of the broad ligament, iliac abscess, pelvi-peritonitis, ovaritis, and metro-peritonitis; and you may have remarked, that some of the earliest symptoms specially suggested the particular affection; but you have also seen that, as the case advanced, the differential characteristics gradually became effaced by the mere advance which was taking place in the disease. In several of the patients, examination of the body after death showed that there was both pelvi-peritonitis, and abscesses in the pelvis, iliac fossa, or even in the renal region. You see, therefore, that it would be very difficult to give you a clinical description of each of these affections, as they have such a variety of causes, embracing traumatic influence, which is the most simple, and the puerperal state, which is the most complex cause.

I am now about to try to present to you some clinical views, by which you may be enabled to distinguish certain varieties of the different affections we are now considering. It is unusual for any one of them to exist separately; they are generally accompanied by numerous lesions of the uterus, ovary, or peritoneum.

When the abscess is situated in the broad ligament, digital examination will enable you to distinguish a very decided resistance to pressure in one side of the uterus; and if you, at the same time, apply the free hand to the anterior wall of the abdomen, you may, by steady gentle pressure, detect a tumor, of greater or less size, in the pelvis. The conditions under which the tumor is developed, the fever, and the pain, are very important elements of diagnosis, and justify the opinion that the phlegmon is situated in the broad ligament. The relations of the uterus to the tumor will also assist in the diagnosis; and when the patients in the beginning of the disease have had neither nausea nor vomiting, and when the seat of the pain has remained confined to the situation of the tumor, it is exceedingly probable that the inflammation has not extended to the pelvic peritoneum. It also not unfrequently happens that the patients complain of only very slight pains in the hypogastrium; in such cases, you will find that the tumor is limited to the region of one of the broad ligaments. In these cases, digital examination by the rectum proves that the retro-uterine cul-de-sac does not contain any product of inflammation. If no peritoneal complication supervene, the abscess will open into the vagina or bladder.

Iliac phlegmons are sometimes the consequence of the propagation of the inflammation to the broad ligaments: at other times, particularly after delivery, they originate primarily in the iliac fossa. Whatever may have been the conditions under which they originated, they can always be easily recognized by the pain and doughy tumidity of the affected region. Moreover, the progress of the inflammation will soon supply the elements necessary for diagnosis, and show these abscesses to be either superficial or aponeurotic.

When the abscesses are superficial, the pains are limited to the affected region; but if opening the abscesses be delayed, they become subaponeurotic; the pains will then extend to the thigh, following the course of the crural nerve; and the abscesses will very soon point above the crural arch, or burrow deep down in the thigh. You have seen the destruction to the coxo-femoral articulation, and sometimes, though not so frequently, to the sacro-iliac articulations, induced by these abscesses.

The prognosis is favorable when the abscess is limited to the broad ligament. On the contrary, if it be situated in the iliac fossa, there is reason to fear its becoming subaponeurotic, and endangering the life of the patient.

I need hardly say, that when the abscess is situated within the cavity of the peritoneum, the diagnosis is simplified by the previous peritonitis. When the abscess is large, its walls are formed by intestine, great omentum, uterus, ovary, ovarian annexes, and bladder. A tumor is thus formed, situated in the true pelvis, and also occupying to a considerable extent the hypogastrium. In these regions a peculiar doughiness is detected, in the midst of which the experienced hand can sometimes recognize, in thin subjects, by palpation, a non-homogeneous resistance. On percussion, different degrees of dulness are perceived, showing that a portion of intestine is involved in the tumor. It is found, by digital examination, that a change has taken place in the relations of the uterus—that it has lost its mobility, and is nailed, as it were, to the middle of the tumor. At the bottom of the vagina, and in the cavity of the pelvis, the finger feels a swelling, which extends to the sides of the pelvis and to the retro-uterine cul-de-sac. When such conditions exist, it is certain that the peritoneum is the seat of inflammation.

But it must not be supposed that pelvi-peritonitis, in whatever cause it originates, has always such precise diagnostic signs. It often becomes developed as a latent affection after delivery and abortion, or arises from inflammation of the uterus and vagina. We then find, that though the patients experience some pains, they neither have vomiting nor nausea. The ordinary assemblage of symptoms of peritonitis is absent, and I confess that it is very difficult to assign to this form of pelvi-peritonitis any distinctive characters. Modern works, however, and particularly the memoir of Goupil and Bernutz, have so completely established the great frequency of pelvi-peritonitis, that you must always endeavor to detect, in the antecedents of the patients, a cause for the inflammation of the peritoneum.

For the present, I avoid introducing the more difficult cases into the discussion; and confine myself to a consideration of the morbid characters which seem to explain the more common facts of pelvi-peritonitis. When patients suffer from pain in the hypogastrium as a sequel of sexual excess, or as an accompaniment of gonorrhœa or metritis, the possible existence of pelvi-peritonitis immediately suggests itself. If it exist, palpation of the hypogastrium will produce pain, and digital examination by the vagina, if pain has existed for some previous days, will detect a tumor occupying the perihysteric region. The pains then occur in paroxysms, while the tumor becomes more and more appreciable to the means of investigation employed. These abscesses of the pelvic peritoneum, sooner or later, and at intervals, open into the vagina, bladder, or rectum.

The prognosis in pelvi-peritonitis varies with the extent and cause of the inflammation. When it involves a portion of the intestine and the annexes of the uterus, the case is one of great gravity, because hectic fever will soon set in. Orchitis in the female, on the contrary, when limited to the pelvis, and not interfering with the functions of the bladder, ovaries, and Fallopian tubes, in general, terminates favorably, especially when the pus has an outlet from the body, and the cause of the inflammation has ceased.

Gentlemen, in presenting to you these clinical considerations on perihysteric abscess, I have not intended to give you a complete description of all the inflammatory affections of the peritoneum and pelvic cellular tissue. In the actual state of science, I do not believe it possible to give such a description. Read the works on this subject of my much-lamented hospital colleagues, the late Drs. Valleix,* Aran, and Goupil;† study with attention the memoir, so rich in facts, of Bernutz on pelvi-peritonitis, or orchitis in the female: you will then be convinced that the subject which I have been now discussing with you is one of the most obscure in pathology.

Be that as it may, I have pleasure in stating, that we are indebted to the authors I have now named for important views: it was a great contribution to science, to establish, that pelvi-peritonitis occurs frequently, and that perihysteric *phlegmon* is relatively rare.

I do not think it necessary to speak at any length on the pathological anatomy of pelvic abscess: the anatomical details given in my accounts of cases will suffice to impress on your minds the principal lesions.

Let me now describe to you a case which illustrates the multiplicity of pelvic lesisons which may originate in inflammation of the uterus: and which also shows that the actual cautery, usually exempt from danger, may sometimes occasion mortal lesions.

A woman, aged twenty-seven, was admitted to St. Bernard Ward. She stated that she menstruated regularly, but always had leucorrhœa, and pains in the loins and lower part of the abdomen. By digital examination it was found that the neck of the uterus was swollen, half open, and deeply excoriated. I resolved to apply the actual cautery. I generally employ it, when the lesions of the cervix are not superficial; and I have done so for more than fifteen years, without any bad results having been produced, till the case occurred which I am now going to relate. I applied the actual cautery to the cervix. Everything seemed to go on exceedingly well: the slough separated in a few days: the menses appeared in the interval, and four days after their cessation, I applied the actual cautery a second time, hoping that it would not be necessary to repeat the application. Five or six days after the second application, there was slight pain in the left iliac fossa, and deepseated doughiness could be detected on palpation. Some days later, the pain increased, and there supervened neuralgia of the crural nerve, with contraction of the thigh on the pelvis. The iliac swelling became more apparent, the pains in the thigh increased, and the power of extending the limb ceased. On attempting extension of the limb, very acute pain was occasioned, shooting deep into the pelvis. Intense fever set in; and I saw clearly that there was phlegmon of the sheath of the psoas and iliacus muscles. A doughiness soon became appreciable below the crural arch, and pus burrowed to the trochanter minor. About five weeks after the last cauterization, the unfortunate young woman died exhausted with fever and diarrhœa.

On examining the body after death, an abscess was found in the left broad ligament, slight peritonitis, and a purulent collection which had dissected the psoas and iliacus muscles, and extended from the lumbar region to the trochanter minor.

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* VALLEIX: Guide de Médecin Practicien.

† GOUPIL: Leçons Cliniques sur les Maladies de l'Utérus et de ses Annexes. Paris, 1860.

I have often asked myself, gentlemen, whether the cauterization occasioned the disaster; and I have answered the question in the affirmative. It is certain that if I had not performed that little operation, the iliac phlegmon had never been formed. But, gentlemen, when I inquire into the results of my own practice, when I interrogate professional brethren who are not afraid to tell the truth as to their practice, I discover, that in a few rare instances, the most superficial cauterization of the cervix has induced inflammation of the broad ligaments and peritoneum: I discover that not only is cauterization with the red-hot iron generally free from danger, but is even more generally harmless than cauterization with potential caustics. As a general proposition, it is correct to say, that the deep sloughs produced by the actual cautery are a guarantee against serious phlegmons of the pelvis.

It must be admitted, that medicine is nearly impotent in the treatment of perihysteric abscesses. When the symptoms have informed us of the existence of phlegmon or peritonitis, I place exceedingly little reliance in the resolutive action of local or general bleeding. Generally, my only aim is to calm the pain by local applications, or the internal administration of preparations of belladonna and opium. By silencing the pain, it frequently happens that the inflammatory fluxion is moderated; and when I accomplish that object, I am well pleased with the result of the treatment.

Although we can do but little for the cure of pelvi-peritonitis and phlegmons of the pelvis, we can do a great deal in respect of the causes on which the inflammation depends. All our efforts, therefore, ought to be directed towards prevention, towards diminishing the influence of the causes, and studying the organic susceptibility before we interfere with chronic affections of the womb. For example, when menstruation is painful, and profuse menorrhagia testifies to the existence of an abnormal fluxion towards the genital organs, the woman ought to be advised to avoid everything likely to increase pain or hemorrhage. Delivery, as I have said, is the most common cause of pelvic abscess; but we learn from experience, that unless an epidemic influence be prevailing, pelvi-peritonitis and perihysteric abscess are hardly ever observed, except in persons in whom labor has been abnormal or protracted, or in patients who have committed some imprudence within a few days of delivery. To state the etiological conditions, is to indicate the rules which ought to be followed to avoid the consequences of these conditions.

In the cases in which it is necessary to use caustics to modify inflammation of the cervix uteri, or in uterine catarrh, always proceed with extreme caution: always fear setting up an inflammation which may extend to the peritoneum. Interrogate—if I may use the expression—interrogate the susceptibility of the organ on which you are going to act; and before resorting to powerful caustics, use those which are feebler: stop your applications whenever you find they occasion acute pain.

Likewise, in specific inflammation of the mucous membrane of the vagina, abstain from interfering too brusquely by substitutive treatment. Recollect, that in these cases also, there is a risk of causing pelvi-peritonitis.

Gentlemen, in making a digital examination of the uterus, you cannot exercise too much caution. I have related to you a case in which a somewhat roughly performed digital examination occasioned pelvi-peritonitis, which lasted for three long years!

Never employ the hysterometer except in the exceptional cases in which catheterism is indispensable for diagnostic purposes. Such cases are of infinitely rare occurrence. In a still more emphatic manner, I denounce intra-uterine pessaries, which ought not to be employed under any circumstances: this opinion I the more hold to, from having been taught by experience, that deviations of the womb are almost never the cause of the pains and discomforts from which women suffer. These pains are generally symptomatic of metritis, uterine catarrh, or chronic pelvi-peritonitis, which are only aggravated by the introduction of a foreign body into the uterus.

When pelvic abscesses point in the vagina or rectum, they ought not to be opened. The physician ought likewise to refrain from active interference in cases of retro-uterine hæmatocele. On the other hand, when the perihysteric abscess has invaded the iliac fossa, operative interference is demanded. You are acquainted with all the grave complications of these abscesses when allowed to remain too long in contact with the psoas muscle and the cellular tissue of the iliac fossa: you know that they have a tendency to work their way towards the sacro-iliac and coxo-femoral articulations, and that when this takes place, the issue of the case is nearly always mortal. Consequently, as soon as you have become satisfied by an attentive examination, that there is pus in the iliac fossa, you ought to open the abscess by the bistoury; or at least, you ought, by the use of caustics, to hasten the formation of adhesions, so that you may safely make the opening. In these circumstances, you must not wait too long; for prolonged waiting may render ultimate interference completely useless.

LECTURE XCVIII.

NEW SPECIES OF ANASARCA, THE SEQUEL OF RETENTION OF URINE.

The Anasarca is observed, and the Retention of Urine is not recognized.—Relation of Cause and Effect between the Anasarca and the Retention is, with greater reason, not recognized.—The Distended Bladder may be mistaken for a Malignant Tumor.—Accumulation of Urine.—The Anasarca is rapidly cured by the Evacuation of the Urine.—Why Retention of Urine causes Anasarca.

GENTLEMEN: I wish to avail myself of this opportunity of speaking to you regarding a species of general dropsy which accompanies retention of urine. Though we see less frequently in the medical than in the surgical wards patients suffering from affections of the urinary passages, yet it is not unusual for persons with anasarca to be directed to our wards, because dropsy is generally looked upon as the consequence of some grave internal lesion. Hospital surgeons themselves do not hesitate to send this class of patients into the medical wards, because they nearly all ignore the influence which retention of urine sometimes has in producing anasarca. For a similar reason, we physicians receive these patients in our consulting-rooms; whereas, had this species of anasarca been better known, they would have been sent to our colleagues who devote themselves to surgery.

Gentlemen, you have not forgotten the patient who, during the summer of 1864, was admitted to St. Agnes Ward, and there occupied bed No. 3.

With me, you were struck by the great amount of anasarca in every part of the body. When, after examining the case for a minute, and discovering that the bladder was distended, I intimated that that condition was very probably the cause of the dropsy, which would disappear when the urine was drawn off by the catheter or passed freely, I observed a smile of incredulity on your faces. Gentlemen, the urine was not albuminous: the liver, heart, and lungs presented no abnormal indication: no trace of cancerous or tuberculous disease could be detected. Some days later, you were convinced that I was right: at all events, you admitted, that in that particular case I was not mistaken, and you learned from the patient who had not attached much importance to the circumstance, that for two months, micturition had been becoming more and more difficult; and that the patient had long had stricture of the urethra and disease of the prostate. When cured of his dropsy, I sent him into the wards of my colleague M. Maisonneuve, whose province it was to treat the affection of the urinary passages, the cause of the anasarca.

The diagnosis which I made with so much ease, and the favorable prognosis which I formed in this case, apparently so very serious, were, gentlemen, neither the results of divination nor of chance. More than ten years previously, I had learned from Dr. Bourgeois (of Etampes) to recognize cases of this kind. In 1855, he described to me a very remarkable form of general dropsy, which he had observed after incomplete retention and insufficient emission of urine. I strongly urged him to send a note on the subject to the Academy of Medicine, which he did soon afterwards.

From that time, my attention has been keenly directed to the subject; and I have had frequent opportunities of verifying the lesson I learned from Dr. Bourgeois. Few years pass in which I do not see patients of this kind in the clinical wards, and still greater numbers of them come to me in my private consulting-room. I exceedingly regret that I have kept no note of these curious cases: but during the current year, within three months, I have seen four cases, one in my wards in the Hôtel-Dieu, one in my consulting-room, and the other two in the practice of two brother-practitioners of Paris.

Before describing these cases which I have examined recently, allow me, gentlemen, to read to you the account of the two cases which constitute the basis of the memoir which Dr. Bourgeois sent to the Academy of Medicine in 1855, which account he was so obliging as to communicate to me.

"About the year 1846," says Dr. Bourgeois, "I was called to the neighborhood of Pithiviers, to a gentleman aged thirty, affected with dropsy, for which he had been under treatment by two of my brother-practitioners for some weeks. I found the patient in bed, to which I was told he was chiefly confined. The face was pale and swollen; but neither the features nor the color of the skin were much altered. The whole body was greatly swollen: there was no cyanosis: the pulse was quick and compressible: the appetite was deficient: the thirst was great: inspiration became impeded in the horizontal decubitus. The abdomen, much distended, communicated the sensation of fluctuation; and by pressing on it somewhat strongly, a large, isolated, oval tumor was felt, which reached from above the umbilicus to deep down in the pelvis. The cellular tissue of the limbs and trunk were much distended. On examining the heart and lungs, no lesion was detected, except that, at the base of the chest, in the precordial region, there was dulness, due to the presence of a certain amount of fluid in the pleuræ and pericardium. I forgot to mention that the patient had never had a complete stoppage of urine: he was always passing urine unconsciously, and without any desire to urinate. His constitution was slightly lymphatic, but otherwise good. When in a heated state, he had bathed in very cold water, and had in consequence suffered from slight diarrhœa and colic in the lower part of the abdomen: then came the almost total inability to pass urine, which began at last to dribble from him constantly. Some time after this, seeing that his limbs were swollen, and being still more alarmed by the diminution of his strength, he summoned the doctor of Pithiviers: matters becoming worse, I was called in.

"The examination which I made of the abdomen led me to think that the cause of the anasarca was to be found in the state of the urinary passages: that the bladder was enormously distended; and that the patient had urinated by overflow from engorgement, which had misled my colleagues. In all such cases, as a preliminary measure, I propose the use of the catheter: in this case the proposal was accepted by the patient and his medical attendants. Hardly had the silver catheter entered the bladder, than the urine gushed forth in a full stream, till at least three litres had passed. The relief was immediate: before we left the patient, the desire to urinate, absent for several weeks, was felt, but without the ability to satisfy it. The catheter was introduced a second time, and we were astonished at the quantity of urine evacuated: it was clear, limpid, and nearly without odor. A catheter was left in the bladder, which was unstopped, at the patient's request, every half hour. This system was continued for two or three days, during which period, the patient passed from twelve to fifteen litres of urine. Within the same period, the anasarca entirely disappeared, and all the functions were very speedily restored, excepting the voluntary emission of urine. The patient was obliged to draw off his water by the catheter, till his death, which occurred several years later, of cerebral disease.

"Some years later, I was sent for to Pussay, to visit a strong old man of sanguineous temperament, nearly seventy-five years of age, who for some months had had difficulty in completely emptying his bladder. This old man, urinating with increasing difficulty, and passing a certain quantity of urine unconsciously, seeing, moreover, that his limbs were swollen, called in a physician of Augerville, who considered the case as one in which there was incontinence of urine, and incipient dropsy, the latter affection not having any relation to the former. As matters increased in seriousness day by day, I was asked to meet in consultation the ordinary medical attendant. I found the patient in the following state: there was no sensible alteration of the features: there was infiltration of the face: the decubitus was dorsal: he was confined to bed: there was very little oppression in the breathing: the pulse was tolerably full, but compressible, and not quick: the appetite was impaired: the bowels were seldom opened: there was a constant dribbling of urine, by which great erythema of the surrounding skin had been caused: the state of general swelling was very marked.

"On applying some force in the palpation of the abdomen, there was discovered, in the hypogastric region, a large oval tumor, extending from the pubes to above the umbilicus: no desire to urinate was excited by pressure on the tumor. On seeing this poor old man, and hearing a short statement by his relations, I immediately recollected my previous case, and concluded, that the anasarca was due to insufficient micturition, and that there was also a reflux of fluid into the splanchnic cavities and interstitial tissue. I was convinced of the nature of the case by a most thorough examination, and immediately proposed catheterism: this was attempted in vain by a silver catheter, the only instrument at our disposal. After numerous trials, we were obliged to adjourn the operation till we were provided with more suitable instruments: but the family, one of whose members was at that time under the treatment of M. Ségalas, proposed that that able practitioner should be called in, a proposal to which we assented with satisfaction. Next morning, we were accompanied in our visit to the old gentleman by M. Ségalas. In vain, M. Ségalas attempted to surmount the obstacle by using metallic catheters and soft sounds of cylindrical, conical, and olivary shape: for a long time, he was unable to pass an instrument into the bladder, till, at last, he succeeded in introducing a curved catheter. The instrument had no sooner entered the bladder, than the urine issued forth in a rapid forcible stream. Without a pause, the patient passed several litres of urine. As the introduction of the catheter had been a matter of so much difficulty, we deemed it prudent to leave it in the bladder. Some time after the first evacuation—which was complete—the patient felt, for the first time, a desire to pass urine: the stopper was then taken out of the instrument, when, considering the short time which had elapsed since the bladder was emptied, a surprising quantity was discharged. Every half hour (or hour at the longest), the bladder was emptied by request of the patient; and, as in the previous case, the dropsy disappeared within two or three days. The power of urinating voluntarily never returned, although, with the exception of this inconvenience, he lived in completely restored health, till he died, four or five years afterwards, of a disease in no way connected with the affection now described. Till the end of his life, it was necessary to draw off his urine by the catheter. The operation was regularly performed by a member of the family, and always without difficulty. In this case, there was no real urethral obstacle—only a deviation at the prostatic portion of the canal."

I now proceed, gentlemen, to describe the cases which have recently occurred in my own practice.

In July, 1864, a gentleman, aged sixty-four, consulted me in my cabinet. He ascended my stair with difficulty, and although he had rested more than an hour in the waiting-room he was panting dreadfully when he entered my consulting-room. His face and hands were infiltrated; and forthwith, I discovered that the inferior extremities were equally swollen. The abdomen was very large.

The patient informed me, that the general swelling of his body had commenced two months previously, without any appreciable disturbance of his health except severe abdominal pains. The anasarca began in the legs; and in eight or ten days, had extended to the whole body. The physician in attendance had then ascertained that there was a tumor in the abdomen; and in sending his client to me, he directed my attention to this tumor, which he regarded as the starting-point of all the symptoms.

Ere long, the swelling increased enormously; and at last the patient's miseries culminated in orthopnœa.

I conjectured that he had Bright's disease; and asked him to give me some of his urine. He passed urine in my presence, without much difficulty; but the quantity passed was small. I found neither albumen nor glucose in the urine. I auscultated the heart and lungs, without finding anything to explain his serious condition.

Having caused the patient to lie down on the sofa, I very carefully examined his abdomen. I there found an enormous tumor, reaching from the pelvis to above the umbilicus. It was elastic, and perfectly round. I at once perceived that it was the bladder. Without saying anything to the patient, I introduced a catheter into his bladder, which I did without any difficulty; and withdrew three litres of limpid urine. The tumor forthwith disappeared.

I then inquired into all the circumstances prior to the invasion of the symptoms which had led him to consult me. I learned facts which had not been stated to me in the first instance, as the patient's attention had not been directed to them.

For two or three years his bladder had been sluggish. He urinated frequently, both by day and by night; and on every occasion he had to use efforts. A month before the commencement of the general dropsy, he observed that he could not urinate when in bed, lying down: he required to kneel. A few days later he became obliged to get out of bed to make water. After some days passed in this state, he became unable to urinate on first taking up the vessel; he was obliged to walk, barefooted, up and down his room for some minutes ere any urine passed; and then, it was only with great straining, that he could pass above half a tumbler.

It was at this stage that his feet began to swell. He had then called in the physician who had found in the hypogastrium a very hard tumor which he considered a malignant growth. He had inquired about the urine, and had been told by the patient that his urine was rather more abundant than when he was in a normal condition: the physician, consequently, paid no more attention to that matter. Strange to say, when the patient came to me, he did not mention his urinary difficulties; and it was not till after I had used the catheter, that I obtained from him the details I have now given you—in fact, not till after I had, to a certain extent, guided his recollections.

I saw clearly that all the dropsical symptoms were consequent upon the retention of urine. As he had an affection of the prostate, and as I felt that I was quite incompetent to deal with such an affair, I placed him under the care of a colleague, more able than me, who taught him to introduce the catheter. As soon as the evacuation of the urine was regularly attended to, the anasarca disappeared, as well as the dyspnœa and other serious symptoms under which the patient had suffered for two months.

On the 5th September, 1864, I saw, in consultation with Dr. Lepère, a case of which I am now about to give you a very succinct account.

The patient, a man of fifty-five years of age, was very subject to hæmorrhoidal flux, by which he had been greatly weakened. From time to time, during the three preceding years, he had experienced, during the night, a stoppage in the stream of urine of which he did not take much notice, attributing it to spasms induced by the hæmorrhoids. During the summer of 1864, he often felt difficulty in passing his urine: there was an obvious change in his state of health: he became thin, and his appetite was impaired. In the meantime, during July, he went to Ems with his wife, for whom the waters had been prescribed.

About the 15th of August, M. Lepère was informed by letter that the patient had suffered dreadfully from his hæmorrhoids; that hands, legs, and abdomen were swollen; and that the physicians had discovered a cancerous tumor connected with the liver. Our Parisian colleague replied, that the latter statement could not be correct, for he had, a month previously, very carefully examined the abdomen by palpation, and had then ascertained that the organs were in a normal state. In answer, M. Lepère received an assertion still more categorical than the first. When the wife had concluded her course of the waters, the husband was sent to Kissingen. There, the severity of the symptoms increased rather than diminished. M. Lepère received a letter, in which he was requested to invite me to meet him in consultation in the case on the 5th September.

We found the patient seated. He was suffering considerably from oppressed breathing: the face was pale and puffy: the hands were œdematous: the legs and abdomen were enormously swollen. Before making a more minute investigation, we asked for some urine: it was clear, and did not give an albuminous precipitate when treated by heat. When we proceeded to palpation, we at once found that there was a large, round, elastic tumor in the hypogastrium and reaching far above the umbilicus: in size and form, it resembled the uterine tumor at the eighth month of pregnancy. The tumor, I perceived, was evidently the bladder distended with urine; and I had no difficulty in seeing that the case was similar to those reported by Dr. Bourgeois, and to others like them which I had myself observed. A catheter was introduced into the bladder, when eight litres of urine were withdrawn.

It is unnecessary for me to add, that the tumor entirely disappeared. After four days of the use of the catheter, the swelling was gone; and the secretion of urine was very abundant.

It becomes very interesting to inquire how it was, that this patient, a man of great intelligence, and very observant of his different symptoms, should have forgotten all that pertained to the bladder in the history of his disease. When we had brought back his recollections to their proper bearings, the patient told us that since his sojourn at Ems, he had been obliged to rise every night, and even to walk about the room when he wished to urinate: he had never had retention of urine, and the night preceding my visit, he had urinated voluntarily five or six times, each time passing one hundred and fifty grammes of urine.

Gentlemen, I dwell on these circumstances, because this patient, like the other whose case I have described to you, never spoke of retention of urine; never even mentioned having suffered from any inconvenience referable to the urinary passages. It was only by palpation of the abdomen, that a clue to the diagnosis was obtained.

My part in the case of M. Lepère's patient was at an end. As he had an affection of the bladder, M. Ricord was called in. He found that there was a very large calculus; but did not consider it advisable to attempt lithotrity. In two months, the patient died, with symptoms of purulent infection depending on extensive disease of the prostate.

During the same month, I saw a patient living in one of the streets of the Marais, to whom I was called by one of my colleagues. There was general anasarca, which had lasted nearly six weeks. The patient was a man about sixty years of age, who had had sluggishness in urinating, but no incontinence of urine. My colleague, who had perfectly ascertained that there was repletion of the bladder, feared, however, from the existence of dropsy, that there was serious organic disease. I related to him some of the cases which I had seen; and I had no difficulty in getting him to adopt some of my ideas as to the absence of danger. It was agreed that the catheter should be used several times a day. Soon afterwards, I learned that the anasarca had disappeared from the time that free passage had been afforded to the urine.

Recently, when in company with Dr. Follin, I was speaking to him of the singular relation between retention of urine and general dropsy, when he mentioned to me that he had been called to the country to see a patient whom he found in the conditions similar to those I have been describing; and that he had been surprised and alarmed at the anasarca which existed.

It is quite certain, then, gentlemen, as stated by Dr. Bourgeois, that retention of urine may occasion general dropsy. The recognition of this fact has a very important bearing on practice. We know that anasarca may show itself in the form in which it usually appears in Bright's disease, and yet be as easy of cure, as it is inexorably rebellious to treatment when associated with that malady.

Again, there is the curious fact, that many patients are not aware that they have retention of urine. When we see dropsy supervene slowly, extending even to the face, and when, at the same time, the health of the patient becomes deteriorated in a manner similar to that which takes place in diseases of the urinary organs, even in those which arise unknown to the patient, it is very difficult not to believe in the existence of some serious organic mischief; and even after it has been ascertained by abdominal palpation, that the bladder is distended by urine, one would be apt to attribute the retention to a malignant tumor, were it not for the favorable view suggested by such cases as I have just laid before you.

Gentlemen, it is difficult to explain the manner in which the species of anasarca now under consideration is produced. I have often asked myself whether, when the urine, this excrementitious fluid, has been long pent up in the bladder, it does not flow backwards by the ureters into the pelves and calices of the kidneys, so as to distend the organs and impede their function. By the blood being thus prevented from throwing off its excess of water, which flows abundantly to the renal surface, general dropsy is produced. I have a great dislike to this mechanical explanation, and hazard it with much timidity. Perhaps we are justified in supposing, that the compression of the kidneys prevents that complete emunction which they ought to perform; and that, in consequence, the constitution of the blood undergoes a great alteration—an alteration which ceases on removal of the cause.

For the present, gentlemen, let it suffice that you know that there is a species of anasarca due to retention of urine; and that you learn to detect it and treat it.

LECTURE XCIX.

MOVABLE KIDNEY.

Frequency of Movable Kidney.—Reason of this Frequency is the Feebleness of the Attachment of the Kidneys.—Frequency greater in Women than in Men; and on the Right than on the Left Side.—Explanation.—Movable Kidneys are not always Painful.—How they become Painful.—Numerous Errors of Diagnosis: Means of avoiding them.—Treatment.

GENTLEMEN: You have seen very recently among the out-patients at the Hôtel-Dieu, a man thirty-five years of age, robust in appearance, of powerful muscle, and presenting every condition of good health, who complained, nevertheless, of having an abdominal tumor, and of suffering from *peritonitis*, to which affection he said he was very subject. A tumor in the abdomen is very unusual, and peritonitis is still more unusual, in a man who generally enjoys good health. The man's face expressed pain, but had not a pinched appearance; and he had no fever. The possibility of displacement of the kidney at once presented itself to my mind; and I told the patient to take off his clothes. You saw that the abdomen was furrowed by the cicatrices of cuppings and leechings, showing that the patient had many times previously suffered from similar pains, and had on each occasion been treated for peritonitis.

Although thickness of the abdominal parietes rendered exploration difficult, and although it was rendered still more difficult by the pain, I could easily recognize and enable you to verify for yourselves the existence of a tumor in the right side. It was hard, shaped like an orbicular ring, and rather painful. It could be easily moved backwards and forwards, but it could not be brought to the median line. By steady gentle pressure, it could be pushed into the right renal region.

The patient was free from fever, and consequently free from inflammation: pressure on the abdomen did not occasion pain, consequently, there was no peritonitis: there was neither vomiting nor difficulty in passing water, consequently, neither nephritic colic nor renal lesion existed. The tumor in question, moreover, had really the form of the kidney, and could be pushed into the renal region. In your presence, I made a little experiment, which was quite a demonstration. Pressing on the tumor, I caused a certain amount of pain: placing then the hand on the left renal region, and making similar pressure, I produced pain in all respects similar, the patient said, to the pain occasioned by pressing the tumor in the right side. The tumor, therefore, could be nothing else than the right kidney: in fact, the case was one of movable kidney, and not of peritonitis, as had till then been supposed.

I ordered this man to wear a concave cushioned bandage, for the double purpose of supporting the kidney and protecting it from being injured by external objects. I also advised him to abstain from all antiphlogistic treatment. I recommended use of baths and poultices when the kidney was painful.

You will remark, that in this case it was the right kidney which was movable. It is that kidney which is generally affected by mobility and displacement. You will also remark, that the patient was a man: according to statistics, the subjects are more frequently women.

Why do the kidneys become movable? Why does the right become more frequently movable than the left kidney? Why does the movable kidney so easily become the seat of pain? These are questions which I wish to discuss with you.

Gentlemen, we cannot too carefully study the marvellous arrangements which nature has contrived for the protection of our organs. There is a simplicity of means, and a grandeur of results, presenting an aggregate which I am never weary of admiring. Arrived though I be at that period of life when the susceptibility to enthusiasm is pretty well past, I still feel an enthusiastic admiration for the works of nature.

Protection of organs is one of the fundamental principles which preside over the structure of the living body. Every organ requires to be protected from the objects in the external world; and yet, every organ requires to communicate with the external world.

The brain dwells and moves within a box of bone which is both thin and strong. It is thin, that its weight may not impede the movements of the head: it is strong, in virtue of its spheroidal form; and all the bones of which it is composed are dovetailed into one another by a series of indentations which diminishes strain, and deadens shock. The spinal cord is likewise protected by a bony case, the vertebral canal, which, with very great strength, unites elasticity to give power of resistance, and flexibility to impart ease of movement. You know to how great an extent the lungs are capable of expansion within their cage formed by flexible resisting arcs, the ribs, and by elastic planes, the intercostal muscles. Within the same cage, the heart is contained. The liver lies on the right side, hidden behind the lower false ribs, and under the diaphragmatic arch. The spleen lies on the left side, protected by the lower false ribs. The kidneys rest on thick muscular masses, formed by the quadrati lumborum and the origins of the psoas muscles. On the inside, they are protected by the vertebral column; and on the outside and posteriorly, by the quadrati lumborum, transverse processes of the lumbar vertebræ, the sacro-lumbar muscular mass, the spinalis dorsi, and very strong aponeuroses: in front, the intestinal convolutions separate them from the abdominal parietes. The bladder and uterus are protected inferiorly and posteriorly by the pubic girdle, within the cavity of the pelvis. The intestines alone seem to be badly protected: they have only the protection of a muscular wall. But then, as they are subject, by the requirements of digestion, to alternate expansion and contraction, it is essential that they be placed within a cavity which is extensible like themselves. Here, nevertheless, protection is also assured: to permit the free transit of their contents, the intestines are distended with gas: these gases form an elastic, and consequently a protective cushion. To promote the passage from above downwards of the contents of the digestive canal, that canal is endowed with an incredible facility of movement; and to this same facility of movement, it owes its protection from injury by shocks. You are acquainted with the great instinctive contractile power with which the abdominal muscles are endowed. When palpation of the abdomen is performed without due precaution, there is immediately perceived a stiffening, caused by an automatic contraction of the muscles, which comes to the protection of the subjacent organs. Within the abdomen, it appears then, that we find only those organs which are soft and elastic, the stomach and intestines, in relation with the soft abdominal parietes: all the hard organs—the liver, spleen, kidneys, and uterus—are placed deep, where they are protected by osseous bulwarks. From their solidity, pressure on them occasions pain and injurious consequences. You now perceive why it is, that the kidneys when movable are exposed to pressure and painful friction, giving rise to many untoward symptoms.

Are movable kidneys always the seat of pain? To be able to answer this question, it would be necessary to examine the kidneys of all the subjects submitted to our medical investigation. This has been done by Dr. Walther, an accomplished physician of Dresden; and I will tell you forthwith the results of his researches. I hardly ever diagnose movable kidneys. When I do diagnose them, is when an individual suffering from them comes to me complaining of them. As he complains to me of symptoms caused by movable kidney, I conclude that a movable kidney is a distressing or painful affair. This, however, is a very illogical mode of reasoning; and yet it is one too frequently employed in medicine. It is by reasoning in this way, that the fatal mistake has arisen of supposing that deviations of the uterus are the cause of the pain, the pain being in reality due to the concomitant metritis. A woman complains to a physician of pain in the uterus, and manifold discomforts: he makes a digital examination, ascertains that there is a particular deviation of the uterus; and, ignoring the metritis, he concludes that the deviation is the cause of the uterine symptoms. But investigate by another mode. Follow the method of inquiry pursued by M. Gosselin at the Hôpital de Lourcine: he submitted indiscriminately all the women at that institution to digital examination of the uterus, those who were and those who were not suffering from a uterine affection, with the result of ascertaining, that every kind of deviation of the uterus was of very frequent occurrence: he found, moreover, that deviations of the uterus exist in women who do not suffer the very slightest uterine discomfort. Consequently, when women who suffer from the uterus have a deviation of the organ, their sufferings must not be attributed to that deviation, but to some other uterine affection, the deviation being in itself a very harmless affair.

Were all physicians accustomed to scientific precision, they would feel, that it was not logical to arrive at a conclusion until they had examined all data, and instituted experiments as counter-proofs. In the question now before us, the counter-proof consists in the examination of the kidneys of a great many persons not suffering pain. This, Dr. Walther has done: and he has obtained the curious result, that the kidneys are movable in a considerable number of persons who suffer in no degree whatever therefrom, who give no thought to the peculiarity, and are even ignorant that they have a movable kidney.

Movable kidneys generally become painful as a consequence of great and unusual pressure, of a blow, or of prolonged fatigue: and it is after such occurrences that the patients come to us with their complaints.

My *chef de clinique*, Dr. Peter, was one day summoned to a client, a robust man, usually in the enjoyment of good health. He was a distinguished architect, a person of great intelligence, who gave a very good account of his sensations. He stated, that from the evening of the preceding day, he had been suffering acute pain in the right side of the abdomen, particularly at one special place which he indicated. Dr. Peter, placing his hand on that place—being well instructed in the affection by a recent visit to Dresden—had no difficulty in recognizing, that at the special seat of pain there existed a tumor, that the tumor was movable, and that the movable tumor was the right kidney. But how was it, that this kidney, assuredly movable for a long period and not till then painful, should have suddenly become the seat of pain? Dr. Peter inquired whether the patient had ever received a blow on the region, or had worn too tight a garment. At once enlightened by the questions, the patient stated that, on the previous evening, he had been on duty as a national guard, which duty he had, he said, unfortunately to perform about once in six months; and still more unfortunately, since his last time of service, he had become much fatter, so that he had had great difficulty in putting on his military trowsers, now too tight for him. He persisted, however: the result was discomfort, which went on increasing till the following day, when it had become positive pain. Excuse the apparent triviality of these details: they teach their own lesson.

The friends by whom the patient was surrounded were already speaking of the application of leeches, because on a previous occasion they had been employed for similar symptoms. Dr. Peter, however, only prescribed the application of a poultice over the very sensitive kidney, a protracted general bath, and rest in bed for twenty-four hours. As he had foreseen, all the symptoms then ceased. As the patient was robust and stout—as his abdominal walls formed a thick cushion for the displaced kidney, and as generally, he suffered neither discomfort nor pain, Dr. Peter did not order a bandage to be worn, reserving that measure to be resorted to should any unpleasant symptoms supervene.

A very distinguished physician, Dr. Becquet, has published opinions which I believe to be correct on the pathogeny of movable kidneys, and on the causes which occasionally render painful those movable kidneys which are generally free from pain. He thinks that during the catamenial fluxion, the kidneys participate in the congestion of the genital organs, and become tumefied. This would explain the pains in the renal regions so frequently felt at the menstrual periods, particularly by women who are not regular in their courses. Being thus swollen and heavy, the kidney, particularly the right kidney, acquires a tendency to surmount the feeble obstacles by which it is retained in its place. When the congestion ceases, the organ returns to its original position; but similar congestion renewed again and again, pushes it farther and farther each time from its place. The kidney, at each congestion, becomes permanently heavier, and so descends into a lower position. In this way, says Dr. Becquet, the kidney, by slow degrees, but not without causing suffering, at last appears free and floating in the abdomen.*

In support of this statement Dr. Becquet mentions the case of a woman in whom, at each menstrual period, the kidney, swollen and very painful on pressure, could be felt bulging under the ribs. As soon as the periodical congestion ceased, the kidney regained its usual volume, indolence, and place. The movable character became permanent at a later date; and (as formerly) at each menstrual period, the now movable kidney became the seat of pain. On one occasion, the renal fluxion being excessive, partial peritonitis arose, followed by the formation of false membranes. This resulted in the displaced kidney ceasing to be movable, and becoming definitively fixed in an abnormal position.

My accomplished friend and colleague, Dr. Gueneau de Mussy, in the excellent lectures which he gave on this subject at the Hôtel-Dieu, quite adopted Dr. Becquet's opinion, and stated that he had met with a case which supported it. Dr. Gueneau de Mussy, however, added, that while he quite recognized that congestion may supervene, sometimes as a pathological condition, and sometimes as an epiphenomenon in the malady we are now considering, it must be admitted to be neither a constant cause nor a necessary complication, because floating kidneys are not uncommon in men, in whom their displacement cannot be attributed to any fluxionary process.† The correct view of the question could not be better expressed.

Dr. Gueneau de Mussy almost believes that hysteria and arthritis, though not causes of displacement of the kidneys, are causes of pain in displaced kidneys. He states, that he has met with movable kidneys most frequently in hysterical and gouty persons.

My pupil, Dr. Peter, has just met with a case, similar to that reported by Dr. Becquet: A robust negress, nurse in a family belonging to the Havana, on returning fatigued, one day, from the Exposition Universelle, was seized with pains in the right side. This occurred on the last day of her courses, which had been as abundant as usual. The pain became very acute; and the patient when she walked was doubled up in two. The family was alarmed: Dr. Peter was sent for. When he arrived, the pains had continued for about twenty-four hours. He ascertained that there was an oval tumor in the right renal region, directed obliquely towards the hypogastrium, passing the umbilicus, and reaching within three fingerbreadths of the pubes. This last-mentioned condition excluded the idea of pelvic hæmatocele, even though digital examination had not demonstrated the absolute integrity of the entire circumuterine region. The tumor was hard, painful on pressure, almost immovable: it was about twenty centimetres long and about ten centimetres in breadth. Dr. Peter had no hesitation in concluding that the tumor was a kidney out of place, bulky, congested during menstruation, and become painful, probably in consequence of fatigue from too much walking. The pain increased for three days; and there supervened, as in Dr. Becquet's case, slight circumscribed peritonitis. It was necessary to apply leeches twice, and to employ often-repeated baths for fifteen days. The pain then yielded. The size of the tumor diminished a little: its limits could be made out very exactly by palpation, without giving the patient any pain. The examination post morbum confirmed the accuracy of the diagnosis. It was unquestionably the right kidney which had been painful and swollen. It remained in its abnormal position, out of which it could not be moved up more than two centimetres, and could not be restored to its proper anatomical position, which remained unoccupied. The pain had quite left the patient; and for some days after ceasing to be confined to bed, she made no complaint except of a feeling of weight. There was no return of pain at the next monthly period. Subsequently, Dr. Peter made several examinations, and satisfied himself that the displacement of the kidney continued. It is well to add, that there was nothing abnormal in the state of the urine during the period of the pain: it may have been a little less abundant than natural; but it never contained albumen, blood, nor pus.

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* Becquet: Essai sur la Pathologie des Reins Flottants. [*Archives Générales de Médecine*, t. i, 1865.]

† Gueneau de Mussy: Union Médicale, 1867.

I very recently saw, in consultation, a patient suffering from displacement of the right kidney. The nature of the affection had been mistaken by his ordinary medical attendant, who was one of my colleagues in the service of the hospitals, and an exceedingly well-informed man. He thought that there was an abdominal tumor, which was true; and he used every effort to dissolve it by iodide of potassium without succeeding, which was fortunate. Notwithstanding all the treatment, or rather in consequence of it, the tumor became very painful, and gave great annoyance to the patient, who suffered also from the conviction that he had an abdominal tumor which could not be dissolved, and was destined to have an evil issue. The poor man had in fact fallen into a most dismal state of hypochondria. I had not much difficulty in satisfying the physician as to the real nature of the tumor, nor in satisfying the patient that his case was not one of much gravity. The only treatment required was the use of a suitable bandage.

You observe that six of the patients of whom I have spoken to you were men. Dr. Rayner, however, has observed that displacement of the kidney is much more frequent in women than in men; and of thirty-five cases collected by Dr. Fritz, thirty occurred in women.*

An attempt has been made to explain this greater frequency of displacement of the kidney in women, by their use, or rather their abuse, of the corset. That is the opinion of my accomplished colleague Professor Cruveilhier. "Displacement of the kidney," he says, "occurs when pressure by the corset is made on the liver: the kidney is then driven out from the sort of recess which it occupies in the anterior surface of the liver, very much as a nut is, by pressure, squeezed out from between the fingers." Professor Cruveilhier adds: "The left kidney is less frequently displaced than the right, because the left hypochondrium, occupied by the spleen and great curvature of the stomach, supports much better than the left hypochondrium, the pressure of the corset."

In the case of men, this explanation is not admissible. Moreover, I am far from believing that a change in the volume of the liver can be a frequent determining cause of pressing down, and of consequently producing mobility of, the right kidney.

Let me lay before you the account of another case of displacement of the kidney in a woman, for which I am indebted to Dr. Peter.

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* FRITZ: Des Reins Flottants. [*Archives Générales de Médecine*, 1859, p. 158.]

A woman, about thirty years of age, presented herself in the out-patients' consulting-room at the Hôtel-Dieu. She was tall, well made, and presented all the characteristics of the nervo-lymphatic temperament. The face, however, had an almost waxy paleness: the sclerotics were bluish: and the general expression of the countenance indicated long-continued suffering.

The first thing of which this woman complained was a *tumor of the liver*, a tumor the existence of which had been long previously ascertained by many physicians, who all concurred in regarding it as incurable. She had never had the paroxysm of pain which characterizes hepatic colic, nor the jaundice which is its sequel. She had never suffered from dyspeptic symptoms, invariably associated with organic disease of the liver. She had never had epistaxis. Her general state did not in any way indicate that radical alteration of the organism always observable when a grave lesion of the liver has existed for three years.

Dr. Peter, deferring for greater leisure his examination of the liver, pursued his inquiry with a view to elicit whether there existed any other cause to explain the anæmic cachexia indicated by the waxy paleness of the face. He soon discovered that the patient had been confined three years previously, that after delivery she had had metritis, that that metritis became chronic, and caused profuse menorrhagia at each menstrual epoch. Having ascertained these facts, Dr. Peter placed the patient on a bed, and methodically examined the abdomen by palpation. He found that the liver extended three finger-breadths beyond the false ribs in the mammary region, that its vertical diameter at that point was twelve centimetres, which is nearly normal. A little lower down, there was felt a hard ovoid tumor, the upper margin of which was in juxtaposition with the liver. This was the only relation which the tumor had with the liver: there was no point at which it adhered to the liver. The tumor could be made to float in the abdomen, so as to be brought to the umbilicus: it was very painful to the touch. Dr. Peter had already formed his opinion; but to have superabundance of proof, he pressed his hand down into the region of the right kidney, and found it empty. This settled the question beyond any possibility of doubt. To make the matter more evident to pupils, he delineated both the liver and the movable tumor with his plessigraph: it could then easily be *seen* that the liver had its normal form, that the tumor had the ovoid form of the kidney, and that there was a space of nearly two centimetres between the most accessible margin of the tumor and the lower surface of the liver, from which a tympanitic sound was elicited on percussion. It was, therefore, evident that the tumor in question did not adhere to the liver, and was in fact the displaced right kidney.

Consequent upon delivery, the abdomen of this woman had become very flaccid; and it was some time after her confinement that she experienced, for the first time, pretty severe pains in the right side. These pains were not accompanied by vomiting, nor by any general disorder of the system. Some time previously, this woman had become a servant in one of Duval's eating-houses. As you know, the female servants in these establishments are constantly on their legs, running from table to table, and quickly going up and down stairs. Cannot you now understand how the right kidney, already movable, perhaps, had become definitively displaced from not being adequately supported by the relaxed abdominal parietes?—how this displacement had increased by an occupation in which the displaced organ was being constantly subjected to shocks in the movements of the body?—and how the pains should be more severe than usual after a day of more than usual fatigue? It is well to remark that the patient simultaneously gave all these details—details which, when rationally grouped, throw a flood of light on the etiology of the affection.

The most curious circumstance in relation to the diagnosis of this case, is that the woman—by her own account—had been examined by more than ten physicians, and that all, with one exception, were of opinion, that she had a malignant tumor of the liver. The physician who dissented in opinion from the others had a still more strange view of the case. Without taking into account the seat of the tumor, its form, and the circumstance that its distance from the pubes was more than ten centimetres, he believed it to be a tumor of the uterus. True, this doctor was a homœopath. He treated the metritis, which really existed; but he cured neither it nor the tumor. The unfortunate woman had to pay him two hundred francs.

Being a very intelligent person, she had observed, that when her abdomen was sufficiently supported, she either suffered very little, or not at all, in the tumor, from the fatigues of the day. Acting on this observation, she had a belt made for herself, which was rather ingeniously constructed, but too tight. Dr. Peter had no difficulty in getting her to make another in place of it, of strong drill, formed so as to embrace all the soft parietes of the abdomen, and capable of being laced and unlaced at pleasure. This girdle, in the situation corresponding to the tumor, was provided with a concave elastic cushion, intended at once to support and fix the tumor.

When we think of the feeble means by which nature has fixed the kidneys, it is rather matter of astonishment that they are generally retained in place, than that they are sometimes displaced. They are appended to the vascular system by the renal artery and vein—a feeble means of attachment, it must be admitted. The circumrenal cellulo-adipose tissue cannot keep the kidney fixed in its place. In reality, there is only the peritoneum to keep the kidney in apposition with the quadratus lumborum and psoas muscles; and this statement is proved to be correct by the facility with which the organ is removed when the peritoneum is torn. But the peritoneum is an agent very inadequate to fix the kidney. You perceive, then, that a sufficient reason for displacement of the kidney exists in the feebleness of the attachments which retain it in its position.

It is quite evident, that with such anatomical predispositions to displacement, increased volume, necessarily involving increased weight of the kidney, must almost inevitably lead to its position being lowered, and its being rendered movable. Hydronephrosis is also a cause of displacement of the kidney: cases of this kind are mentioned by authors. This affection did not exist in any of my patients; and the great relative frequency of movable kidney ascertained by Dr. Walther can only be explained by the feeble fixings of the organ.

As to the *symptoms* of this ectopia, the truth is, that in the great majority of cases, there are none: that is the conclusion derived from Dr. Walther's researches. Sometimes, the individual affected accidentally perceives a hard movable tumor, which is painful on pressure. In such a case, when the physician is consulted, he will be able to ascertain that the tumor is a smooth and ovoid body, presenting the physical characters of the kidney. In most cases, the entire outline of the organ cannot be traced out: generally, we can only feel the upper part, from the organ lying in a direction obliquely from behind forwards, and from without inwards. This tumor is dull on percussion. By skilful palpation, it is discoverable that the renal region, on the side of the tumor, does not contain a kidney. The examination will be specially convincing, if we explore in succession the two renal regions. Such an investigation serves as a counter-proof confirmatory of the diagnosis.

Functional disturbance is absent in the majority of cases: when present, it consists in general discomfort, a feeling of weight, dragging, and pinching, but rarely amounting to actual pain. When pain does exist, it is generally of a dull character: sometimes, it causes depression of spirits. At times, patients speak of feeling as if one of their organs was detached [*décroché*], and floating free in the abdomen.

Whatever painful sensations are experienced by the patients, it is evident that they must be aggravated by great muscular exertion, prolonged or rapid walking, dancing, riding, and jolting in an ill-hung carriage. It sometimes happens, that the first uncomfortable sensations are experienced under the influence of one or other of these causes. At other times, as in the second case which I described to you, pain was first set up by the pressure of a too tight garment.

As direct or indirect symptoms of movable kidney, Dr. Gueneau de Mussy likewise enumerates lumbar hyperæsthesia, pleuralgia, and dyspeptic symptoms. That eminent clinician remarks, that rest, and the horizontal decubitus, subdue the symptoms which have been augmented or renewed by walking, jolting in a carriage, or the disturbance excited in the system by menstruation or the gouty fluxion. At other times, the pains manifest themselves in remote parts—in the thigh, epigastrium, or lower intercostal spaces, for example. Dr. Gueneau de Mussy considers these pains as reflex phenomena, the starting-point being the kidney, and which, through the ganglia of the sympathetic and spinal cord, are transmitted to the spinal nerves.

Dr. Gueneau de Mussy believes that hypochondria, particularly in gouty and hysterical persons, is a possible consequence of pains depending on reflex action in cases of movable kidney.

Neither the secretion of urine nor micturition are in the slightest degree influenced by the kidney being movable.

As nothing frightens patients so much as the existence of an abdominal tumor, particularly when the physician appears ignorant of the true nature of the tumor, and when his endeavors to disperse or diminish it are too clearly unavailing, ectopia in a certain proportion of cases, induces melancholia and hypochondriasis. Such was the state of my patient, when I saw her in consultation.

Movable kidneys may cause endless errors in diagnosis. Our Hôtel-Dieu out-patient, you have seen, was supposed to have had a series of attacks of peritonitis. In the private patient whom I saw with my colleague, it was supposed that there was a malignant tumor.

Professor Cruveilhier says: "I have seen the tumor formed by the displaced right kidney treated as an *obstruction of the liver* or a *morbid* growth." Dr. Rayer says: "The pains which sometimes accompany mobility of the kidney have been mistaken for *nervous colic*, for the phenomena of *hypochondriasis*, and sometimes even for *lumbar and sciatic neuralgia*." You will recollect that my private patient had become hypochondriacal.

Let me now explain to you the manner of searching for a movable kidney. The physician placing himself at the side of the ectopia—the right side let us suppose—will glide the left hand along the margin of the lower false ribs, between them and the crest of the ilium: with the right hand, he will then slowly depress the wall of the abdomen, and so push aside the intestines, and be thus enabled to reach the displaced kidney, and get it between his hands. In this way, he will be able to ascertain what the organ is, and also its abnormal mobility. By following the mode of examination which I have now described, Dr. Walther has ascertained that mobility of the kidney is exceedingly frequent; that it is a condition which generally remains unknown to the individual affected by it; and that it is a source of innumerable mistakes.

The tumor constituted by the displaced kidney may be mistaken for a tumor of the liver, gall-bladder, spleen, mesentery, intestine, or for a fibrous tumor of the ovary. The intestinal tumors for which it may be mistaken are invaginations, and collections of fæces. Lumbar pains arising from displaced kidney have often been supposed to depend on metritis in women who had leucorrhœa along with the renal ectopia.

It will, however, be sufficient to search the renal region on the side on which the tumor is situated, to determine whether or not it be empty; and to find out, whether (as in the Hôtel-Dieu patient) pressure on the tumor and on the kidney which is *in situ*, does not produce exactly similar sensations. A tumor of the liver is not movable. When the spleen is low down in the abdomen, it is more voluminous than the displaced kidney. Intestinal tumors give rise to symptoms which are special and characteristic. A fibrous tumor of the ovary is indolent. Digital examination, if required, and the use of the speculum, by revealing the state of the uterus, will enable us to set down to their true cause the pains attributed to metritis.

In itself, displaced kidney does not present any gravity in the prognosis. The prognosis only becomes serious through blunders in diagnosis, and consequent improper treatment—treatment generally the more active, the less sure the physician is as to the nature of the affection. You have seen the abdomen of our Hôtel-Dieu patient covered by numerous cicatrices of leechings and cuppings, resorted to for the treatment of imaginary peritonitis. Taking blood from that man was useless: it weakened him, and did not cure him. It was equally useless to employ blisters and resolving ointments in the case of the private patient whom I saw in town. The too evident want of success of these measures in that case was shown by the confusion of the physician, and the hypochondriasis of the patient.

When the existence of renal ectopia has been discovered, its treatment follows as a necessary deduction. A prior indication must, however, be fulfilled—the kidney must be replaced. But to do this is almost impossible. In the case of women, however, who lace the corset too tightly, there is reason to hope that the kidney will return to a certain extent to its normal situation, upon a system of more moderate constriction being adopted. There are two secondary indications which remain to be stated,—to support the kidney, and to protect it. One apparatus will fulfil both intentions. The patient may be recommended to wear either a broad belt formed of woven elastic caoutchouc material, similar to that of which stockings are made for persons having varicose veins; or they may be advised to wear a belt made on the plan of the hypogastric belt, furnished with a slightly concave cushion adapted to the particular case. I found an apparatus of this description succeed in the two cases of which I have been speaking to you. I need not say that the bandage must be provided with thigh straps, to prevent it rising up out of its place. It is unnecessary to say more on this means of support. When you have made your diagnosis, you will easily find the means of supporting and protecting the displaced kidney when it is the cause of pain. I have said nothing of rest, baths, or cataplasms: but quite understand, that they must be resorted to when required. What I particularly wish you to bear in mind, is the frequency of ectopia of the kidney; and the frequency with which it leads to mistakes not less injurious to the reputation of the physician, than to the health of the patient. Finally, movable kidney is an infirmity which is not serious, which we can always hope to alleviate, but hardly can ever hope to cure.

LECTURE C.

LOOSENING OF THE PELVIC SYMPHYSES.

Condition which is generally mistaken.—Mistaken for Disease of the Spinal Cord or Uterus.—Locomotion is Difficult or Impossible.—Patients suffering from it have a Peculiar Walk.—Pain in Pelvic Symphyses. Constriction by a Bandage at once facilitates Walking.—Conditions to be fulfilled by the Bandage.—Puerperal State may lead to Suppuration of the Pelvic Articulations and Death.

GENTLEMEN: I have pointed out to you the errors in diagnosis and treatment which displacement of the kidney may occasion.

To-day, I propose to speak to you of an affection which is, generally, not one of gravity; but which, nevertheless, may embitter a woman's life, by leading her to suppose that she has a disease of the spinal cord or uterus, when there only exists a loosening of the pelvic symphyses.

Before recalling to your recollection the two cases which you have had an opportunity of observing in the clinical wards, I will describe the cases of the two ladies in whom I first observed this affection, which till then had escaped my notice.

Madame X. was married at the age of twenty-three. Her first child was born a year after marriage; a second was born two years later; and a third, when she was more than thirty-six years of age. Her husband had not always been a well-conducted man. Four or five years after his marriage I treated him for constitutional syphilis. Soon afterwards, his wife had exostoses and baldness, symptoms which led me to recognize in her the same malady for which I was treating her husband. These occurrences took place long before the lady's last confinement. The child was born without any sign of syphilis, and still enjoys good health. Labor was natural, and all went on favorably after delivery. There was nothing unusual in the size of the child's head. From excess of precaution, I desired this lady to keep her bed for fifteen days, and to lie on the sofa for another similar period. I then allowed her to get up. When she tried to walk in her bed-room, she complained of pain in the kidneys and throughout the pelvis: more than a month elapsed before she was able to walk round her room. As she had a little leucorrhœa, I thought slight metritis, a rather frequent sequel of delivery, might be the cause of all her symptoms. I recommended injections, and waited. But one day, when visiting her, I saw her rise from her seat to walk, and was struck by her manner of proceeding; she waddled, dragging one leg after the other with difficulty, leaning greatly to right or left according to the foot she was advancing. She could not stand on one foot; and, on attempting to do so, became doubled up, complaining of acute pain in the hips and loins. It appeared that what she complained of was a feeling of extreme weakness. The possibility of a spinal affection flashed through my mind; but on carefully investigating her sensibility and movements, I ascertained that the skin had lost none of its tactile aptitudes, and that the different movements were well performed, provided the horizontal position was maintained.

It then occurred to me that there might be separation of the symphyses. As the patient was very plump, it was impossible for me to make out the existence of this separation, as I so easily did in the woman who lay in bed 13, St. Bernard Ward, whom you all examined.

Pain, somewhat acute, was produced, however, on pressing the mons veneris at the symphysis of the pubes, and on pressing the hips in the situation of the two sacro-iliac symphyses. I forthwith rolled a folded sheet round the hips and pelvis, drawing it as tightly as possible. I then told the patient to walk, which she did at once with the greatest ease, astonished, moreover, to find that her strength, which she had considered as lost, was restored, and that the pains were gone. I directed her to make a laced bandage of drill, so constructed as to encircle tightly the whole of the pelvis and upper part of the thighs. From the time that she began to wear this apparatus, she was able to go about her household affairs, and take some walking exercise. Six weeks later, she was able to discontinue the bandage. Her recovery was complete.

Some time after the occurrence of the case I have now related a lady was brought to my consulting-room: she was twenty-five years of age, the wife of an officer of one of our special schools. Her husband had carried her to the top of my stair, yet it was with great difficulty that she walked to the sofa. She had not come to consult me for this supposed weakness of the legs, which she considered as a sequel of delivery prior to menstruating. She came to be cured of a very painful temporo-facial neuralgia, for which she had already tried a multiplicity of means of treatment. My prescription proved very useful to her. A month later she asked me to call on her. It was on that occasion, after thanking me for having cured her neuralgia, that she spoke to me of an affection which, on the first occasion, had very little attracted her notice. She told me that she had had two confinements, quickly following one another, unattended by any untoward occurrences. Her last confinement had taken place three months previously, leaving what she believed was debility of the legs, preventing her from walking. She had no uterine lesion. Leaning on two arms, she walked with great distress, dragging her feet like a paralyzed person: when she attempted to raise the leg, as one does to step forward, the other leg, having then to sustain the entire weight of the body, immediately bent under her, causing her to fall unless supported by some one. She was quite unable to walk alone. I ascertained, just as I did in the case of the other lady, that there was no paralysis: and it at once occurred to me that I might succeed in this, as I had in the former case. I tightly applied a small table-cloth [*naperon*] round the pelvis, and upper part of the thighs: as soon as this impromptu bandage was fixed, I asked the lady to lean on my arm, and try to walk; which she did, but, at first, very timidly. Feeling no pain, by degrees, she gained confidence, and leaned less and less on my arm. When she had reached the end of the drawing-room I asked her to walk back alone, which she did with a good grace, and with great joy. I then had made for her a doeskin girdle, so formed as to encircle tightly the pelvis and both trochanters. A fortnight or three weeks later I had the satisfaction of again seeing this lady in my consulting-room. She continued to wear the bandage. She had ascended my stair without difficulty. She told me that she had been trying to walk out a little. Two months later her recovery was complete.

You recollect the big wench who occupied bed 20 in the nursery ward. She was brought to the hospital some days after her second confinement. There were neither uterine nor peritoneal symptoms to make us uneasy. As she was in a perfectly satisfactory state of health, I was not at all thinking about her, when the nurse told me that this woman was quite unable to stand on her legs when she rose in the morning to make her bed. I examined her with care. I ascertained that she had no lesion of the uterus, and that when in bed she could pretty easily perform all movements. Sensibility was unimpaired. However, even when in bed this woman felt pains in the sacro-iliac and pubic symphyses. It was rather distressing debility than real pain [*une faiblesse pénible qu'une véritable douleur*] that the patient experienced when she moved. She had difficulty in standing, unless she steadied herself by her hands; walking taxed her utmost powers, obliging her to drag her legs, to stop every three or four steps, and to lean on some one's arm or on the back of a chair. She walked from bed to bed, laying hold of the iron bars of the beds. Pressure on the pelvic symphyses did not occasion pain; there was neither swelling nor redness in that situation. There was only a little loosening of the articulations, a relaxation of which the patient was conscious, and of which the physician became aware when he tried to move separately the iliac bones. I pointed out to those who followed my hospital visit, that it was possible to restore the use of her legs to this woman by constriction with a bandage. I at once made the experiment. A bandage was applied very tightly round the pelvis, so as to render the articular surfaces immovable; immediately the patient was able to walk. She only remained at the Hôtel-Dieu till a suitable drill bandage was made for her use

I have learned from persons who have seen this woman that she has quite recovered. She is able to go about her rather hard work as a charwoman, without fatigue or discomfort.

On the 12th July last, a tall, well-made, robust woman, twenty-four years of age, was admitted to St. Bernard Ward. She had been confined, for the first time, on the 19th of June. The labor was in all respects good, except that it was somewhat painful from the great size of the child. On the ninth day after delivery, she wished to get up, but found it absolutely impossible. From the time she made the attempt, she experienced great debility in the inferior extremities, as well as an acute pain in the genital organs, a pain which she compared to the sensation of an existing obstacle.* She likewise experienced a feeling of weight in the loins.

From that time, she suffered the same distressing sensations when she attempted to turn quickly in bed, as when she stood or tried to walk.

Consequent upon this first unsuccessful attempt to get up, this woman took to her bed for some days longer, hoping that more prolonged rest might restore her strength and free her from pain. This result was not obtained. When she renewed her attempt to get up, she found it as impossible to walk as on the former occasion. It was then—the twenty-third day after delivery—that she resolved to come into the hospital.

On her admission, I ascertained that standing gave her very great pain, and that walking occasioned acute suffering. It was observed that she immediately edged herself backwards and turned over on her bed, in which she begged to be at once replaced. When interrogated as to the nature and seat of her sufferings, she complained of the genital organs [*des parties génitales*], without more precisely indicating the specially painful parts.

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* "Douleur qu'elle comparait à la sensation d'une *barre*." The word "*barre*," in obstetrics, signifies a projection or prolongation of the symphysis pubis; and a female who has this pelvic deformity—a cause of difficult labor—is termed "*une barrée*."—TRANSLATOR.

As the general state of the patient was very good, as she had a satisfactory appetite, and was free from fever, it was improbable that pains so acute could depend on inflammation of the uterus or its annexes: my attention was, therefore, at once directed to the state of the pelvic symphysis. In exploring the hypogastrium, I was able to demonstrate to you, that in place of suffering pain in the "genital organs," as she stated, the woman was suffering in the pubic arch, the sole seat of pain being a point corresponding to the pubic symphysis. By palpation in that situation a very considerable separation of the articular surfaces was recognized; the extremity of the index finger could easily be introduced between the two bones of the pubes, and it could at the same time be felt that the interarticular cartilage was softened. This exploration was very painful. I avoided trying to move the one bone on the other: this experiment, which would have caused great pain, would not have given me any additional information; it was clearly a case of loosening of the symphysis pubis. To make certainty more certain, I examined the internal genital organs, and found that they were in a perfectly healthy state.

When in bed this woman could move her legs perfectly well, and she made no complaint of any symptom of paralysis. There could, therefore, no longer be any doubt as to the nature of the case; the symptoms were evidently dependent on disjunction of the symphysis pubis.

Had there still remained the possibility of doubt in the diagnosis, it would at once have been dispelled by the result of the treatment. I girded an abdominal bandage on the hips, pressing tightly round the pelvis, including trochanters and pubic arch. No sooner had this improvised bandage been applied than the woman, who a minute before could not stand, was able to walk easily, carrying her infant in her arms at the same time.

I had all the trouble in the world to get her to remain some days in the hospital. She wished at once to leave on foot. She only remained till her bandage was made. I had it constructed with the greatest possible simplicity—that is to say, it was a strong, broad girdle, wherewith to encircle the sacro-iliac symphyses, the trochanters, and the pubes.

You will remark, gentlemen, how easily a superficial observer might have formed an erroneous diagnosis in this case. The woman had been recently delivered; she vainly complained of pain in the genital organs; she said she could not walk without pain; in point of fact she could not walk. Would it not have been very natural, in the first instance, to conclude that the patient was suffering from a lesion of the uterus? But this error could not have been committed, if, in interrogating the patient, she had been pressed to indicate the precise point where she felt pain, the finger being forthwith employed to make such an examination as you saw me make; there would then have been ascertained—what you saw me ascertain—separation of the bones, and softening of the cartilages.

Errors in diagnosis are apt to arise from the existence of lumbar and hypogastric pains. There may be leucorrhœa: on digital examination, we may discover lacerations or persistent granulations of the cervix uteri: with such symptoms existing, and no additional data except the vague statements of the patients to guide us, the impossibility to walk or stand might quite naturally be attributed to metritis. The mistake is the more excusable, that metritis occurs very often, and loosening of the symphyses very seldom.

I have no claim, gentlemen, to having made a discovery. Disjunction of the pelvic symphyses has been pointed out by obstetricians. They have even tried to explain it by the great size of the head of the fœtus; they have suggested, that by acting like a wedge it separates the previously softened symphyses. This probably occurred in the case I last related to you. The woman said that her infant was very large.

In this lecture, I have been particularly anxious to call your attention to an affection of rare occurrence, and consequently little known. So little is it known, that some of your text-books on midwifery, including Cazeaux's treatise, do not even mention it as a possible sequel of labor. There is an indisposition to admit the presence of a pathological state of rare occurrence—a tendency consequently, to attribute symptoms rather to an anomalous metritis than to a loosening of the pelvic symphyses. I wish you to be on your guard against committing a mistake of this kind.

Loosening of the pelvic symphyses is so far a serious affection that it absolutely prevents walking, the inability increasing, the longer the patient makes no attempt to walk. You saw that the patient whose case I first related to you could with difficulty walk a few steps in her apartment, two months after delivery: that the second patient, three months after delivery, could not walk at all. There is no physiological reason why such a condition should not continue indefinitely. We have learned from experience, that rest alone is not sufficient to cure the diastasis when it is considerable. To promote consolidation, it seems necessary to bring the articular surfaces into contact with one another by artificial means.

From these considerations, you can at once deduce the proper treatment. As you know, man, being a biped, requires a pelvis sufficiently solid to be proof against all trials of strength in walking. If the sacrum could move on the ischium, or if the bones of the pubes were not solidly united, walking would be impossible; for the weight of the body would then inevitably cause disjunction of the bones of the pelvis. When loosening of the pelvic symphyses is an obstacle to locomotion, they must be artificially consolidated. A girdle requires to be placed round a pelvis which has its staves separated: it is necessary to supply the temporary deficiency of intrinsic contention by an extrinsic contention—that is to say, by tight application of a bandage, in such a way as to bring into contact the separated surfaces of the symphyses.

As you have seen, this bandage can be improvised. A strong towel properly tightened answers quite well. But to secure a solid durable apparatus, it is better to have a bandage made of strong drill or doeskin, which can be laced more or less tightly at pleasure; and which is constructed in such a way as to embrace the trochanter, as well as the bones of the pelvis. Should such an apparatus be found insufficient, a steel spring may be added to it, arranged so as to make simultaneous pressure on the sacrum, ilium, and pubes. If the loosening be considerable, and the pain very acute, rest must be enjoined: but let me repeat, that rest is not enough by itself: to wait for consolidation would be losing time: it is absolutely necessary for the patient to have recourse to an apparatus till she can walk without its aid.

We have seen how a bandage of the simplest possible description may relieve and rapidly cure loosening of the symphyses. I cannot terminate this lecture without endeavoring to explain to you how this pathological softening is merely the exaggeration of a physiological state, having as its final cause the facilitating of the exit of the fœtus. During pregnancy, the pelvic articulations gradually lose, to a certain extent, their intimate union: the ligaments become relaxed, permitting during labor, a minute augmentation of the diameters of the pelvis to allow the head of the fœtus to traverse the outlet more easily. This physiological loosening may become excessive, and render walking difficult during the later weeks of pregnancy:

and walking may even be rendered completely impossible by the wide separation of the pelvic symphyses which has taken place during labor.

I have said enough on this point. Let me now remark, that in consequence of the existence of the puerperal state, inflammation may complicate post-mortem loosening of the pelvic symphyses, and lead to death.

A woman, forty years of age, occupying bed 3 of St. Bernard Ward, was admitted to our wards some weeks after her confinement, in consequence of a pain in the right iliac fossa, and the continuance of fever from the date of her delivery. She had become very thin, and was losing strength day by day. She had nearly altogether lost her appetite; my *chef de clinique* observed that every evening she became very hot, that condition being generally preceded by rigors. The progress of the disease, and the absence of characteristic symptoms, afforded no ground for concluding that the case was one of purulent infection or continued fever. There was no pulmonary affection to explain the paroxysms of fever. The uterus was not the seat of pain: there was no discharge: there was no pelvic abscess: and the right iliac fossa, the seat of pain, did not contain a tumor. One day the patient told me that the pain had extended to the right hip. My first examination was without result: but at an interval of some days, as the pain continued, I more carefully explored the region of the nates, and found that there was œdema in that situation, that pressure there occasioned pain, particularly over the right sacro-iliac symphyses. I made an exploratory puncture with a capillary trocar, by which I withdrew several drops of greenish fetid pus. The patient left the hospital; so that the history of her case is incomplete.

Some months later, in October, 1862, a patient was admitted to bed 30 St. Bernard Ward, four weeks after her confinement. Five days after delivery, she had felt pain in the right hip of so acute a character as to prevent her from getting up. Some days later, she was seized with rigors and fever; and for three weeks prior to admission, she had never been without fever. The pain had invaded the right hip, and the symphysis of the pubes. The right hip was painful; and although the patient presented none of the symptoms of purulent infection, I had no hesitation in saying that in all probability the sacro-iliac and pubic symphyses were the seat of an inflammation which explained the constant fever. The frequent rigors, I regarded as indicating suppuration of the weakened joints. Both hips soon became œdematous, and gave clear evidence of deepseated fluctuation. I introduced a bistoury in the direction of the right and left sacro-iliac symphyses. A great quantity of pus flowed from the incision: the opening was prevented from closing by the introduction of a dossil of charpie. I had formed an exceedingly unfavorable prognosis. The fever continued: and the patient died a few days after the opening of the abscess.

At the autopsy, it was found that the sacro-iliac symphyses were denuded of their cartilages, and that the greater part of the articular surface showed the appearance of ostalgitis. There was pus also in the symphyses of the pubes, but it was in small quantity; and in some places, only the cartilage was frayed. No morbid appearances were presented by the uterus or its annexes. No metastatic abscesses were found in the lungs, liver, or any other organ. In the right iliac fossa, under the muscle, there was an abscess communicating with the corresponding articulation.

Bear in mind, gentlemen, that this woman was delivered at her own home, away from epidemic influence. Also, bear in mind, that fifteen days prior to delivery, she had had pain in the right hip. What information are we to deduce from these facts? Are we to infer, that in recently delivered women, loosening of the pelvic symphyses may become so painful as to be accompanied by inflammation, leading sometimes to the most serious consequences? You cannot pay too much attention to pains complained of in the pelvic symphyses: you must place the patients in conditions the most favorable for calming the pain, and arresting the progress of the inflammation, of which pain is sometimes the origin.

LECTURE CI.

PERCUSSION.

Influence of the Sensualistic Philosophy on Contemporary Science and on the Tendencies of the Parisian School.—Pinel, and the Natural History of Diseases.—Pathological and Semeiotic Anatomy inaugurated by Corvisart.—Discovery of Percussion by Avenbrugger, and of Auscultation by Laennec.—Succession of Works on Semeiology.—Immediate and Mediate Percussion.—The Pleximeter.—The Plessigraph: manner of using it.—Comparative Value of the Modes of Percussion.—Medicine does not consist solely in the study of Morbid Anatomy and Semeiology.—Micrography and Nihilism in Therapeutics.—Necessity of associating Modern Precision with the Medical Doctrines of Past Times.

GENTLEMEN: Before I leave this clinical chair, I wish to speak to you on the subject of percussion; and keeping that object in view, I desire to take with you a rapid glance at contemporary medical doctrines. It may be said, that the dominant characteristic of our medical age is the application of physical methods of investigation, and an ambition to attain in medicine that precision and rigor which belongs to sciences called exact.

You know, gentlemen, that medicine always takes its inspiration from the reigning philosophy; which means that every philosophical doctrine has, in the history of medicine, a corresponding medical doctrine. The actual medical tendencies are certainly those which the grandsons of the eighteenth century ought to possess. They are, moreover, the universal tendencies of contemporary science.

No one in France, to whatever philosophical religion he may belong, will deny that sensualistic philosophy has exercised a great influence on the scientific progress of our age.

Whether it be to blame or to laud them, it must be admitted, that the philosophers of the eighteenth century, who with one exception, invoke the "*sage* Locke," and derive their inspiration from the sensualism of Condillac, have not uselessly explored French philosophy to its lowest depths.

At the voice of these philosophers, who were by turns scientific, ironical, and impassioned—but always accessible from the simplicity of their language—the era of revolutions opened. On so deeply disturbed a soil, new institutions alone were possible. Social and scientific rights had alike undergone a change.

Medicine came under the same influences; and also had its own revolution. By one of these providential occurrences, of which history presents examples, the needed man—Laennec—appeared: a great discovery was made—auscultation. From that time, the spirit of medical inquiry, obedient to the impulse which it had received, unresistingly surrendered itself to the pursuit of analytical methods.

Laennec's discovery, however, was not an isolated fact: it was the progeny, so to speak, of other discoveries, but its widespread celebrity and admirable results gave it a dominant power over medicine, and definitively stamped the movement with a character which it still retains.

Medicine, as I remarked, aspired to precision and rigorous exactness; and wished to be one of the "exact sciences." The study of lesions—that is pathological anatomy—and the study of symptoms—that is semeiology—being the departments of medical science most accessible, even to mediocre intellects, and being departments easily admitting of culture by rigorously scientific means, were at once impetuously taken up. On the other hand, therapeutics, the study of which is infinitely more complex and difficult, became almost entirely neglected. Medicine was thus cultivated for the sake of science, and without a thought—so to speak—as to the means of assuaging the sufferings or promoting the well-being of patients. A disease became a subject of abstract study: it was studied as if it were a plant, an animal, or a physical phenomenon. Pathology became no more than the natural history of diseases.

It was under the influence of that bias that Pinel wrote his Nosography. In systematizing morbid conditions, in seeking to classify them according to types representing general species and varieties; he did what was of great use for the cause of study, because the mind likes to repose on a distinctly defined type: but he did an injury to practical medicine, and in the end to science; because he believed, and led others to believe, in the abstract types which he had created.

I say that Pinel did an injury to practical medicine: the young practitioner imbued with his doctrines, when at the bedside of his patient, sought in vain for the well-depicted entity of disease which he became acquainted with when a student on the benches of the school. The doctrines to which I refer, led practitioners to believe in the necessary and almost methodical progress of diseases, which naturally produced the serious evil of abstinence from all treatment—of "expectation" in therapeutics.

I say that, in the first place, Pinel inflicted a direct injury on science, because he taught the untruth, that diseases are entities admitting of classification like animals and plants; and, in the second place, he did mischief indirectly to science, by inculcating minute analysis, a searching after the slightest differences in symptoms, which tends to introduce confusion, by infinitely multiplying morbid species and varieties.

Nearly at the same epoch, Corvisart, a man clear in his conceptions, ready and reliable in judgment, lucid and incisive as a speaker, inaugurated the new era of pathological anatomy in France, by passionately espousing the science illustrated by Morgagni. Around him were grouped Dupuytren, Bayle, and Laennec, men whose names were destined to become renowned.

Then it was, that a series of discoveries were made, the filiation of which I now wish to elucidate to you. In what I am going to say, you will find additional proof of the correctness of the statement I have often made to you, that nothing new is ever achieved in science at one single bound. The appreciation of one fact leads to the appreciation of another, as a consequence or corollary from the first: and one discovery begets another discovery. Thus it was, that by a chain of facts and ideas Avenbrugger was the precursor of Laennec.

In 1761, Avenbrugger, a laborious physician of Vienna, published a modest duodecimo entitled: "*Inventum Novum ex Percussione Thoracis ut* *signo abstrusos interni pectoris morbos detegendi.*" This title had the double drawback of being too long, and of not being very intelligible. Avenbrugger's discovery, the starting-point as you will see of all modern works on precise observation, made no impression in Germany. The author had timidly and indirectly, but uselessly, placed his work under the patronage of "the very illustrious Baron Van Swieten:" the book had but little success: the inventor of percussion lived in obscurity, and died unknown. In 1773, Rozière de la Chassagne translated Avenbrugger's work into French.* The translation had as little success as the original. Corvisart, imbued with the positive doctrines of his time, and searching for the means of discovering during life the lesions found after death, having seen in Stoll, his favorite author, that information such as he desired could be obtained by percussion of the chest in cases of disease of the thoracic organs, resolved to read the entirely forgotten work of the German physician. This resolution brought good fortune to Avenbrugger's discovery, though not perhaps to his book.† Corvisart immediately made experiments in respect of percussion; and for twenty years, he practiced it before his clinical pupils at the Hôpital de la Charité. Nor was that all: when palpation and percussion failed to furnish him with sufficient data, he was in the habit of applying his ear to the chest of the patient to enable him better to distinguish the sounds of the heart. Thus, Corvisart supplemented insufficiency of touch by the aid of hearing. You perceive, how one method of physical examination thus led to the discovery of another physical method. But though Corvisart was possessed of a talent for diffusing knowledge, he was not equally endowed with a talent for invention. By a small intellectual effort, he might have rendered his name immortal by the discovery of auscultation. This effort he failed to make. Among his pupils, however, there was a young physician, a man of grave and meditative mind, full of ancient lore, and yet inspired with the spirit of modern inquiry. Laennec, following his master's example, applied his ear to the chest; but he was more fortunate than his master in knowing how to listen and understand. He discovered auscultation.‡

Laennec has himself related how he came to make the discovery—how he came to think of practicing mediate auscultation. In 1816, half a century after the discovery had been made by Avenbrugger, he was consulted by a young woman who had the general symptoms of heart-disease, and in whom, from her plump condition, the application of the hand, and percussion, afforded little insight into the nature of the malady. Laennec, "on account of the age and sex of the patient," felt himself interdicted from applying his ear to the chest: he recollected "a very well-known acoustic phenomenon, viz., that if the ear be applied to one extremity of a post, the stroke of a pin at its other extremity will be heard very distinctly." He then applied to the precordial region one end of a very tightly compacted roll of paper, and placed his ear at the other end of it. He "was equally surprised and pleased, to hear the pulsations of the heart with much more precision and distinctness than by the immediate application of the ear." It was not by chance, therefore, but by reasoning, that Laennec discovered auscultation: in the true meaning of the word, he *invented* auscultation, because he *sought* for it, the means of search he employed being induction.

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* As an appendix to his "Manuel des Pulmoniques:" Paris, 1773.

† "La Nouvelle Methode [d'Avenbrugger] pour connaître les Maladies Internes de la Poitrine par la Percussion de cette Cavité: traduite en français par J. N. Corvisart:" 1808.

‡ See LAENNEC'S work, entitled: "De l'auscultation Médiate: ou Traité du Diagnostic des Maladies des Poumons et du Cœur."

The physical examination of the chest, installed with so much eclat by Laennec, suggested the examination of, if not all the organs, at least the products of secretion; the urine was submitted to the action of chemical reagents, as was likewise the blood, the great nutrient secretion. Albuminuria was discovered: and glycosuria became better known. To the anæmia of the old physicians, were added uræmia, leucocythæmia, and melanæmia. Examination by the speculum was brought to perfection and generalized—too much generalized perhaps: the laryngoscope was used for examining the larynx: the ophthalmoscope was used for examining the eye—the interior of the eye—that expansion of the brain—by ascertaining the state of which we can sometimes judge of the state of the encephalon. The most inacessible organs can now be seen. These conquests of modern semeiology are due to the spirit of investigation developed in the contemporary generation by the successive discoveries of Avenbrugger and Laennec.*

Let me now return to the subject of percussion. Avenbrugger recommended percussion to be performed by striking the chest slowly and gently with the extremities of the extended fingers placed close to one another: "*Percuti, verius pulsari thorax debet, adductis ad se mutuo et in rectum protensis digitorum apicibus, lente et leniter.*"

To this method of employing percussion, Corvisart added percussion with the open hand [*percussion à main ouverte*], which, he said, is "a method exceedingly useful for ascertaining the extent of the part of the thorax which is not resonant, and appreciating more correctly the amount of the obstacle."

Avenbrugger's method is suited for ascertaining differences in elasticity, that is to say, differences in the consistence of the parts percussed: but, strange to say, he never mentioned this, and very probably was not aware of it. He speaks only of the difference of sound. However, one obtains a much better knowledge of difference of consistence by striking with the pulp of the extended fingers than with the open hand as Corvisart recommended.

It is also very remarkable, that *Avenbrugger really practiced mediate percussion:* he recommended percussion to be made over the shirt rendered very tense, or with a gloved hand, provided the glove was not made of smooth leather: "*Thoraci supertensum sit indusium vel manûs percutientis chirothecâ* (*modo ex polite corio non sit*) *muniatur.*" Corvisart regards this precept as useless, believing that it matters not which way is adopted. Avenbrugger was careful to add a note, stating that in percussion of the naked chest by the naked hand, the contact of the smooth surfaces produces a particular sound (*strepitus*) which modifies the quality of the real sound which ought to be elicited: "*Si nudum pectus nudâ manu pulsatur, superficierum politarum concursus strepitum producit, et soni evocandi constitutionem obscurat.*" It is very evident that Avenbrugger wished to get rid of the noise of the skin, or at least to deaden it, so as to get the pure sound from the deep-lying parts; and that he practiced a species of mediate percussion. In place of employing a plate according to the modern plan, he interposed a piece of stuff or leather between his fingers and the chest; and in place of striking with the open hand, he percussed with the extremities of the fingers, exactly as has been recommended, long after his day, by the advocates of mediate percussion.

We have seen that Avenbrugger percussed with an intermediate piece of stuff or rough leather; and Corvisart regarded the use of an intermediate substance as a matter of indifference. There is some uncertainty as to who it was who first employed the finger as the medium in percussion: but it was the mode of percussion habitually adopted by Récamier. Dr. Piorry proposed to substitute for the interposed finger, a plate, which he called a *pleximeter*. The innovation was a real advance. You are all acquainted with Piorry's instrument, which is a plate with ears at the sides to enable it to be firmly held in the left hand. It is sometimes made of metal; but the pleximeters most used are of ivory. One of my pupils, M. Horteloup, son of my good friend and colleague of the Hôtel-Dieu, has had a pleximeter made of gutta-percha, which has not the disadvantage of giving the dry sound of the ivory. Another of my pupils, M. Oldfield, has brought out a percussion hammer, formed of a flexible whalebone stem terminating at one of its extremities in a very hard sphere formed of plates of leather very tightly compacted. Thus, we have a hammer and pleximeter formed of two substances nearly identical, which do not vibrate in the least degree, and yield with great purity the sound of the percussed parts. These are the two instruments which you generally see me use. I find them very serviceable; and, contrary to what may be supposed, I obtain by their aid a very precise conception of the consistence of bodies.

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* See the Lecture on Aphonia and Cauterization of the Larynx, volume i, p. 509, and the Lecture on Cerebral Fever, volume i, p. 871.

In Germany, very much use is made of a pleximeter half the size of that employed in France, and of a small steel hammer, or metallic hammer covered with caoutchouc.

The pleximeter, so long as it is not childishly used as a toy in cases where it is not required—so long as it is not employed to percuss a bone or an artery, the limits of which can be quite well determined by the eye and palpation—so long as it is not made a pretext for pleximetric exercises under which patients, common sense, and truth equally groan—the pleximeter is a good instrument, an instrument which renders essential service.

Percussion on the finger or pleximeter has the very great drawback of not giving a *simple*, but a *mixed* sound. At the junction of organs, the finger, and still more the pleximeter, are over two or more organs, which percussion causes to vibrate simultaneously, so that the sound elicited is a mixture of two or more orders of vibrations. For example, at the point where the liver and right lung intersect, the finger or pleximeter is generally placed over the liver and lung in such a way as to give a mixed sound on percussion, which is neither the sound of the liver nor of the lung. The sound is much duller than that of the lung, but it has not the dulness which would be obtained were the liver isolated when percussed. To ascertain the exact point at which the liver commences, it is necessary to feel one's way, to percuss a little above and a little below the place where the mixed sound begins, and after thus groping, the operator, if aided by great practice, discovers with considerable precision the point at which the liver begins. Similar remarks are applicable to percussion of the heart at the mediastinum. Again, we generally lose trace of the heart at its left margin, where it dips down into the chest, hiding itself, as it were, behind the lung.

The ideal of percussion, then, is to percuss the smallest possible surface, so as to disturb the smallest possible portion of an organ—to percuss in such a way, that at the distance of a few millimetres, the space not percussed should not be made to vibrate, and should not mingle its sounds with the sound of the percussed space. It is then very evident, that immediately the sound changes, we are within a few millimetres of the limits of another organ. To attain this ideal, Dr. Michel Peter, my former *chef de clinique*, devised an instrument by which he reduced to a minimum the percussed surface, by reducing to a minimum the percussing surface. As a percussing surface so exceedingly reduced could yield very little sound, the idea occurred to him of amplifying the sound by a supporting stem. In other words, he devised an instrument for percussion consisting of a cylindrical stem, the percussing extremity of which terminates in a truncated cone, and the percussed extremity in a disk larger than the cylindrical portion, and on the flat surface of which the percussing finger easily strikes. The circular plane which rests on the skin does not contain more than five or six square millimetres: it necessarily does not vibrate, nor cause to vibrate a greater extent of surface. In percussing, the instrument is moved progressively in a line, every point in which is tested, so that as soon as a change in the nature of the sound is heard, we know within about one or two millimetres—and without any groping [*sans aucun tâtonnement*]—that a new organ has presented itself under the instrument of percussion.

Dr. Peter's instrument is provided with a crayon, or (which is more simple) a slightly carbonized cylinder of cork, which moves with gentle rubbing, so that by pushing it out, a black mark can be made at every point where there is a change in the nature of the sound. The instrument, therefore, is at once both an agent for percussion and delineation. You can understand how greatly its use will shorten an investigation, by rendering it both more convenient and more precise: it obviates the necessity of holding in the hand which percusses, the pencil with which the limits of organs are marked.

Nor is this all: this instrument of Dr. Peter—which he calls a plessigraph—is exceedingly delicate. It is not necessary to *percuss*—it is sufficient merely to *touch*. The touch ought moreover to be as rapid as possible.

Let me now describe the manner of using this instrument: Hold the instrument firmly between the thumb and index finger of the left hand: the less you put the fingers on it, the less you deaden the sound. Place the thumb on the little knob by which the crayon is moved. Holding the plessigraph, as I have now described, in the left hand, apply it firmly and perpendicularly to the surface of the skin.

Touch the firmly held plessigraph with the palmar surface of the extended index finger of the right hand. Do not *percuss*, as when you use the pleximeter, with the ungual portion of the fingers flexed. You thus avoid hard striking, which might be painful from the smallness of the surface percussed: as the plessigraph is touched by the whole palmar surface of the index finger, and not by the ungual extremity, an incredible delicacy of sensation is obtained. The consistence of tissues is perceived with very great precision, which you can understand, as they are touched by the intermediary of the plessigraph just as in performing palpation for the discovery of the nature of bodies.

Dr. Peter's instrument is ten centimetres in length: it is divided into ten equal parts, each of which is one centimetre: one of these centimetres is divided into millimetres; so that the instrument, besides serving for the percussion and delineation of organs, serves also for their measurement.

To sum up: The plessigraph appears to me fitted to measure organs with extreme precision and with great rapidity. There is no difficulty in using it: the most inexperienced pupil can easily employ it, and obtain, on the first occasion of employing it, results which are not attainable with the pleximeter till after long experience and protracted groping. It has the additional advantage of leaving a series of black points wherever a difference of sound has been detected: whenever the limits of an organ have been ascertained, they become likewise delineated, so that the operator *sees* that which he has *heard:* the image remains permanently under the eye, long after the sound has ceased. You remember the precept of Horace:

> "Segnius irritant animos demissa per aurem :
> Quam quæ sunt oculis subjecta fidelibus."

Once more in conclusion: When one percusses with the extended fingers closely approximated, following Avenbrugger's method—when one percusses with the open hand in accordance with Corvisart's teaching—when one percusses on the finger, on the pleximeter, with or without plessigraph, one does well, provided examination by percussion be indicated. Here, however, a distinction must be made. The immediate percussion of Avenbrugger and Corvisart is preferable for an examination of the chest, with a view to ascertaining at once the general state of the cavity as to sonorousness. It is generally sufficient for the recognition of a pleuritic effusion or a moderately extensive pneumonia. Percussion on the pleximeter is not suited for the examination of the apices of the lungs, particularly for their examination behind. You have seen me sometimes percuss on the clavicle, with the extremity of the fingers using it as a pleximeter; and by this proceeding, I have obtained very satisfactory results in cases of pulmonary tuberculization. But for investigations in which precision is necessary, when it is desired to ascertain the exact limits of the liver, spleen, and other organs, particularly of the heart, and the exact limits of tumors of the mediastinum, such as a deep-lying aneurism of the aorta, the plessigraph seems to me to be preferable in many cases.

Gentlemen, if after mature reflection, we dispassionately sum up the balance-sheet of our knowledge, if, free from the passions which moved our predecessors, we endeavor to assign to every one his just share, if we ask ourselves the value of those instruments of analytical investigation in our hands, if we ask ourselves whether they constitute the whole of medicine, we are forced to answer by an emphatic negative. No: the knowledge they yield does not constitute the whole of medicine.

It verily seems, I deliberately repeat, as if the medical intellect had been upset by Laennec's discovery. Physicians rushed into excesses in physical inquiry: one would give the medical world his *petit bruit de souffle* and another would point out some *nuance* which had been neglected by the otherwise comprehensive genius of Laennec.

There also arose the strange idea that because henceforth lesions and their limits were more ascertainable, the means of curing them were better known. Medicine, it was supposed, was about to rival surgery in precision. This, however, was an enormous mistake; for although surgery sees clear as day the lesions confided to it for cure, it certainly cannot on that account cure them any better.

Assuredly, gentlemen, we owe much to our predecessors and contemporaries; they have advanced diagnosis to a marvellous degree of precision; but in so doing, they have only advanced semeiology. Medical science has progressed; but the art of healing has remained nearly stationary. In therapeutics, experiment is much more difficult; the data of the therapeutic problem are so numerous, the results so uncertain and deceptive, that it is impossible to arrive at a conclusion rapidly; and the conclusion, when attained, is far from being always susceptible of rigorous demonstration.

It is still an open question whether micrography in its most advanced development—the *cellular pathology* of Virchow—by reviving under a new scientific form, more suited to our age, the Epicurean system of atoms, does not lead directly to the annihilation of therapeutics. By regarding the living organism as a microcosm formed of heterogeneous and independent elements, it necessarily rejects all general medication, which can produce no influence on elements which are incongruous, and, to a certain extent, antagonistic. It forgets the man in thinking of the cell, and loses itself in an abyss of infinitesimals.

When medicine as it now exists compares what it knows with what it does, it perceives that pathological anatomy does not always necessarily lead to rational therapeutics; and that the knowledge of lesions does not always enable us to cure them. Here the deception begins. Too much being hoped for, disappointment comes too quickly; the descent from disappointment to skepticism is very rapid. Gird up yourselves, young men, to resist such tendencies! Rich in the possessions bequeathed to you by the physicians of the past, to you belongs the duty of uniting modern science with ancient wisdom, and of rekindling the temporarily despised torch of old medical traditions. Let this be your endeavor! It is a great and splendid aim! Fail not to pursue it!

With this exhortation I conclude.

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